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QUANTITATIVE METHODS AND CAUSAL INFERENCE IN MEDIA EFFECTS RESEARCH

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Although media effects can be (and are) studied from a variety of methodological perspectives, the application of quantitative methods has dominated media effects research since Schramm's (1957) early investigation into journalism research (see also Kamhawi & Weaver, 2003; Lowry, 1979). A recent systematic review of methods used by media effects scholars (Potter & Riddle, 2007) found that 71% of all studies published in 16 prominent mass communication journals between 1993 and 2005 used quantitative methodologies, with in-class surveys (32%) and laboratory experiments (29%) being the most popular methods to study media effects. Other quantitative methods frequently used by media effects scholars include field experiments, quantitative content analysis, and the analysis of archival (secondary) data (Frey, Botan, & Kreps, 2000). This bias toward the use of quantitative methods in media effects research continues to be debated in the field (see Bryant & Cummins, 2007, for a discussion), and it is not our purpose to resolve this debate. Nor is it our purpose to review each of the quantitative methods used to study media effects, and their relative strengths and weaknesses,

central to the pursuit of media effects research from a quantitative research perspective, that of causal inference.

The basic conception of a cause-effect relationship between media stimuli and audience-related outcomes is the defining feature of theories that fall into the "effects tradition" within mass communication research (Littlejohn & Foss, 2008). Indeed, there is a strong expectation from various stakeholders—policymakers, educators, parents, journalists, external funders, scientists in other disciplines, and media consumers themselves—that research findings about media effects be articulated in clear causal terms to the extent possible (Hornik, 2002; Nabi, 2007). Further, in recent years, with the mathematical advances in the modeling of causal relationships, the study of causation and causal inference has seemingly matured in many related fields (psychology, sociology, political science, and epidemiology; Pearl, 2000), developing into an integrative force that has the potential to bring together diverse fields of scientific inquiry.

However, despite the centrality of causation to media effects research, little systematic attention to causation considerations has been given in the field beyond the standard treatment of causation in basic research methods textbooks. This is not to say that media effects scholars have not been concerned with issues of causal inference. Work critical of current research practices that limit causal inference in media effects research, whether focusing on issues of sampling (e.g., Long, Slater, Boiarsky, Stapel, & Keefe, 2005), measurement of key constructs (e.g., Fishbein & Hornik, 2008), or hypothesis testing (e.g., Holbert & Stephenson, 2003; Levine & Hullett, 2002), has been published periodically in communication journals. But such sporadic efforts have been for the most part geared toward increasing the degree of rigor with which media effects researchers engage questions about causal relationships rather than a concentrated, systematic effort to tackle this important issue. Therefore, our primary goal is to begin conceptualizing some analytical tools for guiding the design and evaluation of causally valid media effects research. We do so with an eye toward the key challenges inherent in the study of media effects as we understand them and with the intention of stimulating a much-needed disciplinary conversation about ways to rise to these fundamental challenges in order to take media effects research to the next level.

We begin this chapter with a brief discussion of causation and explanation from a social scientific perspective and describe the traditional approach to causal inference from quantitative data. We then explain why this traditional approach is largely inadequate for addressing causality considerations in media effects research. Next, we provide a short overview of the key challenges to causal inference in media effects research: (1) the multilevel nature of media effects, (2) the dynamic nature of media effects, (3) the complexity of causal chains, (4) the measurement of key constructs, and (5) audience activity. We conceptualize potential threats to causal inference posed by each challenge in terms of logical fallacies and, where appropriate, point to some useful research procedures and/or quantitative applications that may mitigate them. We conclude that (1) more careful explication of mechanisms and processes of media effects and (2) better matching of the methods employed to the complexity of the phenomena under investigation will greatly enhance researchers' ability to address issues of causal inference and facilitate better integration of theory and research on media effects.

◆ *Causation and Causal Inference*

CAUSATION AND EXPLANATION IN THE SOCIAL SCIENCES

Although most people possess an intuitive understanding of causality as it appears in

everyday life, scholars and philosophers, at least as far back as Aristotle, have been debating the notion of causation and the best ways to describe and assess it. Classic conceptions of causation identified agency and manipulation as the forces underlying causal relationships between phenomena (e.g., the earthquake was caused by God). This approach is best exemplified by the logic and language of experiments, where the researcher (agent) creates an intervention in order to produce (manipulate) a certain outcome. The key features of causation in this perspective are contiguity (observed covariation of cause and effect) and temporal precedence (the cause preceding the effect in time). In contrast, modern theories of causation, dating back to Scottish philosopher David Hume, understand causal relationships in terms of mechanisms and capacities that exist naturally (e.g., earthquakes are caused by plate tectonics). For Hume, the *regularity* of the association between causes and their effects is a key feature of causation. This rationale suggests that causal laws can be discovered through empirical observations, and thus justifies the observational (or nonexperimental) approach to research in which the researcher observes (rather than manipulates) naturally occurring relationships among variables (Pearl, 2000).

