Why Is Enacted Social Support Associated With Increased Distress? Using Simulation to Test Two Possible Sources of Spuriousness

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Although perceiving that social support is available is often associated with psychological benefits in times of stress, there is evidence that actually receiving support is associated with increased distress. To assess whether this latter association could be spurious, the authors conducted computer simulation studies to examine two theoretical models that could give rise to it. Study 1 examined a process whereby distress leads to provision of support rather than the reverse. Study 2 examined a process whereby an adverse event increases both support and distress. In both cases, substantial spurious associations were produced only when parameter values were markedly unrealistic. It was concluded that these possible sources of spuriousness are insufficient to explain the association between support and distress found in naturalistic studies.

Keywords: *simulation; theory testing; social support; statistical analysis; stress and coping*

It is well documented that the perceived availability of social support is associated with a variety of positive outcomes (Cohen, 1992; Sarason, Sarason, & Gurung, 1997; Stroebe & Stroebe, 1996). These benefits range from faster recovery following surgery (e.g., Fontana, Kerns, Rosenberg, & Colonese, 1989; Helgeson, 1991) to fewer depressive symptoms (Schwarzer & Leppin, 1992). Perceived availability refers to a person's feeling that they have people to turn to for support in times of need. Based on the findings reported above, one might expect that actual supportive behaviors explain the beneficial effects of perceived support. However, many studies have reported that specific supportive behaviors either have no positive effect on well-being (Barrera, 1986; Bolger, Foster, Vinokur, & Ng, 1996) or may even be detrimental to the recipient (Barrera, 1986; Bolger, Zuckerman, & Kessler, 2000).

One study that presents this paradoxical effect was conducted by Lindorff (2000). Perceived availability of support was measured by asking participants to rate how satisfied they were with the availability of support in six different areas. Received support was measured by asking participants to think of people who helped them in those six types of situations and to rate the extent to which they were supported. The study found that perceived support was associated with reduced strain in both men and women but receipt of emotional support was associated with increased strain for men and had no effect for women.

Similar findings have been recently reported in diary studies. In a diary study in which couples under stress were asked to rate their daily levels of distress and whether they received support, Bolger et al. (2000) found that participants felt more anxious and depressed on days following their reported receipt of support. Gleason, Iida, Bolger, and Shrout (2003) used this same diary method to measure support and distress in a nonstressed sample and obtained similar results.

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Various explanations have been offered to explain why support receipt can be harmful (Coyne, Wortman, & Lehman, 1988). One theory is that receiving support poses a threat to the recipient's self-esteem insofar as the recipient feels that the provider thinks he or she cannot handle the problem alone (Fisher, Nadler, & Whitcher-Alagner, 1982). Other researchers suggest that inequity in support transactions can lead the recipient to experience feelings of indebtedness, which in turn can lead to increased distress (Dunbar, Ford, & Hunt, 1998; Gleason et al., 2003). Another account claims that receiving support may merely draw attention to the problem rather than being useful (Amarel & Bolger, 2005). Aspects of the support itself also may lead to negative consequences. For example, the support provider may engage in behaviors that are intended to be supportive but that in fact are not effective, such as minimizing problems, encouraging suppression, or giving unwanted advice. Also, support may sometimes be ineffective because the support provided does not match the specific needs of the recipient (Cutrona, 1990).

As promising as these explanations and theories might be, a central problem with the findings that support transactions can be harmful is that they have been based on correlational rather than experimental data. Studying naturally occurring support events is often the only way for researchers to examine the validity of these alternative explanations because it is difficult to manipulate support receipt experimentally (see Amarel & Bolger, 2005; Collins & Feeney, 2000; Feeney & Collins, 2001, for exceptions). Studies of enacted support and distress usually rely on participants' reports of ongoing life experience. These reports can be collected using diary designs in which participants record their mood and other experiences, such as support transactions, every day for a period spanning several days (e.g., see also Bolger, Davis, & Rafaeli, 2003; Bolger et al., 2000). Although daily diaries can document the association between receiving support on one day and having more anxiety and depression on the next, the interpretation of the association is still open to competing hypotheses. As most methodology textbooks are quick to point out (e.g., Aron, Aron, & Coups, 2005; Cohen, Cohen, West, & Aiken, 2003), there are usually several competing causal models that need to be considered when interpreting a correlation in survey data.

The most obvious competing model that is proposed by skeptics is that the direction of causation might be reversed. For example, it is possible that a causal link between enacted support and distress does exist but that it may stem from distress increasing support rather than the reverse. If that support is harmless but ineffective, periods of distress could be accompanied by periods of active support transactions, making it appear that support increased distress.

A second competing model that needs to be considered is that a third variable could influence both support and distress and result in a spurious association between them. Barrera (1986) observed that stressful life events can lead to both increased distress and increased support. If such events are noticed by a potential support provider who then provides ineffective support, this would create the false impression that support increases distress.

