# The Impact of a Prevention Effort on the Community

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**Abstract:** Commonly used measures of the impact of an exposure on disease are inadequate for assessing the potential benefit of community-based efforts to prevent disease caused by the exposure in question. Because relative measures of effect, including the risk ratio, odds ratio, and population attributable risk (PAR) do not account for the absolute risk of disease, they lack the most crucial element for evaluating the opportunity for prevention of disease caused by the exposure. Attributable community risk (ACR), defined as the difference between the crude risk (or overall risk in the population) and the risk in the unexposed, better captures the potential impact of a prevention effort. PAR can be expressed as the proportion of the disease incidence in a population that is attributable to the exposure. ACR, by contrast, is the proportion of the population that is affected with the disease due to the exposure. Therefore, unlike PAR, risk ratio or odds ratio, ACR is useful for comparing the potential benefit of programs aimed at the prevention of different diseases. For example, ACR provides a useful comparison of the potential benefits of efforts to prevent breast cancer and to prevent ovarian cancer among women with a BRCA1 or BRCA2 mutation.

(*Epidemiology* 2005;16: 1–3)

In this issue, Rockhill[1] argues that we neglect the prevention perspective in the ordinary course of epidemiologic research. She is right, at least in part. Our best-loved tools focus our attention on relative measures—ratios of hazard, rate, risk, and odds. Although these measures are well suited for assessment of causality, they tell us next to nothing about the burden of disease in the population[2] or, therefore, about the potential impact of preventing disease by removing the cause.

All the commonly used measures are inadequate for assessing the potential benefits of preventive efforts. Consider possible interventions to reduce risk of ovarian and breast cancer from BRCA1 or BRCA2 mutations or from a common exposure or common polymorphism. The table presents plausible scenarios of disease risk and exposure distribution. A common exposure that confers low absolute risk (or, equivalently, a low-penetrance variant in a gene), as in scenario 1, can cause many more breast cancers than high-penetrance but rare BRCA mutations (scenario 2). Comparing either the risks in exposed (the penetrances; denoted by $I_1$) or the risk ratios (RR), which are both far lower for the common exposure, does not give the correct impression of which exposure causes more disease in the community. Similarly, an effective screening program for BRCA mutations among Ashkenazi Jewish women will save more lives than an effective screening in an unselected population (as in scenarios 3 and 2, respectively), even though RR and risk difference (RD) are similar in the 2 populations. BRCA mutations will cause far more breast cancer than ovarian cancer in an unselected population (scenario 2 versus scenario 4) and in Ashkenazi Jewish women (scenario 3 versus scenario 5), even though the risk ratio and population attributable risk (PAR) are far lower for breast cancer.

In fact, the benefit of a preventive maneuver in a population, whether in terms of disease or of death, is captured best in terms of the "body count," or the *number* of cases prevented[3]—a count that relative measures obscure. Analogously, the benefit to an individual is better captured by the absolute, rather than the relative, reduction in probability of an undesirable outcome.

Consider the odds ratio (OR) for exposure X and disease D defined in terms of sensitivity (Se = Pr(X|D)) and specificity (Sp = Pr(not X|not D)), as

$$OR = [Se/(1 - Se)]/[(1 - Sp)/Sp)].$$

Neither Se nor Sp, and, therefore, neither OR nor PAR, nor, indeed, any relative measure, reflect absolute risk of disease; therefore, each measure is severely limited as a guide to potential for prevention.