Hume's widely accepted idea that logical causal claims are empirical rather than analytical (that is, they stem from people's sensory experience with the world rather than from abstract ideas) gave rise to a concern about the type of evidence needed to make legitimate claims about cause-effect relationships. Some, like the 19th-century philosopher John Stuart Mill, were concerned about drawing causal inference from accidental (or chance) regularities, emphasizing that causation is not the same as correlation. Mill's thought in particular popularized three requirements for inferring cause-effect relationships: (1) evidence of covariation between the presumed cause and effect; (2) evidence of the temporal precedence of the cause; and (3) evidence ruling out alternative interpretations for a possible cause-and-effect connection (Cook & Campbell, 1979). Mill's approach to dealing with possible spuriousness of causal relationships emphasized the importance of multiple different replications (i.e., observing the same causal relationship for different people, in different places, and at different times) and the design of crucial experiments that can offer a decisive empirical test of two or more alternative causal explanations (see also Popper, 1959; Stinchcombe, 1968). In their authoritative text on the topic, Cook and Campbell (1979) categorized the universe of alternative causal explanations into four threats to the validity of causal inference: internal validity, external validity, construct validity, and statistical conclusion validity. Simply put, one may infer a cause-effect relationship if he or she can demonstrate that (1) no other causal mechanism accounts for this relationship (internal validity); (2) the observed causal relationships can be generalized to and across alternate measures of cause and effect, and across different types of units, settings, and times (external validity); (3) the way variables are observed (measured) empirically fits tightly with their theoretical meaning (construct validity); and (4) conclusions about causal relationships in a population are drawn from representative and unbiased samples (statistical conclusion validity). Their framework suggests that a potentially large number of alternative hypotheses must be falsified before a causal claim can be considered valid.

David Lewis's counterfactual approach to causation (see Winship & Morgan, 1999) greatly simplified the task of eliminating alternative causal explanations. The counterfactual logic asks what would have happened if a certain premise were true. For example, what would have been the effect on a person had that person received a treatment for cancer rather than being denied one. The counterfactual claim assumes a causal relationship between the treatment and effect (if X, then Y). Hence,

evidence supporting the counterfactual claim (if no X, then no Y) also supports that causal claim. The key to this approach is the ability of the researcher to create a counterfactual condition that is identical (or at least very similar) to the condition under which the actual effect is observed, with the exception that X is not present. This is achieved through controlled experiments (where subjects are randomized to either an experimental or control group) or by statistical means such as the use of propensity scores (see Yanovitzky, Hornik, & Zanutto, 2008). Accordingly, classic experimental designs (or more specifically, the use of a control group and random assignment to treatment or control) became the gold standard for establishing causation whereas observational studies are perceived to be more vulnerable to spurious causal inference because temporal precedence is difficult to establish (as nothing is manipulated) and there is no way to know whether those cases that get the treatment and those that do not differ from one another in other ways (the problem of selection bias).

In summary, the traditional approach to causation in the social sciences is inherently deterministic in that it assumes a lawlike relationship between causes and effects where the same initial conditions inevitably produce the same outcome. Further, it views causal inference as contingent upon sound research design choices, such as experiments incorporating a control group and random assignment of units to conditions, a probability sampling scheme, reliable and valid measures, and statistical guards against chance occurrences of relationships in the population (see Cook & Campbell, 1979).

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With one notable exception, media effects researchers' understanding of causal inference conforms to the traditional approach to causation described in the preceding paragraphs. This is evidenced by the fact that controlled experiments are frequently used in the field and that researchers employing observational studies typically limit themselves to correlational (rather than causal) hypotheses. The notable exception is the tendency of many to confuse causal inference with statistical inference. As demonstrated previously, causal inference is inherently a function of the research design; it cannot be derived or inferred from statistical claims alone. Thus, when researchers claim to have supported their causal hypotheses simply by virtue of rejecting the null statistical hypothesis (which is, following Cook and Campbell, only one type of possible threat to the validity of causal claims), they are stepping beyond the bounds of their own evidence.

Beyond that, however, lies the far more fundamental question about the extent to which the traditional social scientific approach to causation and causal inference is adequate for making legitimate causal claims about media effects. We believe it is not, for several reasons. First, getting a grip on causality requires researchers to have a detailed understanding of the kinds of mechanisms that could link one event with another. Most psychological theories, with their emphasis on stimulus-response relationships between causes and effects, understand causation in terms of agency and manipulation, leading psychologists to prefer evidence from experimental studies when making legitimate (or valid) causal claims. Most sociological theories, in contrast, understand causation in terms of naturally occurring regularities, and this notion imposes an effort to uncover mechanisms and capacities that enable causal relationships through the use of observational studies. Theories of media effects are not equally single-minded in this respect. They tend to combine the agency and manipulation conception of

causation with that of the "regularities" conception of causation. As a result, it is unlikely that a single research method can produce the empirical evidence needed for making legitimate causal claims about media effects.

