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Sexual Abuse, Family Environment, and Psychological Symptoms: On the Validity of Statistical Control

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M. R. Nash, T. L. Hulsey, M. C. Sexton, T. L. Harralson, and W. Lambert (1993) reported on the effects of controlling for family environment when studying sexual abuse sequelae. Sexual abuse history was associated with elevated Minnesota Multiphasic Personality Inventory and Rorschach scores in a sample of 105 women, but many of the reported differences disappeared when a Family Functioning Scale score was used as a covariate. The present article considers the findings of Nash et al. in terms of the theoretical and statistical constraints placed on analysis of covariance and other partializing procedures. Because family dysfunction is not always causally antecedent to sexual abuse, and given the quasi-experimental quality of most abuse research, the use of covariate techniques to test hypotheses about the causal role of family environment in the impacts of sexual abuse may be ill advised. Analyses of a 2,964-subject data set illustrate these concerns.

With only a few exceptions, more than a decade of published studies on childhood sexual abuse report an association between self-reported childhood molestation experiences and subsequent mental health problems in adolescence and adulthood (Briere & Runtz, 1991; Browne & Finkelhor, 1986). Most of this research has been correlational, however, and thus interpretation of these data remains equivocal. The most critical issue is well known to behavioral scientists: Does the statistical relationship between abuse and later distress reflect a causal phenomenon (i.e., does childhood sexual abuse have negative psychological impact), or is the relationship caused by other variables such as concomitant family dysfunction or the impact of other events during or after the abuse? Although the experiences of clinicians in the child abuse field tend to support the former scenario, the need for more empirically based tests of these competing hypotheses is clearly indicated.

Because most of the research has been quasi-experimental (i.e., involving cross-sectional comparisons between intact, potentially nonequivalent groups), investigators have been forced to use statistical methods to control for the possible mediating effects of third variables on the relationship between molestation and psychological symptoms. The results of these efforts have been mixed: Some researchers have found that the effects of sexual abuse disappear when variance associated with family environment or other concomitant forms of maltreatment have been removed (e.g., Fromuth, 1986), whereas others have indicated that this relationship remains relatively robust to third-variable control, although the effects of sexual abuse on psychological functioning may be smaller in size or number of impact types (e.g., Briere & Runtz, 1990; Elliott & Edwards, 1991; Peters, 1988).

In this issue of the *Journal of Consulting and Clinical Psychology*, Nash, Hulsey, Sexton, Harralson, and Lambert (1993) demonstrate the apparent variability in sexual abuse effects that can occur when controlling for negative family environment. Their study presents data on the psychological symptomatology of 105 women from clinical and nonclinical groups who either reported sexual abuse in childhood (defined as sexual contact with someone at least 5 years older that resulted in victim or perpetrator orgasm) or did not do so. Nash et al. found that, although sexual abuse history was associated with elevations on most Minnesota Multiphasic Personality Inventory (MMPI) scales and Rorschach indicators (but not with greater hypnotizability), many of these group differences disappeared when a composite Family Functioning Scale score was used as a covariate. Exceptions were the Hypochondriasis (*Hs*) and Paranoia (*Pa*) scales of the MMPI and the Personal Perception indicator of the Rorschach, each of which remained significant when controlling for family functioning. Nash et al. concluded from these data that "greater nonspecific impairment among abused women may be a consequence, at least in part, of pathogenic family structure rather than sexual abuse per se" (p. 276).

Although Nash et al. (1993) and others (e.g., Fromuth, 1986) have used partializing methods such as analysis of covariance or partial correlation analysis to infer a causal role of family environment, there are several constraints on this approach that limit the inferences that can be made from the results (Briere, 1989, 1992a). Specifically, partializing analyses may be misleading if (a) the design is quasi-experimental (Lord, 1969; Tabachnick & Fidell, 1989), (b) the control variable is unreliable (Cohen & Cohen, 1983; Stevens, 1986), (c) there is significant multicollinearity between control and independent variables (Pedhazur, 1982), or (d) the control variable is not causally antecedent to the independent variables (Davis, 1985; Pedhazur, 1982). Cohen and Cohen (1983) warned in the latter case that "to the extent that there is B [independent variable] → A [covariate] causality, we underestimate the size of the [indepen-

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dent variable] effect" (p. 383) by analysis of covariance (ANCOVA). Similarly, Tabachnick and Fidell (1989) noted that "mean differences on a covariate associated with an IV [independent variable] are quite legitimately corrected for *as long as the covariate differences are not caused by the IV*" [italics added] (p. 322).