There is a neglected measure that does capture the potential impact of a successful intervention in a population.[2,4] *Attributable community risk* (ACR) is the difference between the current crude risk of disease and the risk after the exposure is eliminated, which is assumed to be the risk in the

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ISSN: 1044-3983/05/1601-0001
DOI: 10.1097/01.ede.0000147633.09891.16



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**TABLE 1.** Measuring the Potential of Prevention Strategies in Scenarios With Different Exposures, Different Diseases and Different Populations

| | | Breast Cancer | | | Ovarian Cancer | |
|---|---|---|---|---|---|---|
| | | Unselected Women | | Ashkenazi Jewish Women | Unselected Women | Ashkenazi Jewish Women |
| | | (1) Common Exposure | (2) BRCA Mutation | (3) BRCA Mutation | (4) Common Exposure | (5) BRCA Mutation |
| Population | | | | | | |
| | Population size ($N$) | 10,000 | 10,000 | 10,000 | 10,000 | 10,000 |
| | Crude risk* ($I_C$) | 10% | 10% | 10% | 1% | 1% |
| | Total cases ($N \times I_C$) | 1,000 | 1,000 | 1,000 | 100 | 100 |
| Scenario† | | | | | | |
| | Proportion exposed ($p$) | 10.0% | 0.2% | 1.5% | 0.2% | 1.5% |
| | Risk in exposed* ($I_1$) | 15% | 50% | 50% | 20% | 20% |
| | Risk in unexposed* ($I_0 = [I_C - (p \times I_1)]/(1 - p)$) | 9.44% | 9.92% | 9.39% | 0.962% | 0.711% |
| | No. of exposed cases ($N \times P \times I_1$) | 150 | 10 | 75 | 4 | 30 |
| | No. of unexposed cases ($N \times (\mathrm{I} - \mathrm{p}) \times I_0$) | 850 | 990 | 925 | 96 | 70 |
| | Total cases | 1000 | 1000 | 1000 | 100 | 100 |
| | No. of cases attributable to exposure ($N \times (I_C - I_0)$) | 55.6 | 8.0 | 60.9 | 3.8 | 28.9 |
| Summaries of Risk | | | | | | |
| | Risk ratio (RR = $I_1/I_0$) | 1.59 | 5.04 | 5.32 | 20.83 | 28.17 |
| | Risk Difference* (RD = $I_1 - I_0$) | 5.56% | 40.08% | 40.61% | 19.04% | 19.29% |
| | Population Attributable Risk (PAR = $(I_C - I_0)/I_C$) | 5.6% | 0.8% | 6.1% | 3.8% | 28.9% |
| | Attributable Community Risk* (ACR = $(I_C - I_0)/I_C$) | 0.556% | 0.080% | 0.609% | 0.038% | 0.289% |

*Risks are probabilities that a woman will develop disease from birth to age 70.

†Frequency of BRCA mutations is assumed to be higher in Ashkenazi Jewish women than in unselected women. Crude risk and risk in exposed are assumed to be the same in Ashkenazi Jewish and unselected women. The crude risks would change slightly if, instead, risks in both unexposed and exposed women were assumed to be the same in Ashkenazi Jewish and unselected women.

unexposed. ACR is defined in the 1960 edition of MacMahon's textbook,[2(table 35, p. 230)] as

$$\mathrm{ACR} = I_C - I_0 = (p \times I_1) + (1 - p)I_0 - I_0 = p(I_1 - I_0)$$
$$= p \times \mathrm{RD} = p \times (\mathrm{RR} - 1) \times I_0,$$

where p is the proportion of the population exposed, which is itself a weighted average of Se and 1 − Sp, or $p = [(1 - I_C) \times \mathrm{Sp}] + (I_C \times \mathrm{Se})$, and $I_0 = [I_C - (p \times I_1)]/(1 - p)$. In other words, ACR is the product of the proportion of the population that can benefit from the risk reduction ($p$) and the magnitude of a beneficiary's reduction in risk from the level in the exposed to the level in the unexposed (RD = $I_1 - I_0$). ACR is also the numerator of what is now called *population attributable risk* (PAR) *etiologic fraction*,[5]

$$\mathrm{PAR} = (I_C - I_0)/I_C = \mathrm{ACR}/I_C.$$

Confusing nomenclature has a long history; the 1970 edition of MacMahon's textbook[4(table 30, pp. 233–234)] used *population attributable risk* for what in 1960 was called *attributable community risk*. The 1960 edition also showed the calculation of what we nowadays (and in this paper) call *population attributable risk,* although without giving it a name or formula.