Second, whereas the traditional model of causation is deterministic, empirical findings about media effects suggest overwhelmingly that the *same* initial conditions (or stimuli) produce *different* audience outcomes (i.e., some media outputs influence some people, under some circumstances). Put differently, media effects research has produced many examples where causal relationships appear to be probabilistic. Therefore, it may be unrealistic for media effects researchers to produce legitimate evidence of causation from a deterministic perspective. Rather, causation in media effects research may be better expressed and evaluated in probabilistic terms.

Last, it is important to recognize that media effects research encompasses a set of rather complex phenomena. As Bryant and Thompson (2002) note, media effects may be cognitive, behavioral, or affective; direct or indirect; short term, long term, or delayed; self-contained or cumulative. No doubt, this complexity makes the task of drawing causal inference about media effects ever more difficult and unlikely to be resolved simply by the choice of a research design, as the traditional approach to causation leads us to believe. Rather, we believe that the field is better served by a conception of threats to causal inference that is more directly derived from (but also more directly responsive to) the key theoretical and methodological challenges to media effects research: (1) the multilevel nature of media effects, (2) the dynamic nature of media effects, (3) the complexity of causal chains, (4) the measurement of key constructs, and (5) audience activity. We review each of these challenges in the next section and discuss the threats they pose to causal inference in media effects research. Where appropriate, we also point to some useful research procedures or quantitative applications that may mitigate such potential threats.

◆ *Challenges to Causal Inference in Media Effects Research*

THE MULTILEVEL NATURE OF MEDIA EFFECTS

An important factor contributing to the complexity of media effects as a subject of investigation is that such effects occur simultaneously at multiple levels of analysis. At the most basic level of abstraction, this means that media effects occur *independently* at different levels of analysis: individuals, groups, organizations, and social institutions (Bryant & Thompson, 2002). A more common interpretation of media effects as a set of multilevel phenomena (e.g., Slater, Snyder, & Hayes, 2006) is grounded in the recognition that characteristics or processes that occur at a higher level of analysis (e.g., groups) are bound to influence media effects at a lower level (e.g., individuals). Thus, some similarities and differences in the way individuals respond to or learn from information in the media can be explained by the characteristics of the groups or the organizations to which they belong. In essence, the source of complexity in this conception of media effects as multilevel phenomena is the *structural or relational context* in which media effects take place. But by far the most challenging notion of media effects as multilevel phenomena recognizes that the effect that media stimuli have at one level of analysis is often confounded with (or rarely independent of) the effect they have at another level of analysis (Pan & McLeod, 1991). For example, news coverage of drunk driving over time likely influences individuals' drunk driving–related attitudes and

behaviors, but it also simultaneously affects group norms and public policy on the issue, which themselves are an important source of influence on individuals' attitudes and behaviors (Yanovitzky, 2002; Yanovitzky & Bennett, 1999; Yanovitzky & Stryker, 2001). We return to this issue in our discussion that follows of the complexity of causal chains in media effects.

As others have already noted (Pan & McLeod, 1991; Slater et al., 2006), by virtue of limiting their focus of investigation to a particular (usually the individual) level of analysis, media effects researchers appear to have settled (conceptually and methodologically) on a single-level view of media effects, thus ignoring, for the most part, the cross-level linkages suggested by the multilevel view of effects. Consequently, such researchers likely expose themselves to at least three types of threats to causal inference (see Dansereau, Cho, & Yammarino, 2006; Price, Ritchie, & Eulau, 1991): fallacy of composition, ecological fallacy, and fallacy of the wrong level. *Fallacy of composition* (or atomistic fallacy) refers to cases in which an erroneous conclusion is drawn about a whole based on the features of its parts, such as when data collected from individuals is aggregated to represent the groups to which those individuals belong. The source of error here is that individual-level properties (e.g., personal attitudes, beliefs, or behavior) or relationships between such variables that are observed for individual members of the group are considered to be analytically equivalent to group-level properties (e.g., public opinion, norms) or to relationships between the same variables at the group level, when in fact they are not. For example, members' perceptions of group norms (an individual property) are conceptually different from group norms themselves, which are a property of the group (Yanovitzky & Rimal, 2006). Therefore, inferring group norms from aggregate measures of members' perceptions of norms is inappropriate. To mitigate concerns about the fallacy of composition, researchers should take care to select units that are comparable and representative of the whole (e.g., using information obtained from regular group members rather than from opinion leaders) and to offer empirical evidence that the same mechanism or process that causes one unit to change in response to a media stimulus (for example, information processing) can be generalized to all other units.