Of these various concerns, the quasi-experimental and causal antecedence issues are most relevant to sexual abuse research. If (a) sexual abuse can have negative effects on family functioning, (b) family functioning and sexual abuse can have mutually reciprocating impacts, or (c) the subject's later perception of family functioning can be affected by his or her sexual abuse history, then the assumption of causal antecedence is not met and the partialized test of abuse effects will be misleading. Furthermore, the quasi-experimental nature of the comparison makes the use of covariate analysis in testing hypotheses about the causal role of the covariate especially untenable. Tabachnick and Fidell (1989), for example, noted the following:

> Sources of bias in ANCOVA are many and subtle, and can produce either under- or overadjustment of the DV [dependent variable]. At best, the nonexperimental use of ANCOVA allows you to look at the IV DV relationships (noncausal) adjusted for the effects of the covariates, as measured. . . . Don't expect ANCOVA to permit causal inference of treatment effects with nonequivalent groups. (p. 322)

As a further complication, it is likely that causal antecedence may vary as a function of whether the abuse is intrafamilial or extrafamilial. In the former case, although sexual abuse might arise from negative family dynamics, it might also easily produce or exacerbate negative family roles and relationships. The clinical literature often cites the "family secret" implicit in many instances of incest, wherein certain family members (e.g., the nonoffending spouse or uninvolved siblings) are kept unaware of intrafamilial sexual abuse, and the victim either withdraws from family interaction or becomes sufficiently symptomatic that his or her behavior stresses or disrupts the family system (e.g., Briere, 1989; Courtois, 1988; Finkelhor & Baron, 1986). Writers on the family dynamics of incest also have described the realignment of parent–child and parent–parent relationships that can occur when normal family roles are distorted by sexual exploitation. As Courtois (1988) noted, such dynamics may be exacerbated after the abuse is disclosed:

> Divided loyalty and protection of the family unit are major factors when the abuse has occurred within the nuclear family and may be present to a lesser degree in the extended family. . . . Perpetrators usually react to disclosure with denial, defensiveness, and hostility toward the child and anyone either within or outside of the family who support the child. The family tie allows the perpetrator access, influence, and authority with which to challenge the child. Many mothers of victims respond in a concerned and protective fashion, although it is not uncommon for some to deny the abuse or to attack or blame the child. . . ." (p. 31)

In the case of extrafamilial sexual abuse, on the other hand, the perpetrator is not a family member, and thus family roles and relationships are typically less skewed by the presence of abuse (Courtois, 1988; Meiselman, 1990). When family dysfunction is associated with extrafamilial sexual abuse, negative family dynamics will more likely serve as a risk factor for outside sexual victimization (Finkelhor & Baron, 1986). Because this phenomenon does not violate the principle of causal antecedence, covariate analyses may be more appropriate.

Data on family environment and the impact of sexual abuse as a function of familial locus were recently reported in a study of 2,964 professional women (Elliott & Briere, 1992). We found that familial dysfunction, as measured by the total Family Environment Scale (FES; Moos & Moos, 1986) score varied according to whether sexual abuse was in the immediate family, the extended family, outside of the family, both intra- and extrafamilial, or not present at all, $F(4, 2809) = 34.03$, $p < .0001$; scores on the Trauma Symptom Checklist—40 (TSC-40; Briere & Runtz, 1989) also varied in a similar manner, $F(4, 2826) = 32.31$, $p < .0001$. As presented in Table 1, nonabused subjects reported less family dysfunction and lower TSC-40 scores than did any of the groups of abused subjects. Abuse within the immediate family was associated with more family dysfunction than either abuse in the extended family or extrafamilial abuse, a finding that supports Courtois's (1988) contention that dynamics can differ according to immediate versus extended family locus. Furthermore, when FES was controlled for by ANCOVA, the TSC-40 differences found among the four groups of abused subjects disappeared, although the basic abuse-versus-no-abuse effect remained, $F(4, 2808) = 18.34$, $p < .001$. Such data suggest that family functioning moderates the relationship between sexual abuse and symptomatology but may not explain it.

