

The Fallacy of the Ecological Fallacy: The Potential Misuse of a Concept and the Consequences

ABSTRACT

Ecological studies have been evaluated in epidemiological contexts in terms of the "ecological fallacy." Although the empirical evidence for a lack of comparability between correlations derived from ecological- and individual-level analyses is compelling, the conceptual meaning of the ecological fallacy remains problematic. This paper argues that issues in cross-level inference can be usefully conceptualized as validity problems, problems not peculiar to ecological-level analyses. Such an approach increases the recognition of both potential inference problems in individual-level studies and the unique contributions of ecological variables. This, in turn, expands the terrain for the location of causes for disease and interventions to improve the public's health. (*Am J Public Health*. 1994;84:819-824)

Sharon Schwartz, PhD

Introduction

Epidemiology texts offer a consistent appraisal of ecological studies: they are crude attempts to ascertain individual-level correlations. The flaws in such studies limit their usefulness to "hypothesis generation," leaving the more esteemed process of "hypothesis testing" to individual-level data. The problems are generally attributed to the "ecological fallacy,"¹⁻⁸ a logical fallacy inherent in making causal inferences from group data to individual behaviors.^{9,10}

The consequences of this ecological fallacy are well documented. More than 40 years ago, Robinson demonstrated that the correlation coefficient between two individual-level variables is generally not the same as that between those same variables for aggregates into which the individuals are grouped.^{11,12} Many papers have examined this problem statistically, confirmed Robinson's findings, and suggested methods for making ecological and individual correlations more comparable.¹³⁻²⁰ Epidemiology texts have used these analyses to support their evaluation of ecological studies.

The use of the ecological fallacy to explain the discrepancy between individual and ecological correlations may have unintended consequences. Examining this issue from a different perspective—as a general validity problem—will show that the ecological fallacy, as often used, encourages three interrelated, fallacious notions: (1) that individual-level models are more perfectly specified than ecological-level models, (2) that ecological correlations are always substitutes for individual-level correlations, and (3) that group-level variables do not cause disease. We begin with a description of the validity framework and the definition of key terms.

A Proposed Validity Scheme

Cook and Campbell developed an analytic scheme to assess the validity of causal relationships.²¹ Two questions they pose are of salience here: (1) Given a statistically significant correlation between two variables, is it valid to assert a causal relationship between these two variables *as measured*? (2) Given a plausibly causal relationship between two variables as measured, what are the causal concepts involved in the relationship? The first question deals with internal validity and the second with construct validity.

Internal Validity

The essence of internal validity is accounting for third-variable alternative interpretations of presumed A-B relationships in which A represents the independent variable and B the dependent variable. It is precisely here that a source of noncomparability between an individual and an aggregate correlation of the same variables may arise. "In shifting from one unit of analysis to another, we are very likely to affect the manner in which outside and possibly disturbing influences are operating on the dependent and independent variables."^{22(p97)} As a result of the grouping operation, one may have

The author is with the Division of Epidemiology, School of Public Health, Columbia University, New York, NY.

Requests for reprints should be sent to Sharon Schwartz, PhD, Columbia University, School of Public Health, 600 W 168th St, 7th Floor, PH18-332: Epidemiology, New York, NY 10032.

This paper was accepted January 14, 1994.

Editor's Note. See related articles by Susser (p 825 and p 830) and Koopman and Longini (p 836) and editorial by Poole (p 715) in this issue.

controlled for the effects of other variables, making the ecological estimate less biased than the individual estimate,²³ or one may have included various confounding variables, making the ecological-level correlation more biased.²⁴ If a difference occurs between ecological- and individual-level correlations, the problem may be due to a failure to specify the correct model and not to an inherent logical fallacy in moving from individual to group correlations.

*Construct Validity**

However, discrepancies between individual and ecological correlations often remain after controlling for confounding variables.² To some extent, this may be due to further misspecifications, where other confounding variables are not taken into account. But there may be another problem as well. "The demystification of cross-level bias begins with the recognition that an aggregate variable often measures a different construct than its name-sake at the individual level."^{25(p560)} The construct referenced on the ecological level may be the context or social environment in which individuals live, distinct from the attributes of those individuals.²⁶⁻³⁰ Thus, poverty as an individual characteristic and poverty as a neighborhood characteristic may exert different, independent effects on health. Consequently, individual and aggregate correlations of this variable will be discrepant.