Ecological fallacy (or fallacy of division) is the inverse of the fallacy of composition. It pertains to cases when an erroneous conclusion is drawn from the characteristics of a whole to those of its parts. That is, group-level properties (or relationships observed among group-level variables) are mistakenly assumed to hold for individual members of the group. For example, in the context of the debate about the effects of media violence (see Bushman, Huesmann, & Whitaker, Chapter 24, this volume), some have proposed that links between levels of media violence and levels of societal violence support claims about the causal effect of media violence on individuals' aggression (Centerwall, 1989). However, this proposition has been challenged on the grounds of the ecological fallacy, as evidence indicates that violent criminals tend to be exposed to less (rather than more) media violence compared with their noncriminal counterparts (e.g., Freedman, 2002). Generally, then, one ought to avoid making causal claims about relationships between variables measured at a lower level of analysis from relationships observed among these variables (or their proxies) at a higher level of analysis. One exception to this is when the researcher is using data aggregated from lower-level units, such as when the percentage of women obtaining a mammogram each month is estimated from aggregated survey responses of women who answered a question about this behavior. In these cases

(which are quite characteristic of data used in media effects research), a researcher interested in making causal claims about individual units based on aggregated data may choose to test for the same effect in subsamples representing relatively homogeneous subgroups and to demonstrate that relationships among variables that exist at the higher or population level are also present at the levels of subgroups.

Finally, ignoring cross-level linkages may cause researchers to look for media effects at the wrong level of analysis. This potential error is termed *fallacy of the wrong level* and encompasses cases in which an effect is attributed to a variable, relationship, or process that exists at one level of analysis when it should in fact be attributed to a variable, relationship, or process that exists at another level. For example, while the majority of studies that have tested the effects of anti–drunk driving campaigns on drivers' drunk driving–related cognitions and behavior failed to find evidence of an effect at the individual level, there is credible evidence that these campaigns have been effective in generating anti–drunk driving legislation, which, in turn, influences drivers' behaviors (Yanovitzky, 2002). Thus, the conclusion that these campaigns failed to decrease drunk driving behavior is at best premature (if not simply erroneous) since effects at other levels of analysis were ignored. The only way to avoid an erroneous causal inference in this case is to test causal hypotheses at multiple levels of analysis, provided that there is a good logical or theoretical reason to expect relationships between variables to exist at multiple levels of analysis.

It is worth noting that all three types of fallacies involve a problem of inference, not of measurement or misspecification of statistical models. That is, these fallacies are largely outcomes of inadequate theorizing about the nature of cross-level relationships rather than a problem of using inadequate analytical tools. Whereas many of the media effects theories covered in this volume (e.g., agenda setting, cultivation, and knowledge gap) conceptualize effects at multiple levels of analysis, they fail to offer logical cross-level links (Pan & McLeod, 1991). To illustrate, how may cultivation effects on individuals help us explain cultivation effects that occur at the societal level (such as mainstreaming), and vice versa? If we assume that all members of society are similarly influenced to cultivate a certain perception of the world following exposure to media content, then the societal-level effect of cultivation is simply the aggregate effect of media on individuals, and causal inference is straightforward. However, if other processes of effects are involved in the transition from the individual to the societal level of cultivation (for example, diffusion of information among members of a social group through interpersonal communication), then drawing causal inference about cultivation effects is likely more problematic. Therefore, the first line of defense against these fallacies is more careful cross-level theorizing and clearly explicating plausible mechanisms and processes that logically link effects that are observed at different levels. Perhaps the best-known example of such theorizing is the two-step-flow hypothesis (Katz & Lazarsfeld, 1955): a macrolevel variable (media content) is linked to a microlevel variable (a person's political attitudes and behavior) through a process of interpersonal influence that employs a mechanism of interpersonal communication. Similarly, diffusion of innovation theory provides a logical link between information processing and behavior change at the individual level to processes of social change (see Rogers, 1962). The bottom line is that causal inference about multilevel effects can only be as rigorous as the theory and logical assumptions on which it is based.

The other key to drawing valid causal inference from multilevel relationships

between variables is the use of appropriate methodology. Such methodology necessarily requires sampling of units from multiple levels (e.g., multistage probability sampling) and taking appropriate measures at each level of analysis. Unfortunately, the samples typically used in media effects research are far from meeting this requirement. Furthermore, when aggregated data is used, it is usually collected without regard for the fact that individual-level variations (for example, in political attitudes) are confounded with the characteristics of the groups to which individuals belong (e.g., shared values and norms). Consequently, evidence of causal relationships obtained in these studies may be spurious unless estimates of effects are corrected for bias due to group-level effects. Here it is important to recognize that the standard data analytical tools used in media effects research (e.g., analysis of variance and regression analysis) are not designed to handle multilevel data. Such statistical tools can serve causal inference well, particularly when randomized experiments are used, only as long as higher-level variables or processes are deemed irrelevant to the relationship of interest. If the potential confounding of levels of analysis cannot be logically dismissed, researchers who are interested in making causal claims should consider using more advanced data analytic methods that can handle multilevel relationships, such as multilevel modeling (see Hayes, 2006, for an excellent discussion of the potential use of multilevel modeling in communication research).