Elliott and Briere's (1992) results are at variance with those of Nash et al. (1993), wherein controlling for family dysfunction eliminated the abuse effect. It is likely that the former study's large sample size (with each of the four abused groups containing 125 or more subjects) provided sufficient statistical power to override any underestimation of abuse effects associated with a compromised causal antecedence condition. To test this hypothesis for the present study, we randomly selected 50 abused (25 who reported seeing a psychotherapist at the time of data collection and 25 who did not) and 50 nonabused (25 who reported seeing therapists and 25 who did not) subjects from our full sample of 2,964 (thereby approximating the design of Nash et al.) and reconducted the analyses mentioned earlier. Under these conditions, as occurred for Nash et al., there was a sexual abuse effect, $F(3, 96) = 3.41$, $p < .020$, until FES was included as a covariate, after which the abuse effect dropped out, $F(3, 94) = 1.86$, *ns*.

Directions for Future Research

Given the interpretive constraints placed on covariate analysis of quasi-experimental data, study of the mediating effects of family environment must address a variety of statistical and theoretical concerns. The following are some suggestions in this regard.

Ensure Adequate Sample Size

Limited sample size is a common problem in the abuse effects literature. An insufficient number of subjects can result in low statistical power to reject the null hypothesis when it is false. Ironically, this problem is matched by the tendency of some researchers to include many variables in their designs—a

Table 1
Mean FES and TSC–40 Scores According to Abuse Status

Scale	No abuse (n = 2,068)	Extrafamilial (n = 354)	Extended family (n = 158)	Immediate family (n = 125)	Extra- & intrafamilial (n = 126)
Family Environment Scale	53.39_a	17.91_b	19.01_b	13.19_c	13.15_c
TSC–40 total score	20.86_a	25.33_b	$25.32_{b,c}$	$28.14_{c,d}$	$27.48_{c,d}$
Adjusted TSC–40 scores, controlling for FES	22.03_a	25.51_b	25.78_b	27.15_b	26.56_b

Note. Means not sharing a common subscript are significantly different at $p < .01$. FES = Family Environment Scale; TSC–40 = Trauma Symptom Checklist—40.

practice that, when combined with a low sample size, can result in capitalization on error variance and spurious findings. These concerns are all the more relevant to research in which variance associated with one variable is removed before other variables are analyzed: Instability of the former might result in under- or over statistical "control" in tests of the latter. The problem of low statistical power is demonstrated in the Elliott and Briere data set, where the residualized abuse effect was highly significant at N = 2,964 but not significant at N = 100.

Specify and Evaluate Abuse in Terms of Family Locus

To the extent that the Briere and Elliott findings are generalizable, researchers should consider analyzing immediate family, extended family, and extrafamilial abuse separately when attempting to control for family environment; otherwise, the resultant residualized relationship between abuse and symptomatology may be difficult to interpret. For example, is removing family dysfunction variance from symptoms associated with nuclear family sexual abuse the same as removing it from symptoms related to extrafamilial abuse? Can we make any definitive statements about the implications of such control procedures if the proportion of intra- to extrafamilial abuse victims in a given study is unknown? Most basically, can a researcher who finds a certain relationship among family functioning, intrafamilial sexual abuse, and symptomatology generalize his or her findings to other groups containing extrafamilial abuse victims?

Avoid Causal Interpretations of Partialized, Quasi-Experimental Research Data

The purpose of covariate analysis is to control for important subject differences on phenomena that are not considered to be independent variables. Although even this procedure has been criticized when applied to quasi-experimental data (Lord, 1969), we agree with Tabachnick and Fidell (1989) and others that this constraint may be unnecessarily stringent. It is appropriate, however, to draw the line at the use of such procedures to test causal hypotheses.

Attend to Measurement Issues

When the specific relationship of multiple sets of constructs is studied, precise measurement of the variables at hand becomes especially important. In the present context, this includes specification of the independent variable (sexual abuse), dependent variable (psychological symptomatology), and covariate (family functioning).

In the first instance, different definitions of what minimally constitutes sexual abuse may produce different associations between family functioning and abuse–symptom relationships. This is especially relevant to the Nash et al. (1993) study, because their definition required the presence of perpetrator or victim orgasm during the abuse—a criterion heretofore not found in the sexual abuse research literature. Thus, for example, a subject who was sodomized or digitally penetrated by a parent, but where orgasm did not take place, would be classified as nonabused. As a result, we anticipate that the Nash et al. definition placed into the nonabused comparison group a number of subjects who would be considered abuse victims by other researchers (e.g., Finkelhor, 1979; Russell, 1986). Removing all symptom variance associated with family dysfunction in this abuse group might produce a different effect than if the minimal requirement for abuse had been any physical contact of a sexual nature (along with the 5-year difference criterion) as per Finkelhor (1979) and others.

