From Patients to Policy
Population Intervention Effects in Epidemiology

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Abstract: Interest in implementation science and recent calls for consequentialist epidemiology urge epidemiologists to produce work more immediately applicable to public health practice. A clear vocabulary for such approaches is lacking. Here, we present a potential taxonomy of causal effects, distinguishing between “exposure effects” more relevant to patients and individuals; and “population intervention effects” more relevant to public health policy. We discuss this range of effects using figures and a simple numerical example. (Epidemiology 2017;28: 525–528)

Recent years have seen a marked increase in interest around the translation of research findings into public health policy and practice, often discussed under the heading of “implementation” or “program” science. Although most discussions of translating research into practice regard epidemiology as critical to that process, to date the role of epidemiology has been left largely implicit, with some exceptions. In this age of calls for more consequentialist epidemiology, it is vital to have common vocabulary with which to discuss different types of effect estimates, and their relevance to clinical practice on one hand and intervention planning on another. Here, we synthesize work by numerous authors to describe a range of contrasts that can be produced by epidemiologic analysis. For conceptual clarity, here we consider causal effects in a closed population with a binary, time-fixed, harmful exposure (equivalently, treatment); a dichotomous, time-fixed outcome which can be summarized as a risk; and no competing risks. Further, we largely concentrate on issues of internal validity, ignoring issues of generalizability and transportability. Finally, the main text is conceptual; formal definitions of effects are proposed in the Technical eAppendix.

SELECTED EPIDEMIOLOGIC EFFECTS

The Figure shows several possible contrasts in a single population under real and hypothetical exposure distributions; this Figure owes a debt of influence to Figures 1.1 and 4.1 in Hernán and Robins.

In Figure part A, we show an observed population as a circle in which a minority of individuals are exposed (shaded) and the remainder are unexposed (unshaded); the risk of the outcome is not indicated in the Figure. In Figure part B, we show what is sometimes called a population average causal effect (alternately, average causal effect, average treatment effect). The two contrasting exposure distributions in Figure B (all-exposed; none-exposed) are both counterfactual in the literal sense of not corresponding to the factual exposure distribution in the observed sample (Figure A). In part because neither exposure distribution in Figure B coincides with a population in the real world, all-none comparisons may not be directly applicable in setting policy.

In Figure part C, we show a population attributable average causal effect (hereafter population attributable effect), which compares disease risk in the observed population with the observed level of an exposure) with the risk that would be observed in the same population under a counterfactual exposure distribution in which 100% of the exposure was removed. The best known population attributable effect is the population attributable fraction; closely related quantities have been referred to by other names elsewhere. In passing we note that Greenland and Robins distinguished between the excess and etiologic fractions; here we are interested in their “excess” usage.
In contrast to population average causal effects (Figure B), population attributable effects require only a single counterfactual exposure distribution, as one of the two groups being compared is the observed population (Figure part A). Population attributable effects may therefore be more readily applicable to questions of population interventions, which seek to alter the (observed, factual) exposure distribution of the world to improve population health. Unlike the population average causal effect, however, population attributable effects depend on the population prevalence of the exposure, which may be a particular consideration in transporting a population attributable effect to an external target population.

The Figure (D) shows a generalized intervention average causal effect (hereafter, generalized intervention effect) which compares observed exposure distribution (again identical to the observed exposure in Figure part A) to a counterfactual in which there is less exposure. Because “no exposure” is a special case of “less exposure,” the population attributable effect (Figure part C) is a special case of the generalized intervention effect (Figure part D). Generalized impact (or attributable) fractions were described by several authors in the 1980s; the estimation of the quantity in complex data was explored by Greenland and Drescher, and can proceed either stochastically or deterministically.

A generalized intervention effect assumes that the intervention removes exposure with an equal probability among all participants (homogeneously with respect to participant characteristics). This is frequently not true: an intervention may incidentally succeed at removing a harmful exposure at a higher rate in one group (e.g., younger people) than another (older people), or may be deliberately targeted at a higher-risk group. In such a situation, the dynamic intervention average causal effect (hereafter, dynamic intervention effect) may be useful: the dynamic intervention effect compares the observed exposure distribution (Figure part A) to a counterfactual in which there are fewer exposed participants, allowing for heterogeneity in amount of exposure removed by the intervention within covariate-defined subpopulations. Figure part E shows a dynamic intervention effect: heterogeneity in the study sample is shown with the dotted line, and different amounts of exposure are removed on each side of the line. In general, for a fixed percentage of exposure removed (e.g., 20%) and a fixed intervention the generalized and dynamic intervention effects can be expected to differ if (i) there is heterogeneity of the causal effect of the exposure on the outcome by some set of covariates \(Z\), and (ii) the effectiveness of the intervention at removing the harmful exposure differs by \(Z\), either by design or happenstance (see Numerical eAppendix; http://links.lww.com/EDE/B182 for an example.) The generalized intervention effect can thus be thought of as a special case of the dynamic intervention effect, under at least one homogeneity assumption (or in which the set of covariates \(Z\) is empty). Similar methods and concepts under varying names have been described and

![Figure](image_url)
applied elsewhere. See Technical eAppendix; http://links.lww.com/EDE/B181 for discussion.

REMARKS

We remind the reader that for didactic purposes we are focusing on a binary, time-fixed, harmful exposure. With such an exposure, the population average causal effect (contrasting an entire population under all-exposed, all-unexposed conditions) is the default target of estimation in the bulk of the population health literature. Such a contrast was of primary interest to Neyman, and is estimated by the vast majority of randomized trials. In particular, an intention-to-treat analysis of a randomized trial with a dichotomous exposure frequently estimates this contrast for treatment assignment, while compliance-corrected (or as-treated) analysis estimates this contrast for treatment received. Such “all/none” effects are typically what are estimated in observational analysis as well, using the default tools: standard regression approaches estimate covariate-conditional population average causal effects, while inverse probability weighting generally estimates marginal population average causal effects in a manner equivalent to total population standardization.

The fact that population average causal effects are the result of a contrast in two counterfactual exposure distributions may mean that they have less immediate and direct applicability to questions of setting policy at the population level, differing from measures which compare the factual exposure distribution with a counterfactual one. In broad terms, we therefore consider the population attributable, and generalized and dynamic intervention effects to be population intervention effects (in that they are tied to potential interventions on real-world exposure distributions), while we might think of the population average causal effect as an exposure effect (in that it contrasts all-exposed to none-exposed). These designations have fuzzy boundaries, and may coincide in certain settings: a smoking cessation intervention (which might think of the population average causal effect as an exposure effect) may mean that they have less immediate and direct applicability to questions of setting policy at the population level, differing from measures which compare the factual exposure distribution with a counterfactual one. In broad terms, we therefore consider the population attributable, and generalized and dynamic intervention effects to be population intervention effects (in that they are tied to potential interventions on real-world exposure distributions), while we might think of the population average causal effect as an exposure effect (in that it contrasts all-exposed to none-exposed).

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REFERENCES