THE DYNAMIC NATURE OF MEDIA EFFECTS

A second major source of complexity in studying media effects involves the conception of media effects as a *process*—causal relationships between variables that are dynamically situated in a temporal context, such that time is a central dimension of media effects (see Yanovitzky & VanLear, 2008). Rarely, however, do media effects researchers use quantitative methodology to model processes of media effects (Watt & VanLear, 1996). The methodological approach used in these studies typically seeks to correlate effects (the beginning of a process) with outcomes (the end of the process), treating everything in between as a "black box" or making unverifiable assumptions about the unobserved process that links effects and outcomes. To illustrate, consider information processing from the perspective of the elaboration likelihood model (Petty & Cacioppo, 1986). According to this model, two inherently different routes of information processing (one that involves elaboration of, or thinking about, the claims made in a message and another that does not) can end up causing the same observed outcome (attitude change). A researcher who is primarily concerned with causation would need to trace the process of attitude change over time (short as it may be) to determine or verify which of the two routes was used. Ignoring the process and focusing on the relationship between the stimulus and the outcome could lead to erroneous conclusions about the way the effect came to be. In other words, taking the process of effect into account in the analysis of quantitative data allow us to move from simply saying *what* works to being able to explicate *how* it works, which is a crucial element for making causal claims.

The inclusion of time in the analysis of media effects has four important implications to drawing causal inference. The most obvious of these relates to the *fallacy of confusing cause and effect.* While a theory can (and should) be used to logically justify the expectation that one variable causes another, the task of determining the causal direction between two variables usually depends on knowledge of a time sequence between the effect and the outcome. This is typically a problem in cross-sectional studies, though

the temporal order between variables can be retrospectively constructed in many cases. When longitudinal data is available to the researcher (such as when data on the same variables is collected at multiple time points), the causal direction between two variables can be determined via a Granger causality test (Granger, 1969). The test is predicated on the assumption that past behavior of a variable is the best predictor of this variable's current behavior. According to Granger, X is said to cause Y when the previous (or lagged) values of X significantly predict Y after controlling for the effect of lagged values of Y on Y itself (operationally, this means including the lagged values of Y as an additional predictor of Y in a regression model estimating the effect of the lagged values of X on Y). In the next step, the order of variables is reversed such that X is being predicted from lagged values of Y. If X cannot be predicted from the lagged values of Y, then X is determined to be a cause of Y.

The second implication of time in media effects research relates to the history and future of the phenomena studied. By ignoring the time dimension in the analysis of media effects, we ignore an important source of variation in the relationship between media stimuli and audiences' reactions to these stimuli. Focusing on the conceptualization and operationalization of the effect (namely, exposure), it seems safe to argue that many media effects studies ignore the effect of past cumulative exposure (and its likely interaction with current exposure) on audiences' outcomes. For example, by the time they reach adulthood, most individuals in most parts of the world have been exposed numerous times to fear appeals in persuasive messages. It is only logical to assume that repeated exposure to fear appeals will influence the way they react to yet another use of fear appeal in a message (effects like priming, fatigue, or even reactance come to mind). Yet, studies of fear appeal almost always assume implicitly that subjects were exposed to fear appeals for the first time. This is the *fallacy of ignoring the past.* The *fallacy of ignoring the future* focuses on the outcome itself, where too often the possibility of delayed media effects is ignored (Hornik, 2002; Hornik & Yanovitzky, 2003). Because experimental and cross-sectional designs are limited to the investigation of instantaneous media effects, researchers may commit a Type II error (i.e., claiming to find no media effects when, in fact, effects do exist but are delayed) or a Type I error (i.e., claiming media effects when, in fact, they do not last much beyond exposure to the stimulus). Thus, planning to study variables in relation to their past and future can greatly reduce the potential for errors of causal inference in media effects research.

The third problem associated with ignoring the time dimension in media effects research is the *fallacy of the secular trend.* By "secular trend," we mean natural, long-term change in the characteristics of a phenomenon over time. People, groups, organizations, and societies are in a constant state of change. Therefore, even an ineffective media stimulus (e.g., a social marketing campaign) may appear to cause change in audiences, leading the researcher to erroneously claim campaign effects (Snyder & Hamilton, 2002). It may also be that the effect of the campaign is confounded with that of the secular trend such that a positive secular trend may cause researchers to falsely overestimate the effect of the campaign, whereas a negative secular trend may lead them to underestimate the effect of the campaign. However, this is more a problem of estimation than a problem of causal inference. Interrupted time-series designs (see Cook & Campbell, 1979) are generally considered an effective tool for partitioning the true effect of programs, such as communication campaigns, from the effect of the secular trend.

Finally, it is possible for a process or an outcome variable to assume a cyclical pattern (change in direction) over the study period, thus changing the dynamics

of the relationship between media stimuli and their hypothesized outcomes. Consider, for example, the issue-attention cycle in media coverage of issues (Downs, 1972). Now suppose you want to link media attention to HIV/AIDS to the public's perceived salience of this issue (see Rogers, Dearing, & Chang, 1991). Your conclusion about the power of media coverage to set the public's agenda on this issue is contingent almost entirely upon the time frame chosen for analysis. If you observe the relationship during the time frame in which media attention to the issue is at its peak, you may conclude that the effect of this coverage is positive and strong. However, if you are observing this relationship during the time frame in which media attention to the issue is declining, you may conclude that there is little or no evidence of an agenda-setting effect. We term this potential threat to causal inference the *fallacy of the wrong timing.* Avoiding this fallacy may require researchers to conduct a sort of sensitivity analysis, where relationships between two or more phenomena over time are compared for time periods of different lengths.