Internal validity and construct validity can therefore be used to explain disparities in correlations between individual- and ecological-level variables. We examine the benefits of doing so through a discussion of three assumptions associated with the ecological fallacy.

Evaluations of Ecological Studies Based on the Ecological Fallacy: Hidden Assumptions and Their Consequences

Assumption 1: Individual-Level Models Are More Perfectly Specified Than Ecological-Level Models

The problem of internal validity, confounding, is considered a particularly egregious fault in ecological studies.^{1-4,9} Indeed, the ecological fallacy is often defined as a problem of confounding. For example, Lillienfeld and Lillienfeld contend that ecological correlations "may suffer from an 'ecological fallacy', that is,

the two communities differ in many other factors and one or more of those may be the underlying reason for differences in their observed mortality or morbidity."^{3(p48)} But to conclude that differences in relevant third-variable effects at the ecological- and individual-levels of the same variable constitute an ecological fallacy, a weakness in ecological studies, requires one to assume that individual-level models are more accurately specified than ecological-level models. This is often, but not inherently, true. If individual- and ecological-level analyses are both based on historical records, information necessary for including confounding variables may be extant for the aggregate but not for the individual level, allowing better specification of the ecological study. For example, employee records may have less information on smoking and dietary habits than sales records for company towns. Similarly, certain confounding variables intrinsic to survey research, such as response bias, recall bias, and naysaying, may be avoided in ecological studies. In particular, when the variables of interest probe sensitive issues, ecological-level data may be more accurate. (E.g., sales of alcoholic beverages or rates of abortions may be more useful than statements of alcohol use or an individual's abortion experience.) Additionally, the grouping process itself may control for some confounding variables not controlled for in an individual-level model.^{9,31,32}

In practice, it may be that confounding usually poses a more intractable problem for ecological- than for individual-level studies. But this is due to the greater reliance on secondary data and proxy measures in ecological studies, not to any problem inherent in ecological studies. An inability to control confounding variables occurs under these conditions, no matter what the unit of analysis. However, the view that ecological studies may be used for only hypothesis generation or evaluation of interventions, while not generally supportable, is valid under certain conditions. If it is suspected, in a specific instance, that an ecological correlation will yield a biased estimate of an individual correlation that is perfectly specified, due solely to problems of internal validity (i.e., problems of confounding variables), the solution to the problem would be a careful respecification of the model. If the potentially confounding factors are unknown or unmeasured, the ecological correlation is useless. In such a case, the ecological correlation is merely a poor substitute for

an individual-level correlation. Although it may yield some hypotheses for exploration, it will be of little real help because confounding may alter not only the magnitude of the correlation coefficient (or other measure of association) but the direction of the effect as well.

Seeing this confounding problem in terms of internal validity raises a number of questions to be evaluated on a case-by-case basis. Are internal validity problems the only possible source of discrepancy between a particular ecological correlation and a correlation of these same variables at the individual level? If so, what are the sources of confounding that are likely to be problematic at each level? At what level of analysis can these confounding problems best be controlled? The answers will not always favor the individual-level study.

Assumption 2: Ecological Models Are Substitutes for Individual-Level Models

As we have seen, ecological and individual correlations may be discrepant not only because of internal validity problems but also because of construct validity problems. That is, the aggregated variable may measure a different construct than its namesake on the individual level. This source of discrepancy was not mentioned in any of the epidemiological textbooks reviewed^{1-5,10**} despite considerable discussion in other fields.^{25,32,33} The reason for this lies, perhaps, with an assumption Robinson makes: that researchers undertake ecological studies only when individual-level data are unavailable and that the individual-level analysis is their real concern.¹¹ This assumption is accepted in the main epidemiological texts and is implicit in discussions of the ecological fallacy.²⁻¹⁰

For example, Morgenstern writes:

The key feature of ecological data relative to cohort data is the lack of information about the joint distribution of the study factor and the disease within each group. . . . In ecologic analy-

*This use of construct validity is an expansion of the concept as developed by Cook and Campbell,²¹ who explicitly argue that it refers only to constructs at the same level of reduction. An analysis of Cook and Campbell's position and the development of the reinterpretation used here are available from the author.
**While none of the epidemiological texts refer to construct validity, Morgenstern's article evaluating ecological studies in epidemiology does.⁹ However, Morgenstern views macro-social or contextual effects only as confounding variables and not as causal variables in their own right.

sis the independent variable (X) is the proportion of exposed subjects within the group and the dependent variable (Y) is the rate (or risk) of disease.^{9(p1337)}

This is sometimes the case but only if the ecological variable is an aggregate variable rather than a characteristic of a group and if there are no contextual effects—that is, only when the ecological- and individual-level variables measure the same construct. But when the ecological-level variable measures some group property, it is no longer the proportion of exposed subjects that is the independent variable.^{26–30} Rather, the proportion of subjects with a certain factor of interest is itself the exposure. In this case, the ecological study is not a substitute for an individual-level study but an examination of unique variables not measurable on the individual level.^{34,35} The neglect of this possibility leads to two interrelated problems: (1) a failure to recognize the ecological fallacy in individual-level studies, and (2) a failure to recognize the full range of cross-level studies.

Ecological fallacies in individual-level studies. Epidemiological discussions frame the issue of ecological inference problems in terms of a lack of consistency between the measure of association for the independent (A) and dependent (B) variables at the individual level and the measure of association for the independent (A') and dependent (B') variables at the ecological level. Therefore, the measure of association is the focus of analysis. But when the issue is framed in terms of construct validity, it becomes apparent that there are two other points of potential disagreement between ecological- and individual-level correlations. A may not equal A' and B may not equal B'. This conceptualization helps to clarify the logical fallacy involved in cross-level inference and allows one to think more fully about levels of analysis and their relationships.

As a logical fallacy, the ecological fallacy is a problem of construct validity and not of a measure of association. Aristotle refers to it as “the fallacy of division.”³⁶ It is a problem of confusing the group with the members of that group, of assuming that because a group has a certain characteristic the members of that group also have that characteristic. Zito³⁷ provides an illuminative example. A hung jury is a jury that is indecisive, it cannot decide whether the accused is guilty or innocent. However, to deduce that the members of such a jury are indecisive would be absurd. Members of a hung jury are very decisive, so much so that they can

not be persuaded to change their mind. Attributing to the members of this group the characteristic of that group (indecisiveness) is thus a case of the ecological fallacy. A construct validity approach raises the awareness that the ecological fallacy is a ubiquitous problem and may occur with individual as well as ecological-level data.

Note, for example, the following comment in one epidemiological text: “In most epidemiologic contexts as opposed to sociologic or anthropologic contexts, one is interested in drawing inferences about disease etiology in *individual persons* [emphasis added].”^{2(p208)} But epidemiology is not concerned with disease etiology in individual persons. As defined by Susser et al., “Epidemiology (‘epi’ upon, ‘demos’ the people) is the science concerned with the health of *populations or communities* [emphasis added].”^{38(p16)} Indeed,

the empirical analysis of sample data, whether it is aggregate or individual, cannot be used to study the behavior of individuals. The objective of most empirical analyses is to determine the independent effects, *in a probabilistic way*, of some households or individuals possessing that characteristic [emphasis added].^{24(p99)}

For example, if an experimental vaccine trial provided evidence that 20% of the vaccinated and 50% of the unvaccinated people contracted the disease, one would conclude that there is an association between getting the vaccine and not getting the disease. In fact, one would conclude that there is probably a causal relationship between avoiding the disease and being vaccinated. Yet for any particular vaccinated person it would be a logical fallacy—indeed, an ecological fallacy—to suggest from these data alone that he or she did not contract the disease because of the vaccination.³⁹