Competing Models for Support-Distress Association: Harmful Support Model

Researchers who use nonexperimental data to test causal hypotheses attempt to rule out alternate interpretations by building statistical models that adjust for them. We describe in detail one such statistical model that was proposed by Bolger et al. (2000) to show the harmful effects of received support (the Harmful Support Model). They used multilevel models (see, e.g., Raudenbush & Bryk, 2002) to describe how distress covaried with enacted support within couples over time and how the couples differed from each other in terms of the relation between support and distress. Because associations between concurrent support and distress could imply that distress leads to support as easily as they could imply the reverse, Bolger et al. (2000) focused on longitudinal associations, particularly with a 1-day lag. Specifically, they assumed that support on day $t-1$ led to a change in distress on day t. In addition, they included information about day $t-1$ distress in the models to rule out the possibility that the association between lagged support and current distress was an artifact of initial adjustment (Bolger et al., 1996; see Figure 1). In this type of model, the dependent variable can be described as residualized change in distress from day $t-1$ to day t (Kessler & Greenberg, 1981). The focus of our article is to evaluate how well this model does in adjusting for the two important competing models discussed earlier. We refer to the Harmful Support Model as the focal model, that is, the model that it is being tested.

Reverse Causation Model

The Reverse Causation Model (see Figure 2) proposes that distress in partner A in an intimate relationship is observed by partner B and that B offers social support to A on the same day. In the absence of any effect of the support, this process would induce a positive correlation between support events and increased distress over days. To make the model most competitive with the Harmful Support focal model, we assume that support in the Reverse Causation Model is neither harmful nor helpful. The idea that B would offer support to

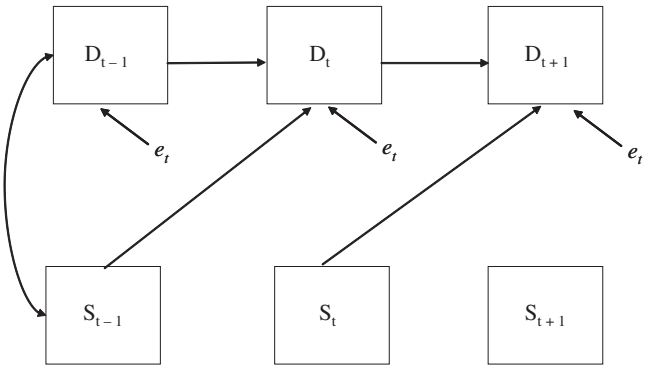

Figure 1 Harmful Support Model.

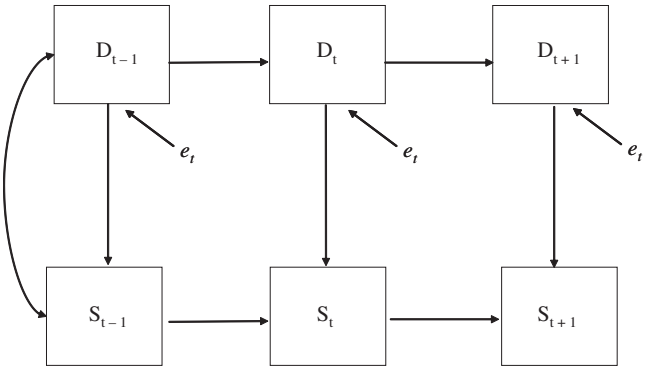

Figure 2 Reverse Causation Model: Distress mobilizes support.

A in response to distress is consistent with research by Revenson and Majerovitz (1990) and Hobfoll and Lerman (1988).

Third-Variable Adversity Model

The Third-Variable Adversity Model proposes that occurrences of stressful life events could lead to both increased support and increased distress. There is considerable evidence showing that adversity mobilizes support, at least in the short term (e.g., Bolger et al., 1996; Kaniasty, 1995; Norris & Kaniasty, 1996), and there is similar evidence that life events are related to increased distress (Kessler, 1997). As shown in Figure 3, we assume that the effects of a life event are experienced on both the same day of the event and on the following day. To retain the positive association between support events and distress, we assume that support is not effective in reducing distress.

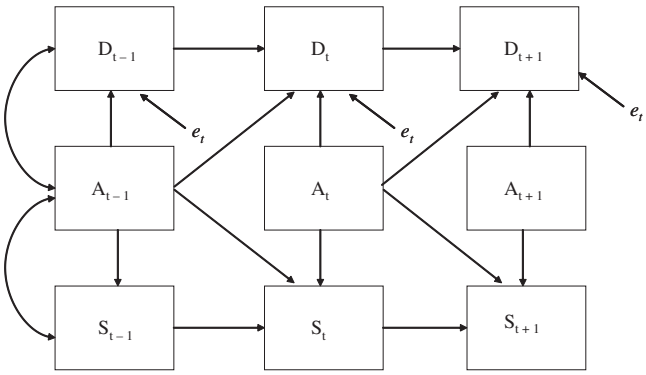

Figure 3 Adversity as Third Variable Model.

Statistical Analysis of Competing Models: Model Specification Error and Statistical Bias

As noted earlier, in nonexperimental studies, the investigator needs to anticipate plausible alternative models and to establish that the reported effect is maintained after adjusting for these competing explanations. The validity of these statistical adjustments, however, depends on whether the statistical models that are the basis of the adjustments are correctly specified. If a researcher's statistical model is incorrect, the estimated effects of the variables in that model are likely to be erroneous (in statistical terms, the effects are biased[1]) and inferences made from that model can be misleading (see, e.g., Fox, 1997). The question that skeptics must address is whether the degree of bias produced by model misspecification in a given analysis is large or trivial.