Measurement of psychological symptomatology is an ongoing issue in abuse effects research (Briere, 1992a; Elliott & Briere, 1991). Measures that are too general may be insensitive to abuse-related distress, and thereby allow underestimation of actual abuse-specific trauma. Thus, for example, although the use of the MMPI and the Rorschach indicators by Nash et al. (1993) is supportable on the basis of established psychometric validity, these measures may not be as relevant to abuse-related symptomatology as other instruments (e.g., the TSC–40 or the PTSD scale of MMPI-2 [Butcher, Dahlstrom, Graham, Tellegen, & Kaemmer, 1989]). In addition, as suggested by Nash et al., controlling for family dysfunction by ANCOVA may eliminate certain abuse main effects (e.g., on depression or anxiety) but not others (e.g., somatization, self issues, posttraumatic symptoms, or sexual dysfunction). As a result, researchers may wish to include a variety of different measures of psychological dysfunction in their designs (assuming a sufficient sample size), and qualify their findings in terms of which symptom types respond to statistical control of family environment differences and which do not.

Researchers who want to examine the impact of family environment on abuse–symptom relationships are constrained by the psychometric quality and criterion coverage (Anastasi,

1988) of the family functioning measure used. An unreliable covariate not only violates an assumption of ANCOVA but increases the likelihood that the association between family functioning and later symptomatology will be underestimated. Family measures without sufficient criterion coverage may not tap the full range of negative family environments, thereby potentially allowing the underassessment of the role of family functioning associated with abuse and symptomatology. Relevant to both Nash et al. (1993) and Elliott and Briere (1992), it is likely that accurate family measures consist of multiple underlying dimensions (e.g., the 10 subscales of Moos and Moos's [1986] FES), rather than being well represented by a single summary score. For example, family dysfunction involving excessive parental control may differ in extent or directionality from a given family's level of conflict or achievement orientation. To the extent that this is true, a single family functioning covariate may be insufficient.

Consider Other Methods of Evaluating Family Environment Impacts

Given the concerns stated earlier, the most prudent course for those interested in the relative roles of family environment and childhood sexual abuse on symptomatology may be to abandon ANCOVA altogether. Statistical procedures that do not "pre-remove" variance associated with family dysfunction may be helpful in this regard. Possible examples of alternative statistical techniques include factorial designs or multiple regression analyses in which, along with sexual abuse, family dysfunction is a simultaneous independent variable and in which there is an Abuse × Family Environment interaction term. Such approaches not only avoid overestimation of the family environment effect but allow evaluation of the possible interaction of family environment and sexual abuse on psychological symptoms—a phenomenon that violates an important assumption of ANCOVA in this context (Briere, 1988).

Finally, researchers may discover that the most comprehensive method of analyzing family environment and abuse history with regard to later symptomatology is by way of multivariate tests. Canonical correlation analysis or causal modeling, for example, would allow the evaluation of family environment variables, sexual abuse in its various forms, and other types of childhood maltreatment as they relate simultaneously to a variety of different symptomatic outcomes. Multivariate methodologies may reveal that different forms of family dysfunction are associated with certain constellations of symptoms, intrafamilial sexual abuse with others, and so on. At this level of analysis, the concept of controlling for family environment becomes less germane (Briere, 1992b).

Conclusions

The present report outlines some of the issues involved in using covariate analysis to test causal hypotheses in quasi-experimental designs. The concerns presented do not necessarily mean that family dysfunction does not mediate between sexual abuse and abuse-associated symptomatology, only that causal hypotheses about the role of family environment cannot be assessed accurately by intact groups procedures using standard covariate analyses. Nash et al. (1993) were careful to emphasize that certain symptoms *may* be a consequence of family dysfunction. Ultimately, as our approach to the complex antecedent and effects of sexual abuse becomes more sophisticated, we may discover that this variable is but one of many pathogenic events present in the early lives of many symptomatic adults, as opposed to an explanatory (or distracting) entity that should be partialed out before the impacts of child abuse are examined.

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The following ethical standards are extracted from the "Ethical Principles of Psychologists and Code of Conduct," which appeared in the December 1992 issue of the *American Psychologist* (Vol. 47, No. 12, pp. 1597–1611). Standards 6.21–6.26 deal with the reporting and publishing of scientific information.

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