Chapter 4: Conclusions

Summary and Recommendations by Key Question

Table 3 presents a summary of the findings of the evidence review for each key question. We have classified the evidence as none, weak, fair or good, defined as follows:

Classification	Evidence
None	no publications that address the key question in women
Weak	some evidence, but no systematic review *or* definitive evidence from randomized trials
Fair	at least one systematic review, but the review is only fair quality or outdated *or* evidence from randomized trials, but the trials are small or have inconsistent findings
Good	at least one recent, good quality systematic review *or* several major randomized trials with consistent findings

As noted in Chapter 2, Methodology, Hierarchy of Evidence and Completeness of Searches, we are confident that we have identified all systematic reviews and major randomized trials, but some large cohort or cross-sectional studies may not have been identified. Importantly, we were only able to include the results of studies if the results were stratified by gender. Thus, many completed studies may have the potential to provide evidence on women if the study investigators are willing and able to produce stratified results.

A new systematic review was considered feasible when we identified at least 5 or more studies using similar methods that could likely be included in a systematic review. In some cases, even though a systematic review is feasible, it was not recommended if a recent, methodologically sound systematic review has already been completed or a definitive randomized trial has been completed or is under way.

Findings by Level of Evidence and Key Question

Assuming that the strength of a risk factor is a different question from the effect of modifying the same risk factor, and that the effects of a treatment in primary prevention are different from the effect in secondary prevention, we reviewed the medical literature related to 42 questions pertaining to CHD in women. We found no data in women to address 13 of the questions, weak data to address 15, fair data for eight and good data to address six questions (Table 4).

Summary of Major Findings

There was fair data suggesting that the accuracy of exercise EKG and exercise thallium testing (using either conventional or SPECT imaging) for CHD in women is poor and that exercise echocardiography might be more accurate.

While evidence for many treatments was lacking, we found fair or good data to suggest that beta-blockers, aspirin and angiotensin converting enzyme inhibitors reduce risk for CHD events in women with coronary disease. Good evidence suggests that nitrates do not reduce risk for CHD events in women with known heart disease. There was fair evidence to suggest that IIb/IIIa drugs given to women undergoing percutaneous revascularization result in a reduced risk of CHD events and need for repeat revascularization. Fair evidence also suggests that IIb/IIIa drugs given to women suffering acute coronary syndromes result in increased mortality. This was the only treatment for which there appeared to be an interaction by gender: men treated with IIb/IIIa drugs during acute coronary syndromes appear to benefit.

We found only weak evidence linking most of the risk factors of interest and CHD risk in women. For the most part, this is because all of the studies addressing the strength of risk factors are observational, and very few good quality systematic reviews have been completed. However, there was fair evidence to suggest that hyperlipidemia and hyperhomocysteinemia are risk factors for CHD in women and good evidence that diabetes is a risk factor. Risk factors seem to be equally strong in men and women with the possible exceptions of age, diabetes, and specific lipoproteins. Increasing age seems to be a stronger risk factor for CHD events in women than men, but the evidence is inconclusive due to the small number of women included in the studies. Diabetes may be a stronger risk factor for CHD in women than in men and patterns of risk associated with lipoprotein subfractions appear to differ in men and women.

There was fair or good evidence to suggest that smoking cessation after MI and treatment of hypertension and of hyperlipidemia lower the risk for CHD events in women. In contrast, we found no evidence for the effectiveness of other interventions to modify risk factors in women.

Available studies suggest that men are more likely than women to undergo diagnostic testing and treatment for CHD, but that women are more likely to be treated for hypertension. These observed differences may be due to inadequate control for differences in severity of disease and comorbidities in men and women or result from overtreatment in men.

We found no evidence to address the prognostic value of troponins, creatine kinase or myoglobin in women with ischemia.

In general, no evidence addressed differences in the accuracy of diagnostic tests, strength of risk factors, effects of treatment or prognostic value of markers for ischemia in women of different races or ethnicity. The only evidence regarding differences by ethnicity suggests that African American women may benefit more from treatment of hypertension than white women.