The magnitude and direction of model misspecification in ordinary linear regression has been well studied (e.g., Ramsey, 1969) but less is known about the effects of model misspecification in multilevel longitudinal analyses. In particular, multilevel models are more complicated than usual linear models in that adjustment at the individual (lower) level is not the same as adjustment at the average (upper) level (Kenny, Korchmaros & Bolger, 2003). Studying these effects in multivariate multilevel models is made more challenging by the fact that estimates are obtained iteratively rather than through explicit solving of equations such as is done for ordinary least squares regression (Raudenbush & Bryk, 2002). Therefore, assessing the impact of model misspecification in analyses of diary data is difficult to approach analytically. One tool that can be used to evaluate model specification issues critically is statistical simulation methodology. The goal of the current article is to illustrate how statistical simulation studies can be used to

examine the credibility of alternative explanations for the association between distress and support in diary studies. Specifically, we show how these methods can be used to estimate the degree to which a misleading association could be inferred if the Harmful Support Model were assumed in the analysis when a different process operated to generate the data.

OVERVIEW OF SIMULATION STUDIES

Simulation studies are ones that use mathematical equations and computer programs to investigate relations among variables under alternative models. For example, statisticians often use simulation studies to study how a statistical technique that assumes normally distributed variables works when the variables are not normally distributed. In such examples, the simulation is usually repeated many times to study the sampling distribution of the results (e.g., see MacKinnon, Warsi, & Dwyer, 1995).

Simulation was first introduced to social psychology by Abelson (1968) but since that time these methods have only been used by a handful of researchers (e.g., Hastie, 1988; Matthews & Harley, 1993; Stasser, 1988; Stasser & Taylor, 1991). Some of these applications have focused on understanding the potential impact of random noise on experimental data (e.g., Matthews & Harley, 1993), whereas others have focused on the long-term implications of complex theories (e.g., Mossler, Schwarz, Florin, & Gutscher, 2001).

Our application of simulation methods involves the creation of artificial data sets that reflect specific alternative statistical models. These are created with enough observations that patterns of bias are readily apparent, if they exist. Once a simulated data set is created, it can be analyzed with the same statistical procedures that are used on actual data. These analyses can apply the correct model (the model used to create the data) or various incorrect models to assess the potential spurious associations implied by these incorrect statistical models (e.g., Shrout, Bolger, & Seidman, 2003) or assist in theory development and clarification (e.g., Mossler et al., 2001). What makes simulated data sets especially valuable, and different from the data that social psychologists typically analyze, is that the causal models underlying the simulated data are known. This allows researchers to examine how misleading results can be if an incorrect model is used to analyze the data.

In this article, we show how longitudinal data sets can be created that approximate the type of data that is typical in diary studies of enacted social support. These data sets include what are known as fixed and random effects. Fixed effects are those effects that are common to all participants. They can be interpreted as average effects. For example, the finding of Bolger et al. (2000) that enacted support is associated with increased distress is a fixed effect. This association was reliably observed for the average couple. The extent to which this association varies from one couple to another is an example of a random effect. Depending on which couple is randomly sampled, the association can be larger or smaller.[2]

Accounting for random effects is especially important in longitudinal studies to recognize that individuals differ from each other systematically. For example, when participants report daily distress over 4 weeks, we expect that each person's daily report will fluctuate around his or her own average. Some participants are inclined to report higher levels of distress on most days and others are inclined to report lower levels. To make our simulations realistic with regard to individual differences, we included in our simulated data a random effect for individuals' baseline levels of distress.

The fixed effects in our simulated data sets are constructed to resemble causal models other than the focal model, the Harmful Support Model. The analysis proposed by Bolger et al. (2000) was used in an attempt to adjust for alternative explanations. If the adjustments are adequate, then the analysis should be able to distinguish data that follow the Harmful Support Model (see Figure 1) from data that arise from models such as the Reverse Causation Model (see Figure 2) or the Third Variable Adversity Model (see Figure 3).

The flowchart in Figure 4 demonstrates the basic simulation method. First, we specify the Harmful Support Model as the focal model that we plan to test (Step 1). We then specify the alternative model to be examined as an explanation for empirical findings that could be construed to support the focal model (Step 2). Once the alternative model has been specified, we explore a range of plausible parameter values that may be used in the simulation (Step 3). These parameter values refer to the effects of the different variables in the alternative model (e.g., support, distress, stressful events) on each other. We then use the smallest plausible effects to generate a data set that follows the alternative model (Step 4). Next we analyze the data from Step 4 using the focal model that we are testing, a model that we know is not the correct explanation for the simulated data (Step 5). Finally, we examine the results produced in Step 5 to see if the analytic plan used by proponents of the focal model leads to misleading results. That is, if the results from Step 5 suggest that support is harmful, we know the analysis is biased in a statistical sense (we answer "yes" to Step 6 in the diagram).

