The concept of social support is almost as old as the Old World. Aristoteles argued around 350 B.C. that friendship was a basic human need along with food, shelter, and clothing. "We naturally desire to love other human beings and to be loved by them. A totally loveless life—a life without friends of any sort—is a life deprived of much needed good" (Alcalay, 1983). Several centuries later, Paracelcus (1599), a physician, alchemist, and natural scientist, prescribed "love as the best possible cure for several diseases." The first scientific evidence of a link between social support and health was offered by Durkheim (1897/1951) in his extensive sociological studies on the origins of suicide and self-destructive behavior, in which he found that marriage and religion were the best protectors against such deviant behavior. Kropotkin (1908), a Russian ethologist and psychobiologist, stated that "mutual help and support is a factor of great significance for the maintenance of life and health in animals and in humans."

Most of the modern epidemiological studies from the Old World concerning the relationship of social support and cardiovascular disease (CVD) have been inspired by studies from the United States, such as the Alameda County study (Berkman & Syme, 1979). Because Chapter 6 discusses U.S. epidemiological evidence, this chapter focuses on internal studies only.
Before reviewing the international evidence, though, a few basic characteristics of epidemiological studies should be noted. As with any study based on statistical associations, results from epidemiological studies cannot be interpreted as providing conclusive evidence about causal relationships. Unlike experimental studies, in which virtually all external influences are kept under control, in observational studies one cannot control for all possible confounding factors or processes that may be underlying causes of the relationships found. One can minimize the possible bias, however, by adhering to some established standards for causal interpretation, such as the eight criteria suggested by Hill (1965):

1. **Temporality of events.** With reference to prospective studies, does the exposure precede the disease outcome?
2. **Strength of the association.** How much of the variance in the outcome variable is explained by the exposure variable? How great is the relative risk associated with the exposure?
3. **Consistency of associations.** Do several studies demonstrate similar results?
4. **Biological gradient.** Is there an increased risk of adverse outcome with increased exposure?
5. **Biological plausibility.** Is there evidence of a plausible pathogenic mechanism linking the exposure to the outcome variable?
6. **Coherence.** Do the conclusions from several kinds of studies (e.g., using animals, human populations, and human patient studies) point in the same direction?
7. **Experimental/intervention evidence.** Is there evidence from such studies that supports the associations found?
8. **Specificity of outcome.** Are several different effects of the exposure variable observed, or are the findings specific to one outcome?

In this chapter several of these criteria, such as temporality, strength, and consistency of the associations, are applied. For example, if it is found that different studies come to similar conclusions about an effect over time, about the strength of an effect and about the kind of pathogenic outcome involved, then the probability of a causal relationship is increased. Another criterion to be addressed is the role of intervention studies. Not only are these studies a test of preventive or therapeutic effects, but they can also be interpreted as experimental support for a causal relationship. For example, if it can be shown that increasing social support leads to a decrease in systolic blood pressure, the inference of a causal relationship between social support and blood pressure is strengthened.

The epidemiological studies reviewed in this chapter are mainly of two kinds. First I discuss longitudinal studies, which analyze the impact