This logical fallacy is ubiquitous when proxy measures are used. For example, in an individual study of the relationship between an exposure and disease, controlling for diet may be desirable. However, data on dietary habits may be difficult to obtain, and another variable collected for each individual—perhaps educational level—may be used as a proxy measure for diet. Doing so involves the ecological fallacy, however, because it implicitly assumes that since, as a group, people with different educational levels exhibit different dietary habits, an individual within a specific educational group will exhibit the dietary pattern of that

group. This is particularly problematic because it leads to significant measurement error and therefore to an underadjustment for this control variable. Thus, one may erroneously conclude that the exposure is associated with the disease controlling for diet when, in fact, diet has not been controlled. Examining the ecological fallacy in terms of construct validity has the advantage of increased vigilance in the search for greater validity in all studies.

The full range of cross-level studies. Viewing ecological studies as substitutes for individual-level studies leads to another consequence. There is a tendency to dichotomize studies as either ecological, in which case the independent and dependent variables are aggregated individual-level variables, or nonecological, in which case the independent and dependent variables are individual level. For example, the usefulness of ecological studies has been limited as follows: “If broad social or cultural processes are of interest then the individual may not be the most appropriate unit of analysis, since inferences are to be drawn about whole societies rather than about individuals.”^{2(p208)} Thus, there is a general conclusion that ecological studies cannot be used to make inferences about individual phenomena or behaviors.^{2,10} But social and cultural factors and processes do not have effects solely on whole societies but on individuals as well. By analyzing ecological studies in terms of an ecological fallacy—a problem in measures of association rather than in terms of the construct validity of component variables—the full range of potential cross-level relationships is attenuated. For example, both the independent and dependent variables in a study can be group characteristics that cannot be measured by the aggregation of individual behaviors (e.g., the relationship between level of industrialization and number of hospitals per capita). A second possibility is a study of contextual effects in which the focus of interest is the relationship between an individual’s behavior and the group context in which that behavior exists. In this instance, the independent variable may be a group-level variable and the dependent variable may be an individual-level one (e.g., the effects of living in a minority neighborhood on infant mortality). There can also be structural analyses that focus on the group but make reference to differentiated roles of individuals that interrelate to form a group’s internal structure (e.g., an examination of the social network pat-

terns of immune and vulnerable individuals that potentiate herd immunity).^{33,40} In this case, the independent variable is individual level and the dependent variable is group level. Thus, there are many study designs that are neither purely ecological nor purely individual level.

One example of the consequence of viewing studies as acting on only one level is the analysis of Durkheim's *Suicide*⁴¹ in epidemiological contexts. We examine this example closely because it is often cited as the exemplar of the ecological fallacy.^{9,10} One analysis in an epidemiology textbook is as follows:

He [Durkheim] found, on the average, [that] provinces with greater proportions of Protestants had higher suicide rates and that provinces with greater proportions of Catholics had lower suicide rates. Durkheim concluded from these data that Protestants are more likely to commit suicide than are Catholics. While the conclusion may be true, the causal inference is not logically correct, because it may have been Catholics in predominantly Protestant provinces who were taking their own lives. This logical flaw, called the ecological fallacy (Selvin, 1958), results from making a causal inference about an individual phenomenon or process (e.g., suicide) on the basis of observations of groups.^{10(p79)}

In fairness to Durkheim, it should be noted that he based his conclusions on ecological-level correlations in tandem with an examination of suicide rates among Catholic and Protestant individuals within provinces.^{41,42*} However, it is worthwhile to examine this ecological fallacy with the assumption that these facts were correct.