If bias is not observed in this first simulation, we return to Step 4. We alter the parameter values used to generate the data to increase the strength of the association between support and distress. We then repeat Steps 5 and 6. We continue to alter the parameter values, cov-

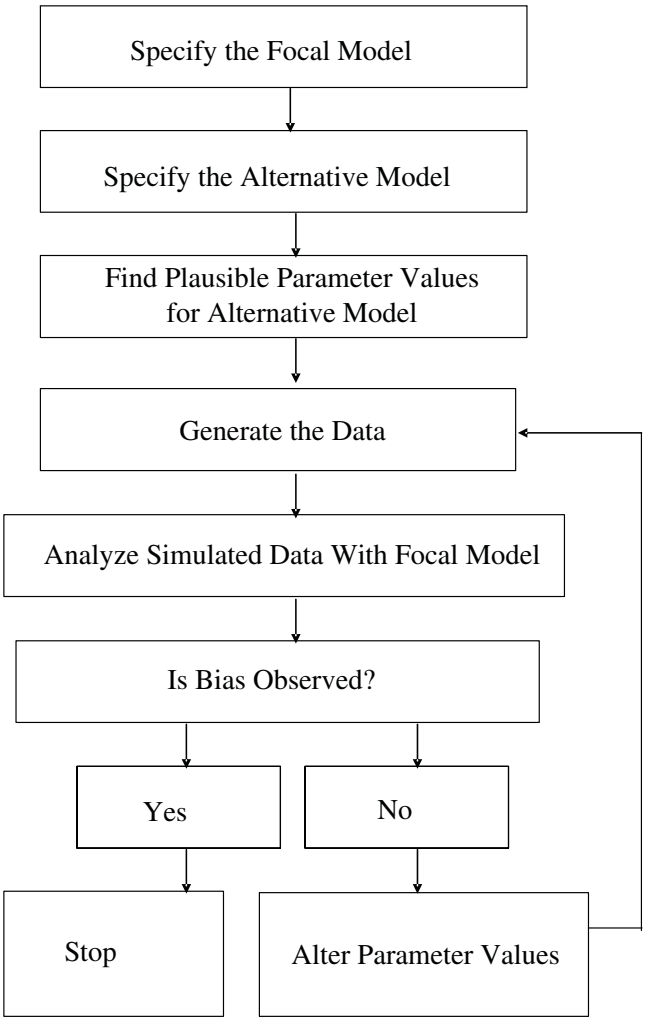

Figure 4 Flowchart demonstrating simulation method.

ering the entire range of plausible values, until bias is consistently observed or the range of plausible values has been exhausted without demonstrating bias.

To provide an overview of our simulation process, we ask you to consider the following analogy. Assume that you inherited your great aunt's art collection. An unsigned painting attracts your attention and you bring it to a local art expert who advises you that it might be a Van Gogh because the painting appears to have certain features that only he could produce. This is analogous to Step 1 in the flowchart in the sense that the painting is a data set and Van Gogh is a hypothesized model that may have generated the data. However, you cannot definitively prove your hypothesis that the painting is a Van Gogh. A skeptical friend of yours suggests that this painting is not the work of Van Gogh but was in fact painted by one of two well-known and highly skilled imitators who have the ability to reproduce those features that you believe are unique to Van Gogh's work. These two imitators are analogous to the plausible alternative models in Step 2 of the flowchart.

To rule out your friend's speculations about the painting being an imitation, you find examples of the imitators' work that resemble the focal painting in their style and Van Gogh period (Step 3) and you choose several to show your expert. The additional paintings that you choose to show the expert are analogous to the simulated data (Step 4). You know these two alternative paintings are not Van Goghs but you will have the expert examine them as though they are (Step 5). If the art expert cannot recognize that the reproductions are not by Van Gogh, the result calls into question his conclusion that the original painting is a Van Gogh (analogous to "yes" at Step 6). However, if the expert concludes that the reproductions are not Van Goghs, there is evidence to conclude that these impersonators are not plausible sources of your painting, lending some additional credence to the idea that Van Gogh is the true source (analogous to "no" at Step 6).

Returning to the substance of our research, we reiterate that the Harmful Support Model claims that persons who receive support on one day are likely to be more distressed the next day than they would have been had they not received support.

The specific form of the model is presented in Equation 1:

$$D_{it} = B_{i0} + B_1 D_{it-1} + B_2 S_{it-1} + r_{it} \quad (1)$$

where D_{it} represents distress on day t for participant i, D_{it-1} represents distress on day $t-1$, S_{it-1} represents support on day $t-1$, and r_{it} represents a random error term. A feature of Equation 1 that we will discuss below is that the intercept, B_{i0}, has a subscript i. The Harmful Support Model does not assume that all persons have the same intercept but rather assumes that each person can have his or her own typical level of distress.

