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Reply to Comment by Briere and Elliott

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M. R. Nash, T. L. Hulsey, M. C. Sexton, T. L. Harralson, and W. Lambert respond to J. Briere and D. M. Elliott's (1993, this issue) comments regarding their study (1993, this issue). The investigation was in no way designed or presented as a causal analysis; covariance analysis is an acceptable means of identifying and controlling for important mediating variables. The authors cite some methodological and conceptual limitations of the survey study database presented by Briere and Elliott. Finally, points of agreement with Briere and Elliott are noted, especially in the joint call for longitudinal, multimethod designs for examining pathogenic factors associated with a history of sexual abuse.

As we have little or no objection to Briere and Elliott's (1993) treatment of the general methodological issues facing the discipline, we focus our comments on their reaction to our study—a reaction that we believe represents a dramatic misreading of our intent, findings, and conclusions.

Causation

First and most important, Briere and Elliott (1993) characterized the study as a causal analysis, testing causal hypotheses. We strongly object to this portrayal of our intent and conclusions. Indeed, nowhere in the study are the words *cause, causal antecedent,* or *causality* used. Mindful that no amount of technical manipulation of cross-sectional correlational data (covariance analysis, canonical correlation, or even path analysis) can yield definitive causal information, we crafted our three hypotheses and our conclusions very carefully to avoid the language of etiology. The three hypotheses tested in our study are (a) "Sexual abuse per se is associated with broad-spectrum, general psychological impairment independent of the effects of perceived family environment" (p. 277), (b) sexually abused women are more dissociative and hypnotizable than nonabused women with group differences not being ". . . explained by variance on the family environment measure" (p. 277), and (c) "there is a specific pattern or cluster of symptoms that distinguishes abused from nonabused subjects, independent of the effects of family environment" (p. 277). We submit that these propositions do not constitute causal hypotheses and that by instead addressing issues of mediation, they lend themselves quite appropriately to the power and limitations of covariance analysis as endorsed by Briere and Elliott (pp. 284–288).

For the same reasons, we agonized over the wording of our conclusions. On the four occasions when we speculated about the effect of sexual abuse, we felt that we were appropriately circumspect: "[Observations of] greater nonspecific impairment among abused women *may* be a consequence, *at least in part,* of pathogenic family structure rather than sexual abuse per se [italics added]" (p. 276); "*For some victims,* sexual abuse *may* be a signal variable that the home environment is profoundly and broadly pathogenic [italics added]" (p. 282); ". . . whether this prevalence of dissociative experiences is a product of sexual abuse, a pathogenic home environment, or an interaction between the two is *unclear* [italics added]" (p. 282); "Whatever association may exist . . . is complex, embedded within the interpersonal context, and not linear in nature" (p. 282). Neither our clinical data nor Briere and Elliott's survey data allow any definite statement about temporal causality.

At this juncture, we expect that researchers will have honest, healthy disagreements about the relative weights and causal importance of specific pathogenic factors associated with a history of sexual abuse. Our hope is that the grand conclusions about univariate cause that abound in the literature (e.g., sexual abuse causes dissociation; sexual abuse causes borderline pathology) will be replaced by empirically testable multivariate models that more fully address the complexity and richness of human experience.

The Reviewer's Study

It is indeed interesting that in the Briere and Elliott (1993) study, subjects who reported extended-family abuse perceived their family to be more disrupted than did subjects who reported extrafamily abuse. However, this still does not support any particular causal model. Perhaps the abuse itself was the proximal or antecedent cause of the family disruption. Maybe a pretraumatic chaotic family environment rendered some subjects more vulnerable to abuse by a family member. Perhaps perception of family environment was distorted by the memory of having been abused. Here again, all we can really say with confidence is that perceived family environment appears to play an important role in the association between a history of early sexual abuse and subsequent psychological impairment.

Briere and Elliott asserted that their findings (Briere & Elliott, 1993) contradict those of our study. We note important points of convergence that are all the more remarkable when we consider that, in sharp contrast to our study, Briere and Elliott surveyed only relatively affluent professional women (with a

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median income greater than $45,000) and used an entirely self-administered format. Across both studies, subjects' perception of family was an important mediator of group differences on measures of dissociation, and in both studies there was a significant effect for abuse status even when perceived family environment was used as a covariate. We believe that these similarities across populations and methodologies underscore the importance of examining pathogenic contextual features of the environment.

One limitation of Briere and Elliott's (1993) study involves the issue of method variance (Cook & Campbell, 1979): With all the data being based on self-report measures, there is the potential that using the same method of measurement contributed greatly to the observed relationships among the measures. This seems especially important in large monomethod survey studies such as that of Briere and Elliott, in which relatively small effect sizes, potentially attributable to method variance, can nevertheless reach statistical significance with a large enough sample size ($N = 2,964$).

Methods and Measurement

Briere and Elliott (1993) made several important technical points on methodology. First, they suggested that our sample size was not sufficient to reveal group differences. This position ignores the fact that we did indeed find main effects for abuse with, and without, family environment as a covariate. One could speculate about why the sample size was sufficient for some effects and not others, but one cannot cast this criticism broadly across the entire study.

Second, our manuscript as submitted for Briere and Elliott's review contained an error on the orgasm criterion for assignment to the abused group. This error was maintained in the published article with a footnote (p. 277). In fact, assignment to the abused group did not require orgasm, it required genital contact. Indeed, because we used Finkelhor's (1979) questionnaire, we had no way of knowing whether orgasm occurred. Briere and Elliott's (1993) comment on this issue is quite valid and remains so even with this clarification. After all, we did require genital contact, a criterion not specified by Finkelhor's definition. By doing so, we might have diluted the nonabused group with subjects who, before the age of 17, were involved in nongenital sexual fondling with someone at least 5 years older. However, review of our data indicates that this was not the case. We did not encounter this particular scenario in our sample of 49 subjects designated as nonabused. It is important to keep in mind that our publicity and referral materials were very explicit about recruiting "women who have, and *have not,* experienced sexual abuse [italics added]" (Nash et al., 1993, p. 277). It seems especially likely that referred cases in both categories would be "nonmarginal" one way or the other.

Finally, Briere and Elliott (1993) suggested that using measures that sample broadly across psychopathology may lead to an underestimation of actual abuse effects. We disagree on empirical and conceptual grounds. First, for many years now researchers have claimed that sexual abuse is associated with almost every conceivable form of pathology (Browne & Finkelhor, 1986). Indeed, by using standard, comprehensive, widely used measures of pathology, we found significant effects for abuse across the Minnesota Multiphasic Personality Inventory, Form R, and the Rorschach Inkblot Test. Second, we believe that it is invalid to assume, a priori, that abuse has only limited effects and that it should only be studied by abuse-oriented instruments. After all, if sexual abuse research is to be brought more fully into the mainstream of psychological research, it must more often use the tools of the trade.

In summary, we heartily concur with Briere and Elliott's (1993) call for more sophisticated, longitudinal, multimethod treatments of pathogenic factors associated with a history of childhood sexual abuse. Only in this way can clinicians and researchers begin to unravel the intricate problem of how and why sexual abuse and psychopathology are linked.

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