In assessing plausible alternative explanations, Morgenstern suggests that minority status may be related to a propensity for suicide.⁹ It may be that the higher suicide rates in Protestant countries are accounted for by the suicides of the Catholics who have minority-group status in such places. Regardless of the merits of this hypothesis, it could not be

*The authors quote the Selvin article on Durkheim as the source for the term *ecological fallacy*.⁴¹ Indeed, this does appear to be the first use of the term in the literature, although Menzel³⁴ and Thorndike⁴³ both referred to the ecological correlation problem as a fallacy prior to this and many sources erroneously cite Robinson as coining it by expression.^{5,27,28} However, while Selvin does say that Durkheim is at points guilty of the ecological fallacy, he explicitly states that Durkheim recognized this problem and solved it by looking at individual-level data when he could—for example, in the religion issue.^{41(p608)}

tested by an individual-level study. A comparison of suicide rates among individuals of different religious persuasions could reveal only a higher or lower rate for Catholics versus Protestants. Only in conjunction with the aggregate variable of “proportion Catholic” and “proportion Protestant”—that is, only in conjunction with contextual analysis—could this hypothesis be tested. Furthermore, according to this definition, Morgenstern's alternative hypothesis would also constitute an ecological fallacy.⁹ Causal inferences about an individual process, suicide, would be made from observations of groups. Thus, this alternative hypothesis also suggests that a group variable influences behaviors carried out by individuals.

Indeed, Durkheim contends that suicide is a social rather than an individual phenomenon,⁴⁴ for although it is an act committed by an individual with idiosyncratic reasons for its commission, variations in suicide rates are caused by social factors. In this case, Durkheim posits the effect of living in a Protestant area as a sociological phenomenon, related to the rules governing attitudes and behaviors that influence the propensity to commit suicide. Examining Durkheim's study in terms of construct validity makes it clear that an ecological fallacy would exist if it were assumed that the variable measured—living in a Protestant country—was equivalent to the individual variable—being Protestant. There is no ecological fallacy in relating observations of groups to behaviors performed by individuals. Thus, while neither studies of groups of groups nor studies of groups of individuals can explain the behavior of a particular individual, they can both help to explain behaviors performed by individuals.

Assumption 3: Only Characteristics of Individuals Cause Disease

The use of the ecological fallacy in epidemiology also fosters a dismissal of social variables as causal factors in disease. First, as seen above, it leads to a consignment of sociological and anthropological studies to examining impacts on whole societies,² and it denies that ecological variables can affect individual processes.²⁸ Second, it reinforces an assumption that aggregated variables are substitutes for individual-level variables. Under such an assumption, the potential etiological influence of aggregate-level variables, distinct from the effects of the same measures on an individual level, would not be considered. Usually, this assumption is implicit in statements about

not making causal inferences about individual phenomena on the basis of observations of groups. Sometimes, however, this denial is made explicit, as in Rothman's statement that “social class . . . itself is presumably causally related to few if any diseases but is a correlate of many causes of disease.”^{4(p90)}

This evaluation is perplexing because the concept of cause in epidemiology does not preclude and often times explicitly includes non-individual-level variables. As Susser suggests, “A determinant can be any factor . . . [that] brings about change for better or worse in a health condition.”^{45(p3)} Such social factors as socioeconomic status and social disorganization surely lie within the purview of this definition.

It may be that the prominence of the “germ theory” paradigm has reinforced a focus on individual factors. While this model is clearly useful, it has limitations because “some health problems may be more parsimoniously understood and more efficiently controlled by viewing them as products of community dynamics.”^{35(p111)} For example, decreasing economic disparity may decrease the rates of a wide range of physical and psychiatric disorders.

The perception of a longer and more indirect chain of causation for social, ecological-level variables may also prompt a neglect of such factors.⁴⁵ But the length and complexity of the causal chain does not determine the importance of the cause. For all variables, “behind the ‘intimate’ cause of disease—disease agent, stands the ultimate causal factors of the social and physical environment providing the linkages between agent and host.”^{46(p11)} The “intimate” and “ultimate” causes each deserve attention, and neither negate the validity of the other.⁴⁷ Indeed, “the idea of cause has become meaningless other than as a convenient designation for the point in the chain of event sequences at which intervention is most practical.”^{48(p181)} No matter what the variable of interest or the level of analysis, unless cause is viewed in terms of a particular purpose, the problem of infinite regress ensues. As Zito writes:

When we begin to supply intervening and antecedent variables to a model . . . there is no end to such a series of questions. We can continuously descend to lower and lower orders of questioning and higher and higher levels of abstraction.^{37(p143)}

