

If we were to measure strength of effect by the difference of the incidence proportions, it would appear from Table 2-2 that for population 1, E=1 has a much stronger effect than B=1, because E=1 increases the incidence proportion by 0.9 (in both levels of B), whereas B=1 appears to increase the incidence proportion by only 0.1 (in both levels of E). Table 2-3 shows the analogous results for population 2. Although the members of this population have exactly the same causal mechanisms operating within them as do the members of population 1, the relative strengths of causative factors E=1 and B=1 appear reversed, again using the incidence proportion difference as the measure of strength. B=1 now appears to have a much stronger effect on the incidence proportion than E=1, despite the fact that B and E are not associated in either population, as is evident from the prevalence of E=1 being 50% for those with B=1 and for those with B=0. (The effect of E, but not of B, is estimable from tables 2-2 and 2-3. The effect of B is not estimable because it is confounded by A, but we will ignore that here. The topic of confounding is introduced in chapter 4.)

The overall difference of incidence proportions contrasting E=1 with E=0 is $(1,900 / 2,000) - (100 / 2,000) = 0.9$ in population 1 and $(1,100 / 2,000) - (900 / 2,000) = 0.1$ in population 2. The key difference between populations 1 and 2 is the difference in the prevalence of the conditions under which E=1 acts to increase risk: that is, the presence of A=0 or B=1, but not both. (When A=0 and B=1, the first of the three sufficient causes in Figure 2-2 is already completed; with E=1 the second and third are also completed, but as the risk is already 1, E=1 does not increase anyone's risk, though it may well shorten the time to the outcome.) The prevalence of the condition, "A=0 or B=1 but not both" is $1,800/2,000 = 90\%$ in both levels of E in population 1. In population 2, this prevalence is only $200/2,000 = 10\%$ in both levels of E. This difference in the prevalence of the conditions sufficient for E=1 to increase risk explains the difference in the strength of the effect of E=1 as measured by the difference in incidence proportions.

As noted above, the set of all other component causes in all sufficient causes in which a causal factor participates is called the *causal complement* of the factor. Thus, A=0, B=1, U₂ and U₃ make up the causal complement of E=1 in the above example. This example shows that the strength of a factor's effect on the occurrence of a disease in a population, measured as the absolute difference in incidence proportions, depends on the prevalence of its causal complement. A factor will have a strong effect, as measured by the difference of proportions getting disease, if its causal complement is common. Conversely, a factor with a rare causal complement will have a weak effect.

If strength of effect is measured by the ratio of proportions getting disease, as opposed to the difference, strength depends on more than a factor's causal complement. In particular, it depends additionally on how common or rare the components are of sufficient causes in which the specified causal factor does *not* play a role. In this example, the effect of E=1 measured in ratio terms depends on the prevalence of E=1's causal complement and on the prevalence of the conjunction of A=0 and B=1. If many people have both A=0 and B=1, the "baseline" incidence proportion (i.e., the proportion of not-E or "unexposed" persons getting disease) will be high and the proportion getting disease due to E will be comparatively low. If few people have both A=0 and B=1, the

baseline incidence proportion will be low and the proportion getting disease due to $E=1$ will be comparatively high. Thus, strength of effect measured by the incidence proportion ratio depends on more conditions than does strength of effect measured by the incidence proportion difference.

Regardless of how strength of a causal factor's effect is measured, the public-health significance of that effect does not imply a corresponding degree of etiologic significance. Each component cause in a given sufficient cause has the same etiologic significance. Given a specific causal mechanism, any of the component causes can have strong or weak effects using either the difference or ratio measure. The actual identities of the components of a sufficient cause are part of the mechanics of causation, whereas the strength of a factor's effect depends on the time-specific distribution of its causal complement (if strength is measured in absolute terms) plus the distribution of the components of all sufficient causes in which the factor does not play a role (if strength is measured in relative terms). Over a span of time, the strength of the effect of a given factor on disease occurrence may change because the prevalence of its causal complement in various mechanisms may also change, even if the causal mechanisms in which the factor and its cofactors act remain unchanged.