

Commentary III

Lack of evolution of epidemiologic “methods and concepts”

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Zhang et al. (2004), while writing about the “evolution of some epidemiologic methods and concepts,” unwittingly illustrate the stagnancy of these. In particular, the still-prevalent commitment to “cohort study” and “case-control study” in etiologic research is evident from the very outset.

Different from what is definitional to a “cohort study” (Last 2001), the etiologic history associated with a case identified in its follow-up of the cohort (closed population) should be defined and documented as of this outcome, not as of the beginning of the subject’s follow-up. It should next be understood that for the thus-documented case series the referent is the population-time of the study cohort’s follow-up. For, the case series is interpretable as the source of rate numerator inputs in reference to this population-time only; this referent of the case series therefore constitutes the study base to which the empirical occurrence relation – for the outcome’s incidence density – refers. And as a final matter of liberation from “the cohort fallacy” (Miettinen 1999) in etiologic research, it should be understood that this case series needs to be coupled with a similarly documented base series, a fair sample of the (infinite number of) person-moments constituting the study base. Given the database formed by these two series, it remains merely to fit to it the logistic-regression counterpart of the designed object of study – of the logarithm of the outcome’s incidence density as a function of the etiologic history, conditionally on modifiers and non-modifier confounders, in a defined domain.

As for the “case-control study,” then, it should be understood for a start that the case series again serves as the source of rate numerator inputs; that it is, again, the rate numerator series. With this beginning of liberation from “the trohoc fallacy” (Miettinen 1999), the concern naturally is not to couple the case series with a “control group” but with a denominator series, a fair sample of the study base. The case series need not arise from follow-up of a directly-defined cohort. Instead, the

source population may be a directly-defined dynamic (open) population; or it may be definable only indirectly – as the catchment population of the directly-defined scheme of case identification. For both series the etiologic histories are defined, again, as of the time of the outcome (case occurring or not occurring), and the rest also proceeds as above – as always in *the* etiologic study. In it, the comparison never is between a “case group” and a “control group.” Instead, it always is between the index and reference segments of the study base. And it is only in reference to this contrast that the alternative to causality – confounding – can be understood.

It is of considerable note that this understanding of the etiologic study is key to understanding the intervention study as well. Cases are identified in a study base formed by a cohort’s follow-up; the associated intervention histories as of case occurrence are documented; a similarly documented sample of the study base is obtained; etc. My current course compendium, the precursor of my upcoming textbook, actually goes well beyond intervention research, even. The working title now is “Scientific medicine: essence and epistemology.” Its implicit overall message to my epidemiology colleagues is this:

Let us move beyond our traditional focus on the theory – concepts and principles – of merely etiologic research to concern for the theory of medicine, including the theory of the research that produces the knowledge base of scientific medicine; let us dedicate ourselves to such quintessentially “applied” research for the advancement of clinical as well as community medicine; and in it, let us be serious about object design – ultimately in terms of a regression function – before methods design, rejoicing in the consequent relevance of the research without concern for whether it still may be characterized as epidemiologic.

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