By viewing discrepancies between ecological and individual studies as valid-

ity issues, it becomes apparent that all causes are indirect and that all variables can be viewed as either ecological or individual level, depending on one's perspective. Robinson contended that, unlike social groups, individual persons constitute an indivisible entity.¹¹ But this is true only if one views the individual as the level of analytic interest. From another viewpoint, the individual is an ecological-level variable, an aggregated measure of body parts that become diseased. For example, smoking, an activity performed by individuals, is generally viewed as a cause of lung cancer although the chain of causation is long and indirect. However, a molecular biologist concerned with a lower order of pathogenesis would not consider smoking a cause of interest, for the smoking behavior of the individual is at a level of analysis too remote from his or her concern. According to Susser et al.:

Investigators conceptualize variables and abstract them from a given ecological setting within a limited frame of reference. . . . The choice is the outcome of the needs and consciousness of an investigator in a particular situation, but on logical grounds it is an arbitrary procedure.^{38(p43)}

Thus, discussions of cause in epidemiology include both the social, ecological level and the individual level as valid arenas of causal inquiry. However, the manner in which Robinson's observations have been adopted tends to hinder a serious consideration of social factors in disease etiology.

Conclusions

In 1979, Kasl suggested that epidemiologists need to develop guidelines for comparing ecological analyses with studies of individuals.⁴⁹ This paper posits that the concept of the ecological fallacy, the framework used to juxtapose and contrast ecological and individual studies, cannot fully address these issues. A validity approach, examining all studies in terms of internal and particularly construct validity problems, may prove a useful addition to understanding cross-level inference. This perspective would suggest, in agreement with the ecological fallacy perspective, that ecological studies cannot usually be used as substitutes for individual correlational studies. However, it does not indicate that ecological studies are etiologically useless, for they are not viewed as crude estimates of individual-level studies. Rather, ecological variables are necessary to examine structural, con-

textual, and sociological effects on human behavior and disease development.

One example of a contextual effect that has important public health consequences is the relationship between job characteristics and myocardial infarction. Karasek and colleagues found that individuals in jobs characterized by high levels of psychological demands coupled with low decision latitude are at increased risk of myocardial infarction.⁵⁰ These job characteristics, while clearly operating through mechanisms that influence individuals, are not reducible to individual characteristics. They are variables describing the psychosocial work environment that has an influence on the workers' health. The most effective intervention to reduce this risk would be to change the organization of these occupations—an intervention at the ecological rather than the individual level.

The question of disease etiology is complex. It is likely that a multitude of causes is involved in the development of any particular disease. Where in the causal chain, among the myriad of variables, one chooses to examine and ascertain causation is often a question of where intervention is most efficacious. That, in turn, is often a political and not a scientific issue. An examination of the full range of variables potentially involved in disease etiology, with a synthesis of findings from all levels of analysis, provides the best opportunity for a full understanding of disease etiology. □

Acknowledgments

This work was supported by National Institute of Mental Health grant T32MH13043.

I would like to thank Janice Husted, Jennifer Kelsey, Bruce Link, Steve Ng, Karen Raphael, and Elmer Struening for their helpful comments on earlier drafts of this manuscript.

References

1. Mausner JS, Kramer S. *Epidemiology—An Introductory Text*. Philadelphia, Pa: WB Saunders; 1985.
2. Kelsey J, Thompson DW, Evans AS. *Methods in Observational Epidemiology*. New York, NY: Oxford University Press; 1986.
3. Lillienfeld AM, Lillienfeld DE. *Foundations of Epidemiology*. New York, NY: Oxford University Press; 1980.
4. Rothman KJ. *Modern Epidemiology*. New York, NY: Little Brown; 1986.
5. Friedman GD. *Primer of Epidemiology*. New York, NY: McGraw-Hill; 1974.
6. Fletcher RH, Fletcher SW, Wagner EH. *Clinical Epidemiology: The Essentials*. Baltimore, Md: Williams & Wilkins; 1988.
7. Abramson JH. *Making Sense of Data*. New York, NY: Oxford University Press; 1988.
8. Selvin S. *Statistical Analysis of Epidemio-*

logic Data. New York, NY: Oxford University Press; 1991.