In general, then, the most efficient way to avoid potential threats to causal inference due to the dynamic nature of media effects phenomena is to employ longitudinal designs (e.g., panel, cohort, and time-series designs) and specialized data analysis tools that can handle time-sequenced data. Different approaches to modeling longitudinal media effects have been employed by media effects researchers (see Yanovitzky & VanLear, 2008, for a review), but the use of these methods remains infrequent despite the continued vitality of theories that conceptualize media effects as a process.

THE COMPLEXITY OF CAUSAL CHAINS

The question of *how exactly* media stimuli influence audiences is as crucial to media effects theory and research as it is to causal inference about media effects. Specifically, while the initial conception of media effects was based on a simple stimulus-response model, empirical findings demonstrating variations in how members of the audience responded to the same media stimuli undermined this basic conception, replacing it with the growing recognition that "(a) communication involves multiple, substantively distinct processes; (b) these processes may be redundant, complementary or contradictory; and (c) processes may mediate or moderate other processes" (Babrow, 1993, p. 110).

This statement suggests a nontrivial potential for misspecification of causal relationships in media effects research. In particular, each of three general types of causal modeling errors is relevant. The first is the *fallacy of the wrong causal process.* That is, the phenomenon of interest is being caused by a different mechanism or process than the one hypothesized. There are several variations of this fallacy. An immediately familiar example is understanding a media effect as either an outcome of learning from media messages (Fishbein & Ajzen, 1975) or an outcome of activation of preexisting knowledge following media exposure (Roskos-Ewoldsen & Fazio, 1992). Both may produce the same observed effect, but they involve very different cognitive processes. Another variation of this is the failure to match constructs' level of analysis with processes' level of analysis. For example, Scheufele (2000) notes that agenda-setting, priming, and framing effects have been examined under the broad category of cognitive effects, using similar research designs, where, in fact, conceptual differences or differences in level of analysis exist among these three types of media effects—framing and priming effects are appropriately conceptualized and tested at the individual level, whereas the agenda-setting effect is appropriately conceptualized and tested at the societal level. One final variation of this fallacy is

the general tendency in media effects research to focus on cognitive (psychological) processes as plausible causes of outcomes while ignoring social (sociological) or biological processes that may cause the same outcomes. For example, both exposure to information in the mass media and a social diffusion process can cause change in health-related attitudes and behaviors (see Yanovitzky & Stryker, 2001, for an example of a study that models both processes), though they may interact in different ways to influence audiences (see Yanovitzky & Blitz, 2000, for an example of a study comparing five plausible models of interaction). An effective strategy to avoid the fallacy of the wrong causal process is to appropriately conceptualize multiple causal mechanisms or processes of media effects and to devise empirical tests that directly compare the fit of two or more alternative causal processes to the data.

A second type of possible causal modeling error is the *fallacy of nonreciprocal causation.* The common hypotheses-testing procedure in media effects research relies on statistical tools that can only estimate recursive (or unidirectional) models of effect. A unidirectional model assumes that no two variables in the model are reciprocally related, with each affecting the other. This is unfortunate, since many theories employed by media effects scholars imply complex processes of effects that contain reciprocal causation and feedback loops (i.e., nonrecursive models). For example, there has been considerable debate in the persuasion literature about the causal relationship involved in the attitude-behavior or the intention-behavior link, with most researchers recognizing that some sort of reciprocal relationship exists between the two such that people's newly formed attitudes influence their behavior but also that people's current behavior is used to infer their attitude (O'Keefe, 2002; see also Eveland, Shah, & Kwak, 2003, for additional empirical evidence of nonrecursive relationships among media-related variables). Although several data analytical methods for estimating nonrecursive causal models exist (Berry, 1984), Stephenson, Holbert, and Zimmerman (2006) note that many nonrecursive models of media effects can be quite easily and efficiently estimated through the use of structural equation modeling (SEM).

The final type of possible causal modeling errors, the *third variable fallacy,* is assumed to be the most common in media effects research (Holbert & Stephenson, 2003). The third variable problem refers to confounding relationships in which a third variable intervenes in (causes, mediates, or moderates) the observed causal relationship between two others. Since randomized experiments and statistical control in observational studies provide reasonable guards against potential spuriousness (i.e., a situation in which the existence of a misleading correlation between two variables is produced through the operation of a third causal variable), mediation poses the greatest potential for misspecified causal relationships in media effects research.