STUDY 1: REVERSE CAUSATION MODEL

Our first simulation assesses whether a Reverse Causation could lead to data that, from a statistical modeling perspective, appear to follow the Harmful Support Model. The Reverse Causation Model states that the association between support and distress is due to distress causing support events. This model is similar to what Barrera (1986) described as the support seeking/triage model, where those individuals who show the

most distress receive or seek the most enacted support. Under this model, today's distress is dependent on yesterday's distress, and today's support is dependent on today's distress and yesterday's support (see Figure 2). We chose a same-day causal path for the Reverse Causation Model rather than a lagged causal path (i.e., a model in which distress on day $t-1$ leads to support on day t) because we believe that this version of the model is more likely to lead to a spurious association between support on day $t-1$ and distress on day t.[3]

Simulation Methods

We created simulated data for 1,000 "persons"[4] as if they were observed on 12 days with a binary (yes = 1, no = 0) report of whether support had been received each day and a distress measure that ranged from 0 (*not at all distressed*) to 4 (*extremely distressed*). SAS statistical software (SAS Institute, 2001) was used to generate the data (the syntax is available at www.psych.nyu.edu/couples/PSPB2005/Appen.doc). We emphasize results from a single sample with a large sample size because we are interested in demonstrating stable bias rather than sampling fluctuations. We chose the sample size to be large enough to reveal the pattern of bias regardless of the particular simulation run. The reader can verify with the program that the pattern of bias would be similar if the sample size were made larger (e.g., N = 2,000 or 3,000; see Appendix A at www.psych.nyu.edu/couples/PSPB2005/Appen.doc).

To simulate the Reverse Causation Model, we needed to specify two equations: one predicting distress and one predicting support. In the following equations for the Reverse Causation Model, D_{it} is distress on day t for person i, D_{it-1} is distress on the previous day (day $t-1$), S_{it} is support on day t, and S_{it-1} is support on the previous day, day $t-1$. The equation for S_{it} is written as a logistic regression model, where logit(S_{it}) refers to the log odds of support[5]

$$D_{it} = \beta_{0i} + \beta_1 D_{it-1} + r_{it} \quad (2a)$$

$$\text{logit}(S_{it}) = \alpha_0 + \alpha_1 S_{it-1} + \alpha_2 D_{it}\,. \quad (2b)$$

The intercept, β_{0i}, is the expected level of today's distress given that the previous day's distress was zero. The subscript, i, on the intercept reflects the fact that each simulated person is allowed to have a somewhat different value of the intercept, which reflects their usual level of distress. We initially specified values of the coefficients (βs and αs) in these formulas to be identical to values obtained from analyses of diary data. The values relating yesterday's distress to today's distress were obtained from Bolger et al. (2000) and the values relating yesterday's support and today's distress to the probability of today's support were reported in Seidman (2004). We built into the simulation variation that is consistent with individual differences. How different the intercepts are is reflected in a variance term of the random effect. To simulate the random effect for the intercept, we generated a distribution of intercept values using the normal distribution random number function in SAS. The mean of this normal distribution was .6 and the variance was .07. These values were based on those found by Bolger et al. (2000). Ninety-five percent of the intercept values in this distribution are expected to be in the range (0.08, 1.12).

We assumed that the other two regression weights in Equation 2a were the same for all the simulated subjects[6] and thus treated them as fixed effects. The coefficient, β_1, is the autoregressive parameter linking distress on day t to distress on day $t-1$. In our simulation, we assumed that the value was .5 (designed to be analogous to a standardized regression parameter and equivalent to an approximately 1 *SD* increase in distress). This term is identical to the effect found by Bolger et al. (2000). We included this effect in our model because it is a rather large effect that is consistently found in this type of data (Bolger et al., 2000; Gleason et al., 2003). The final term in Equation 2a is the residual, r_{it}, which represents the unexplained variation in distress within-subject from day to day. We used a normal random number generating function to create these residuals, with mean 0 and variance .15. Using the parameters described above to generate data, the resulting within-person standard deviation for distress was .41.

The second equation is for support. Because the focal model was based on the empirical report of Bolger et al. (2000) and they defined support events as binary (occurring or not occurring), we devised the competing models to also involve binary support events. To create systematic patterns of binary data, we model the log-odds of support using Equation 2b and then convert the log-odds values for each person on each day to probabilities. (For a useful introduction to these kinds of models, see Cohen et al., 2003, Chap. 13, or Kutner, Nachtsheim, Neter, and Li, 2005, Chap. 14.) The regression weights in Equation 2b were assumed to be the same for all simulated subjects. We set the intercept, α_0, to –2.0 so that the probability of support was about .20 on the day after a day when no support was given. This rate is consistent with the data of Bolger et al. (2000). We set α_1 to the value .7. This is the autoregressive parameter linking support on day t to support on day $t-1$. This value was close to the value of .90 found by Seidman (2004) in analysis of diary data. By choosing .7, support is about twice as likely on day t if support occurred on day $t-1$