9. Morgenstern H. Uses of ecologic analysis in epidemiologic research. *Am J Public Health*. 1982;72:1336–1344.
10. Kleinbaum DG, Kupper LL, Morgenstern H. *Epidemiologic Research: Principles and Quantitative Methods*. London, England: Lifetime Learning; 1982.
11. Robinson WS. Ecological correlations and the behavior of individuals. *Am Sociol Rev*. 1950;15:351–357.
12. Hammond J. Two sources of error in ecological correlations. *Am Sociol Rev*. 1973;38:764–775.
13. Meckstroth TW. Some problems in cross-level inference. *Am J Political Sci*. 1974;18:45–66.
14. Goodman LA. Ecological regressions and the behavior of individuals. *Am Sociol Rev*. 1953;18:663–664.
15. Goodman LA. Some alternatives to ecological correlation. *Am J Sociol*. 1959;64:610–625.
16. Irwin L, Lichtman AJ. Across the great divide: inferring individual level behavior from aggregate data. *Pol Methodology*. 1976;3:411–439.
17. Iversen G. Recovering individual data in the presence of groups and individual effects. *Am J Sociol*. 1974;79:421–434.
18. Scheuch EK. Social context and individual behavior. In: Dogan M, Rokkan S, eds. *Social Ecology*. Cambridge, Mass: Massachusetts Institute of Technology; 1969:133–155.
19. Marx JH, Suchman EA, Heller DB. A systematic framework for relating the behavioral sciences to the health care fields. *Inquiry*. 1967;4:48–58.
20. Hannan M, Burstein L. Estimation from grouped observations. *Am Sociol Rev*. 1974;39:374–393.
21. Cook T, Campbell D. *Quasi-Experimentation: Design and Analysis Issues for Field Settings*. Boston, Mass: Houghton Mifflin; 1979.
22. Blalock H. *Causal Inferences in Nonexperimental Research*. Chapel Hill, NC: University of North Carolina Press; 1964.
23. Gove WR, Hughes M. Reexamining the ecological fallacy: a study in which aggregate data are critical in investigating the pathological effects of living alone. *Soc Forces*. 1980;58:1157–1177.
24. Hanushek E, Jackson JE, Kain JF. Model specification, use of aggregate data and the ecological correlation fallacy. *Pol Methodology*. 1974;1:89–107.
25. Firebaugh G. A rule for inferring individual-level relationships from aggregate data. *Am Sociol Rev*. 1978;43:557–572.
26. Blau P. Structural effects. *Am Sociol Rev*. 1960;25:178–193.
27. Blau J, Blau P. The cost of inequality: metropolitan structure and violent crime. *Am Sociol Rev*. 1982;47:114–129.
28. Davis JA, Spaeth JL, Huson C. A technique for analyzing the effects of group composition. *Am Sociol Rev*. 1961;26:215–225.
29. Farkas G. Specification, residuals and contextual effects. *Sociol Methods Res*. 1974;2:333–363.
30. Meltzer L. Comparing relationships of individual and average variables to indi-