The notion of mediation is explicitly integrated into theories and models that are frequently used in the field, such as the theory of reasoned action (Fishbein & Ajzen, 1975) and the extended parallel processing model (Witte, 1994). However, though tests of mediation are somewhat common in media effects research, tests of more sophisticated, theory-driven hypotheses about processes of media effects are far less common (Preacher & Hayes, 2008; Stephenson et al., 2006). When data are collected from an observational study, the recommendation is to use SEM to test for mediation (Stephenson et al., 2006), though many other alternative approaches exist, including techniques specifically for longitudinal and multilevel data (for a comprehensive review, see MacKinnon, Fairchild, & Fritz, 2007). However, mediation is not only an issue in observational studies. As O'Keefe

(2003) argued convincingly, it is equally crucial to consider mediation relationships in experimental settings to gain more clarity about causal chains.

In summary, the misspecification of causal chains linking media stimuli to their observed effects on audiences presents significant threats to causal inference in media effects research. The most effective way to mitigate these threats is through careful conceptualization and rigorous testing of causal chains, which of course serves as a golden opportunity for researchers to provide more accurate theoretical accounts of the causal mechanisms and processes underlying media effects.

MEASUREMENT OF KEY CONSTRUCTS

Problems with construct measurement are linked to causal inference through the notion of *construct validity,* or the degree of fit between the conceptual definition of the construct and the means by which it is observed. Specifically, Chaffee (1991) identified the explication of communication constructs as one of the main challenges to communication research. Concept explication, he explained, "is about a way of thinking; it is concerned with the disciplined use of words, with observation of human behavior, and especially with the connection between the two" (p. 1). The explication of theoretical constructs has proven to be a difficult task for media effects researchers, not only due to the complexity of these constructs but also because contextual factors such as the rapid evolution of communication technologies and the illusive nature of the audience effectively transform the meaning of constructs from time to time.

Consider the case of media exposure. Despite the centrality of this variable to media effects research as the primary independent variable, "systematic research about measuring exposure is remarkably thin" (Fishbein & Hornik, 2008, p. 1), and little has been published about the stability, reliability, or validity of most exposure measures. The two most common approaches for assessing variations in media exposure are through the use of experimental manipulations and individual self-reports (Rubin, Palmgreen, & Sypher, 1994). Another common approach involves content analysis of archival data (typically news stories appearing in the *New York Times* and the *Washington Post*) to infer exposure potential for members of the audience (Fishbein & Hornik, 2008). Less common are efforts to assess media exposure based on spending or media buys (Southwell, Barmada, Hornik, & Maklan, 2002). Each approach offers a radically different way of conceptualizing and measuring exposure, and each has some notable strengths and weaknesses. For example, experimental manipulations afford researchers control over the timing and dose of exposure to a particular media stimulus, but they raise legitimate concerns about the validity of this measure in real, uncontrolled environments. Self-reported measures of exposure are externally valid but are prone to recall errors (Potter, 2008), and estimating exposure from archival data is plagued with potential inference errors involving flaws in the sampling of sources and units for inclusion in the study, the coding of content units, and inferring the likelihood of exposure (see Stryker, 2008).

One crucial aspect of measuring exposure (and, we would argue, other variables) in media effects research is the lack of conceptual clarity (or clear explication) about what exactly constitutes exposure (see Potter, Chapter 2, this volume). Some focus on the media exposure act itself, equating exposure with time spent with the medium or content (Rubin et al., 1994). Others (Krugman, Cameron, & McKearney White, 1995; Slater, Hayes, & Ford, 2007) conceptualize media exposure in terms of attention to media stimuli, either self-reported or as measured by tracking eye movements. Still others (e.g., Southwell, 2005) conceptualize

exposure in terms of its expected outcomes, such as recall, reception, or encoded exposure (i.e., a minimal memory trace of the message content). There are also proxy measures of exposure, such as measures obtained based on content analysis (e.g., Gonzenbach, 1996), and measures of exposure that are conceptualized in terms of experimental manipulation. The fact that no agreed-on method for measuring exposure has emerged in the field most certainly influences the quality of conclusions drawn from research integrations, such as meta-analysis (Preiss et al., 2007). Different measures have different theoretical implications and yield different results. So what exactly can we claim, with confidence, about the effect of exposure to, for example, media violence on audiences' behavior if our key independent variable is measured in so many different ways?

There are two lines of defense against the threat of construct validity. The primary one involves careful explication of constructs and their meaning. For example, a careful explication of exposure ought to define this act while distinguishing it from its antecedents (the experimental manipulation), its outcomes (e.g., arousal, storage of information in memory), and its proxies (e.g., recall of information), as well as from related terms (e.g., scanning of information). It is also reasonable to expect that, depending on the particular context in which measures are taken, one way of measuring a construct will be superior to other ways of measuring the same construct. The way subjects experience exposure in laboratory experiments may be different than the way they experience it in natural settings; children may experience exposure differently than adults; the hearing-impaired may experience it differently than the vision-impaired. Only by capturing similarities and differences in the ways exposure is experienced by different people and under different circumstances can we begin to achieve clarity about the meaning of this construct and the most valid way to observe it.