TABLE 1: Estimates and Standard Errors for Study 1

	Model R Simulation Values				
Harmful Support Model Predictor	$\beta_1 = .5$, $\alpha_1 = .7$, $\alpha_2 = .7$	$\beta_1 = .6$, $\alpha_1 = 1.1$, $\alpha_2 = 1.1$	$\beta_1 = .7$, $\alpha_1 = 1.4$, $\alpha_2 = 1.4$	$\beta_1 = .8$, $\alpha_1 = 1.6$, $\alpha_2 = 1.6$	$\beta_1 = .9$, $\alpha_1 = 1.8$, $\alpha_2 = 1.8$
Support $(t-1)$ $[\beta_2]$	–0.002 (.008)	0.001 (.008)	0.004 (.009)	–0.011 (.013)	–0.015 (.017)
Distress $(t-1)$ $[\beta_1]$	0.506** (.007)	0.607** (.007)	0.709** (.006)	0.812** (.005)	0.908** (.004)
Intercept $[\beta_0]$	0.592** (.013)	0.587** (.014)	0.578** (.015)	0.576** (.019)	0.567** (.028)
Intercept variance	0.075** (.005)	0.074** (.005)	0.073** (.005)	0.071** (.005)	0.071** (.005)
Residual variance	0.150	0.151	0.151	0.151	0.151

NOTE: The dependent variable is distress on day *t*. Column headers show Reverse Causation Model simulation values for the effects of lagged distress on current distress (β_1), lagged support on current support (α_1), and current distress on current support (α_2).
**$p < .01$.

than if it had not occurred. We included this effect in our model because, similar to the day-to-day distress relationship, the day-to-day support relationship is also strong. The coefficient, α_2, is the effect of today's distress on today's support. Because the model we are simulating predicts that as distress increases support increases as well, we assumed this was a positive effect and we assigned α_2 a positive value, .7 (i.e., support is twice as likely to occur for each point increase in distress). Although this effect is substantially larger than the distress → support link found by Seidman (2004), we used a larger effect to enhance our ability to discover potential bias.

Equation 2b does not have a residual or error term because binary random variation is created as a separate step once an expected probability is calculated. The binary variable is coded 1 if a generated uniform random variable (in the interval 0, 1) is less than the probability value and it is coded 0 otherwise. This so-called Bernoulli variation allows each simulated data set to be different, even if the expected probabilities are the same.

Although we initially set the parameters to the values just discussed, which were suggested by analyses of actual data, we subsequently varied the values to make the strength of the reverse causation even stronger, using a range of reasonable values. We began with a value of .5 (about 1 *SD*) for the effect of previous day's distress on today's distress (β_1) and increased it in increments of .1 until reaching a value of .9. We began with a value of .7 for the effect of previous day's support on today's support, α_1, doubling the likelihood of support. We increased this value in terms of the odds, beginning with a value that would make support 2 times as likely, then 3 times as likely, until reaching a value that made support 6 times as likely when support had occurred the previous day ($\alpha_1 = 1.8$). We also began with a value of .7 for the effect of today's distress on today's support (α_2) and increased this in the exact same manner just described for α_1. We chose to begin with this doubling effect because it is close to as large as one could reasonably expect based on analyses of factors affecting the probability of support, which rarely exceed this size (Seidman, 2004). We used these large increases in the effect size because we wanted to be able to detect bias with as few simulations as possible. We altered all three parameters simultaneously, such that when we increased one parameter by a step, we did the same for the other two.

Results

We analyzed the five sets of data assuming (incorrectly) that the Harmful Support Model was the correct model.[7] We carried out the analysis with the MIXED procedure of SAS (SAS Institute, 2001). Table 1 shows the regression coefficients and standard errors for each term in the Harmful Support Model. Because the intercept, intercept variance, residual variance, and lagged distress were part of the model used to generate the data, the regression coefficients in the table are very close to those used in the simulation itself. Of primary interest are the values in the top row, indicating the size of any spuriously produced association between lagged support and distress. When yesterday's distress is statistically controlled, the spurious association of yesterday's support on today's distress is eliminated. Not only were all the effects of lagged support statistically insignificant but the estimates themselves were very small. This means that there is no evidence of a spurious association between today's distress and yesterday's support when the Harmful Support Model, a model known to be incorrect, was used to analyze the data.

For completeness sake, we note that no bias is observed for the lagged effect of distress either. The estimate shown in the second row of Table 1 corresponds closely to the β_1 values shown for each column head. Because the Harmful Support Model (Equation 1) simply expands on Equation 2a of the Reverse Causation Model, this is expected. We also note that the estimates

of the intercept mean and variance, as well as the residual variance, are very similar to the numbers used to generate the data.

Discussion

We generated data that followed the Reverse Causation Model, a model in which distress was predicted by previous day's distress and support was predicted by previous day's support and concurrent distress. We analyzed simulated daily diary data with a misspecified multilevel model (the Harmful Support Model) in which today's distress is predicted by previous distress and previous support. We then repeatedly altered the parameter values used to generate the data and analyzed each successive data set until reaching the maximum plausible values for those parameters. We established that it is not possible to generate a spurious association between today's distress and yesterday's support when data that follow the Reverse Causation Model are analyzed with the Harmful Support Model. Even when the effects of previous distress on today's distress, previous support on today's support, and current distress on today's support were as large as one could reasonably assume, it never resulted in a significant effect of support on distress. The results of our simulation suggest that a reverse causal relationship between concurrent support and distress cannot account for empirical findings of Bolger et al. (2000) that previous support is associated with increases in current distress. The Harmful Support Model includes lagged distress (yesterday's distress predicting today's distress) to adjust for other determinants of distress besides enacted support, and this adjustment successfully accounted for the association between support and distress induced by the Reverse Causation Model.