PAR is the proportion of the disease incidence in a *population* that can be attributed to the exposure, or the fraction of *cases* that could be prevented by an intervention that lowers everyone's risk to the risk in the unexposed. ACR is the fraction of the *population* that develops disease due


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(attributable) to an exposure, or the fraction of the *population* whose disease could be presented. For example, in scenario 1, we would conclude that completely eliminating breast cancer due to the common exposure is expected to prevent breast cancer in 0.56% of women, or in 56 women in a population of 10,000. By contrast, eliminating breast cancer due to BRCA mutations would prevent cancer in 8 of 10,000 unselected women (scenario 2) and in 61 of 10,000 Ashkenazi Jewish women (scenario 3). Thus, like PAR, ACR is useful for comparing the potential impact of strategies to prevent a single disease due to different exposures or due to the same exposure in different populations.

Unlike PAR, ACR is also useful for comparing the strategies to prevent different diseases.[2,4] For example, in contrast to scenario 1 (for breast cancer), an intervention that prevented all ovarian cancer due to the common exposure would prevent 4 ovarian cancers in an unselected population of 10,000 (scenario 4). Similarly, an intervention that prevented all ovarian cancer due to BRCA mutations in 10,000 Ashkenazi Jewish women would prevent 29 ovarian cancer cases (scenario 5). That is, a fully effective program to prevent ovarian cancer would prevent only half as many cancer cases as a fully effective program to prevent breast cancer in the same population (scenario 3). Thus, of all the summary measures considered in the table, only ACR provides useful comparisons of the potential community-level benefit across interventions, populations and diseases.

ACR helps to resolve a controversy about how to evaluate whether men or women are more susceptible to lung cancer due to smoking. Comparison of relative risks for smoking among men and women[6] to address this question does not allow for consideration of absolute risk. The comparison of absolute risk among smokers in men and women[7–10] can be affected by differences between men and women in the frequency of other causes of lung cancer. Instead, comparisons of both the sex-specific potential reduction in the lung cancer rate from smoking prevention efforts and, under simple assumptions about the joint effects of smoking and other risk factors, the biological effect of smoking are best made by comparing ACRs. The ACRs for a given level of smoking can be calculated by setting $p$ equal to 1 in the ACR formula; when $p$ equals 1, ACR simplifies to the risk difference.

ACR shares some of the shortcomings of PAR. ACR is sensitive to misspecification of the risk model, poor exposure assessment, and lack of reliable estimates of the required parameters in the setting of interest.[5,11,12] In particular, ACR requires measures of frequency of exposure, exposure-disease relationship and absolute risk of disease in the relevant setting. But it is far better to have a rough estimate of the appropriate parameter than a statistically superior estimate of a less relevant parameter. Furthermore, the actual impact of a preventive measure depends on the risk in individuals after the intervention, which may be less than or greater than the risk in the never exposed. Finally, just as no single parameter, such as OR, can capture the 2 dimensions of Se and Sp,[9] no single parameter can summarize the opportunity for prevention from any factor X.

What about Rockhill's[1] general point? We epidemiologists ritualistically declare in the final paragraph of a manuscript that our work will ultimately save lives or at least prevent disease. But, as Rockhill forces us to recognize, we seldom actually grapple with prevention. She urges us to consider a wider range of prevention measures applied at a community level. This proposal will encounter legitimate resistance, grounded in fear for the integrity and standing of epidemiology. Indeed, we have neither a specific mandate nor special qualifications for handling the value-laden cost-benefit tradeoffs inherent in evaluating prevention strategies. Less charitably, one might suspect that sheer habit, turf protection, and lack of imagination explain the gulf between epidemiologic practice and public health.

Some epidemiologists will join public health battles while others will scrupulously avoid them—but all need measures of impact of possible preventive actions. Rehabilitating ACR and other measures of burden of disease takes us a small step in the right direction.

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