- vidual response. *Am Sociol Rev.* 1963;28:117-123.
31. Lichtman A. Correlation, regression and the ecological fallacy: a critique. *J Interdisciplinary Hist.* 1974;4:417-433.
 32. Langbein LI, Lichtman AJ. *Ecological Inference.* Beverly Hills, Calif: Sage; 1978.
 33. Riley MW. *Sociological Research: A Case Approach.* New York, NY: Harcourt, Brace and Work; 1963.
 34. Menzel H. Comment on Robinson's "ecological correlations and the behavior of individuals." *Am Sociol Rev.* 1950;15:674.
 35. Catalano R. *Health, Behavior and the Community: An Ecological Perspective.* New York, NY: Pergamon Press; 1979.
 36. Wolf A. *Textbook of Logic.* New York, NY: Collier; 1938.
 37. Zito G. *Methodology and Meanings: Varieties of Sociological Inquiry.* New York, NY: Praeger; 1975.
 38. Susser M, Watson W, Hopper K. *Sociology in Medicine.* New York, NY: Oxford University Press; 1985.
 39. MacMahon B, Pugh TF. *Epidemiology: Principles and Methods.* Boston, Mass: Little Brown; 1970.
 40. Lazarsfeld PF, Menzel H. On the relation between individual and collective properties. In: Etzioni A, ed. *A Sociological Reader on Complex Organizations.* New York, NY: Holt, Rinehart and Winston; 1969:499-516.
 41. Durkheim E. *Suicide.* New York, NY: Free Press; 1951.
 42. Selvin H. Durkheim's suicide and problems of empirical research. *Am J Sociol.* 1958;63:607-619.
 43. Thorndike EL. On the fallacy of imputing the correlations found for groups to the individuals or smaller groups composing them. *Am J Psychol.* 1939;52:122-124.
 44. Durkheim E. *Rules of the Sociological Method.* New York, NY: Free Press; 1959.
 45. Susser M. *Causal Thinking in the Health Sciences.* New York, NY: Oxford University Press; 1973.
 46. Coe R. *Sociology of Medicine.* New York, NY: McGraw Hill; 1978.
 47. Bahnson CB. Epistemological perspectives of physical disease from the psychodynamic point of view. *Am J Public Health.* 1974;64:1034-1039.
 48. Helman C. *Culture, Health and Illness.* Bristol, England: Wright; 1984.
 49. Kasl SV. Mortality and the business cycle: some questions about research strategies when utilizing macro-social and ecological data. *Am J Public Health.* 1979;69:784-788.
 50. Karasek RA, Theorell T, Schwartz JE, Schnall PL, Peiper CF, Michela JL. Job characteristics in relation to the prevalence of myocardial infarction in the US Health Examination Survey (HES) and the Health and Nutrition Examination Survey (HANES). *Am J Public Health.* 1978;910-919.

Call for 1993 Student Papers on Injuries

The Southern California Injury Prevention Research Center announces the William Haddon, Jr, competition for the best student paper on injury. Papers written during the calendar year 1993 or the current 1993/94 academic year will be accepted. Papers must focus on public health aspects of *intentional* injury (homicide, suicide, sexual assault, battering, child abuse, etc.) or *unintentional* injury (motor vehicle crashes, falls, drownings, burns, etc.). Priority will be given to original scientific contributions that address injuries in ethnic or racial minorities, the poor, or other traditionally under-served populations.

Three awards are anticipated in the amounts of \$500, \$250, and \$150 for manuscripts that demonstrate original approaches in injury research, analyze new data, address emerging problems, or emphasize findings with countermeasure potential. The award will be made directly to the student.

The 1992 award winners were Alexandra T. Clyde, MA, Harvard School of Public Health, "Seat Belt Use, Insurance Status and Hospital Bad Debt," and Caroline Kramer, The School of Allied Health Professions, SUNY at Stony Brook, "Adequacy of Death Certificate and Hospital Discharge Summary Information for Determining Injury Mortality/Morbidity Patterns."

Only unpublished manuscripts will be considered. Papers

under journal review but not yet accepted will also be considered. (The student must be able to include acknowledgment of this award in the final published version.) This award is separate from awards promoted by the Injury Control and Emergency Medicine section of the American Public Health Association. Submission to APHA's student paper competition does not preclude submission for this award. Manuscripts should be prepared according to *American Journal of Public Health* style guidelines.

In addition to the manuscript, a letter from the student's advisor should be submitted that verifies that the work and writing was conducted by the student who has been enrolled in graduate training since January 1993 and/or during the 1993/94 academic year. Full acknowledgment of the student's primary role in the work should be reflected through his or her position as first author of the paper. All submissions must include the student's name, address, and daytime telephone number. All materials must be submitted in triplicate and postmarked by July 31, 1994.

Please send submissions to Jess F. Kraus, PhD, Southern California Injury Prevention Research Center, UCLA School of Public Health, 10833 Le Conte Ave, Los Angeles, CA 90024-1772. Inquiries may be directed to Renee Goetz, MSW, at the same address, telephone (310) 206-4115.