STUDY 2: THIRD-VARIABLE ADVERSITY MODEL

We now consider the second competing model, whereby some adverse, stressful event occurs on a particular day and affects both support and distress. Similar to the Reverse Causation Model, this model is consistent with a version of the support-seeking/triage model described by Barrera (1986), in which both distress and support are associated with stressful events. In our example, a negative event has an immediate impact on mood and support and continues to have an impact on the following day, albeit to a lesser extent.

When developing the Adversity Model, we chose to represent the effects of adversity on same day distress, as well as on the lagged day distress. We believe this alternative model is a more stringent test of the Harmful Support Model than a simpler model that assumes only an effect of lagged adversity on current distress.

Figure 3 represents the Third-Variable Adversity Model for three diary days. In the figure, distress is dependent on the occurrence of an adverse event today, the occurrence of such an event on the previous day, and the previous day's distress; support is dependent on the occurrence of an adverse event today, the occurrence of such an event on the previous day, and the previous day's support. This relationship may make it appear that support increases distress when, in fact, a third variable, the adverse event, is responsible for the association.

Simulation Methods

As in the first simulation study, we created simulated data for 1,000 persons[8] as if they were observed on 12 days with a binary report of whether support had been received each day, a binary report of whether an adverse event had occurred each day, and a distress measure that ranged from 0 to 4. Once again, SAS statistical software was used to generate the data (the syntax is available at www.psych.nyu.edu/couples/PSPB2005/Appen.doc).

According to the Third-Variable Adversity Model, (a) the likelihood of support being received on a given day increases if support occurred yesterday, if an adverse event occurs on that day, and increases to a lesser extent if an adverse event occurred the previous day; and (b) distress on a given day is related to the previous day's distress, the occurrence of an adverse event occurred on that day, and the occurrence of an adverse event occurred on the previous day. We assume that support *has no effect* on distress. Similar to the model described in Study 1, this model implies two equations: one for distress and one for support. In the following equations for the Third-Variable Adversity Model, D_{it} is distress on day t, A_{it} is adversity on day t, and S_{it} is support on day t:

$$D_{it} = \beta_{0i} + \beta_1 D_{it-1} + \beta_2 A_{it} + \beta_3 A_{it-1} + r_{it} \quad (3a)$$

$$\text{logit}(S_{it}) = \alpha_0 + \alpha_1 S_{it-1} + \alpha_2 A_{it} + \alpha_3 A_{it-1}. \quad (3b)$$

As in Study 1, the values of coefficients in these formulas were chosen to reflect values presented in Bolger et al. (2000). We expect strong autoregressive effects for both distress and support. We expect weaker effects of the adverse event on distress and support on the day it occurs and even smaller effects on the day after it occurs. These small effects of adversity on distress are based on previous research by Gleason et al. (2003) that found that the effect of daily stressors on distress (including both minor and major occurrences) was .057, given a 5-point scale for distress.

Once again, a random intercept was included in the model and is denoted by the subscript i that follows the coefficient β_0. The intercept β_{0i} is the expected level of distress on a day when an adverse event does not occur, when the previous day's distress was zero. In our simu-

lation, we generated a distribution of intercept values using the normal distribution random number function in SAS. The mean of this normal distribution was .6 and the variance was .07. Ninety-five percent of the intercept values in this distribution would be in the range (0.08, 1.12).

We assumed that the other two regression weights in Equation 3a were the same for all the simulated subjects. The autoregressive parameter linking distress on day t to distress on day $t-1$, β_1, was assigned the value of .5, as in Study 1.

The coefficient β_2 is the effect of the occurrence of an adverse event today on today's distress. Because the model we are simulating predicts that distress increases on the day an adverse event occurs, we assumed this was a positive effect and we assigned β_2 a positive value, .20, a somewhat larger value than the .057 found by Gleason et al. (2003).

The coefficient β_3 is the effect of the occurrence of an adverse event yesterday on today's distress. Because the model we are simulating predicts that the occurrence of an adverse event on one day increases distress the following day, but to a lesser extent than it does on the day it occurs, we assigned β_3 a value of .10.

The final term in Equation 3a is the residual, r_{it}, which represents the unexplained variation in distress within-subject from day to day. We used a normal random number generating function to simulate these residuals, with mean 0 and variance .15. Using the parameters described above to generate data, the resulting within-person standard deviation for distress was .42.

Equation 3b represents the logistic probability function used to generate binary support data. We assumed that regression weights in Equation 3b were the same for all simulated subjects. The intercept α_0 is the expected odds of support on a day when an adverse event does not occur, when the previous day's support was 0.

The coefficient α_1 is the autoregressive parameter linking support on day t to support on day $t-1$. As in Study 1, we assumed that the value was .7. (Transforming this to odds, this means that support is twice as likely on day t if support occurred on day $t-1$ than if it had not occurred.)