The other way to improve construct validity is to seek rigorous, unbiased measures of key variables. One goal to pursue in this respect is the development of measures that are independent of person factors, such as differential capacities or motivations to engage with the measurement instrument. Item response theory (see Embretson & Reise, 2000) provides the framework and statistical tools needed to achieve this goal, and the time is ripe for media effects researchers to take advantage of procedures developed in other fields for improving measurement in this way. Combined with cross-validation of measures through triangulation (DeVellis, 2003), this approach can greatly simplify the construction of valid measures of variables in media effects research.

THE ACTIVE AUDIENCE

As the initial conception of audiences as passive receivers of information from the mass media has all but dissipated in favor of the active audience conception (see Potter, Chapter 2, this volume), an additional set of challenges to causal inference was introduced into media effects research. Specifically, audience activity introduces a significant potential for *selection bias* into the study of media effects. Selection bias is associated with the researcher's lack of control over the assignment of units to different levels of the independent variable because the units self-select into these levels based on factors such as personal relevance and opportunity to participate. In the context of media effects research, selection bias is actually a function of three documented forms of audience activity. First is selective exposure, or people's tendency to expose themselves predominately and preferentially to information that is consistent with their own beliefs, attitudes, and prior experience (Zillmann & Bryant, 1985). The second form of audience activity is selective processing (or interpretation) of information

received from the media (Hall, 1980; Kunda, 1990). The third form of audience activity involves message production by the audience as an alternative to consuming information produced by mass media organizations (Riegner, 2007).

The first form of audience activity (selective exposure) fits neatly into the traditional definition of selection bias in that audiences (and not the researcher) control their exposure to the media stimuli. The second (selective processing) involves lack of researchers' control over the mechanism of effect (information processing). The third form of audience activity undermines researchers' control over the direction of effect. Importantly, there is a reason to believe that these three forms of audience activity are not independent of one another, and their likely interaction makes the task of controlling for selection bias in media effects research ever more complicated. The potential threat of audience activity to causal inference relates to both internal and external validity. Correcting for potential selection bias under these circumstances inevitably means controlling for an ever-growing number of audience-centered variables in statistical models that estimate media effects (Nabi, 2007). This also means collecting data from larger and more heterogeneous samples to ensure sufficient power and representativeness. Methods for bias correction, such as propensity score models (see Yanovitzky et al., 2008), make this task more manageable and facilitate causal inference about media effects through a matching procedure that closely approximates random assignment to experimental conditions.

♦ *Conclusion*

We agree with Nabi (2007) that "it is this diversity—in outcomes assessed, stimuli considered, and methods employed—that makes conclusions about the psychological and social effects of the media so difficult to pin down" (p. 137). At the same time, we also recognize that the field's engagement with the issue of causal inference has been less than optimal and believe that more attention to causation consideration has the potential for promoting greater integration and synthesis of empirical findings in media effects research. Throughout this chapter, we made three central claims about causal inference in media effects research. First, we claim that the traditional social scientific approach to causation is largely inadequate as a framework for studying causal relationships in media effects research and should be replaced with a framework that is more directly derived from and responsive to the challenges inherent in studying media effects phenomena (the multilevel nature of media effects, the dynamic nature of media effects, the complexity of causal chains, the measurement of key constructs, and audience activity). In all, we identified 13 unique threats to causal inference in media effects research, but there are likely others that we missed and that we count on others to point out.

Our second central claim is that getting a grip on causality in media effects research requires researchers to have a detailed understanding of the kinds of mechanisms and processes that could link one event with another. Researchers must think about what these processes might be and how they operate before they test causal propositions. Existing theories of media effects (particularly those that were refined over time) can provide important guidance in terms of the conceptualization of key constructs and relationships, but only if researchers engage them fully and more frequently than they have done in the past (Bryant & Cummins, 2007). Better explication of causal relationships is also directly linked to more compelling and less abstract theorizing of media effects (Hornik & Yanovitzky, 2003), which is necessary for translating scientific knowledge about media effects into practical, real-world applications.

Our third claim is that researchers interested in causal inference ought to

make a concerted effort to match their method of investigation with the relative complexity of the phenomenon they study. Standard research designs and methods are reasonably equipped to handle basic research on causal relationships and causal mechanisms. More complex causal phenomena require more sophisticated designs and data analysis tools. Both kinds of studies have a role in advancing the science of media effects, providing that causation considerations are no longer secondary to statistical inference. This leaves much room for the legitimate use of methods, ranging from basic laboratory experiments to complex evaluations of public communication campaigns, to test causal relationships in media effects research. A greater attention to causation considerations implies that media effects researchers spend more of their time designing causally valid research and less time acquiring advanced statistical skills that they can easily tap by calling on the statistician next door.

As media effects research continues to produce evidence about the significance of media effects phenomena to the everyday lives of people, communities, and institutions, media effects scholars ought to take more responsibility for the implications of the theories they use and of the findings they generate. The path to greater accountability must go through serious discussion among scholars about issues of causation in media effects research. The task is not easy, but the potential payoff in terms of integration and synthesis of media effects research makes this a worthwhile investment in the field's future.

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