The coefficient α_2 is the effect of the occurrence of an adverse event today on today's support. Because the Third-Variable Adversity Model predicts that an adverse event increases the likelihood of support, we assumed this was a positive effect and we assigned α_2 a positive value, .5. (That is, support is 1.65 times more likely to occur on a given day when an adverse event occurs on that day.) Previous analyses of diary data showed that small daily hassles had no effect on the likelihood of support provision (Seidman, 2004), so we chose this relatively small effect to begin our simulation. The Third-Variable Adversity Model also predicts that support increases on the day after an adverse event occurs. Realistically, we believe the effect would be smaller than on the day it occurs, and so we assigned α_3 a value of .01. (That is, support is 1.01 times more likely to occur on a given day when an adverse event occurs on the previous day.)

As in Study 1, we computed the probability of support using Equation 3b for each day and then used this probability in a Bernoulli trial to generate a binary (0, 1) outcome. Specifically, we coded support as 1 if the probability value was greater than a random number selected by a uniform random number–generating function (with a range of 0 to 1) and coded support as 0 otherwise. This two-step procedure introduces random variation into the support variable, even though the probability of support is structured by Equation 3b. We used the same principle to generate binary indicators of whether adverse events occurred on a given day. In the Third-Variable Adversity Model, these events are represented as fateful—the events are occurring randomly rather than as a function of characteristics of the subject (Shrout et al., 1989).

As seen in Figure 3, neither distress nor support is related to the probability of an adverse event occurrence. For each subject, we assumed that such an event could occur any day with probability .70, a value consistent with research by Seidman (2004) on the probability of the occurrence of minor stressors. The binary indicator was generated as a Bernoulli trial in the way just described.

To examine a range of parameter values for this model, we initially varied the values of α_2, α_3, β_2, and β_3. However, increasing the value of α_3 had no effect on the results, so we retained the original value of .01. Increasing the value of β_2 (the effect of today's adverse event on today's distress) also does not increase the size of the spurious association between today's distress and yesterday's support, but we increased it whenever we increased β_3 (the effect of yesterday's adverse event on today's distress) because it is implausible that the effect of the adverse event on the day after it occurs would be larger than on the day that it occurs. Therefore, we gradually increased the values of α_2, β_2, and β_3, beginning with the starting values discussed earlier and ending with final values of .4 for β_2, .3 for β_3, and .8 for α_2.

Analysis Methods

The various simulated data sets that were created under the Third-Variable Adversity Model with parameter values set as described above were analyzed using the focal model (the Harmful Support Model) in which today's distress is predicted by yesterday's support and lagged distress is adjusted statistically. The within-subject part of this multilevel model is described by Equation 1.

TABLE 2: Estimates and Standard Errors for Study 2

	Model A Simulation Values		
	$\beta_1 = .50$, $\beta_2 = .20$, $\beta_3 = .10$, $\alpha_1 = .70$, $\alpha_2 = .50$,	$\beta_1 = .50$, $\beta_2 = .30$, $\beta_3 = .20$, $\alpha_1 = .70$, $\alpha_2 = .70$,	$\beta_1 = .50$, $\beta_2 = .40$, $\beta_3 = .30$, $\alpha_1 = .70$, $\alpha_2 = .80$,
Harmful Support Model Predictors	$\alpha_3 = .01$	$\alpha_3 = .01$	$\alpha_3 = .01$
Support $(t-1)$ $[\beta_2]$	–0.001 (.008)	0.017 (.009)	0.032** (.009)
Distress $(t-1)$ $[\beta_1]$	0.525** (.008)	0.560** (.007)	0.600** (.007)
Intercept $[\beta_0]$	0.785** (.016)	0.845** (.017)	0.878** (.018)
Intercept variance	0.060** (.004)	0.050** (.004)	0.041** (.004)
Residual variance	0.165	0.182	0.207

NOTE: The dependent variable is distress on day *t*. Column headers show Adversity Model simulation values for the effects of adversity on same day distress (β_2), next day's distress (β_3), same day support (α_2), and next day's support (α_3).
**$p < .01$.

As in Study 1, we carried out the analysis using the MIXED procedure of SAS and specified the intercept of the model to be a random effect.

Results

Although we systematically constructed many variations of the Third-Variable Adversity Model, it will be most useful to the reader to see the results of three of them. The three simulated data sets were analyzed by assuming (incorrectly) that the Harmful Support Model was the correct model.[9] Table 2 shows the regression coefficients and standard errors for each term in the Harmful Support Model. Because the intercept, intercept variance, residual variance, and lagged distress were part of the model used to generate the data, the regression coefficients in the table are very close to those used in the simulation itself. Of primary interest are the values in the top row, indicating the size of any spuriously produced association between lagged support and distress. The first column shows values (for β_2, β_3, α_2, and α_3) that are modest in size, and in this case, no bias is observed for the lagged support variable. That is, there is no false effect of lagged support on today's distress. In the second column, the coefficient for lagged support is positive, consistent with the Harmful Support theory, but the coefficient is small and not statistically significant. In the third column, however, the coefficient is larger and significant. Even though the data were created by a model that had no causal link between lagged support and distress, the analysis by the Harmful Support Model implies that support on one day would lead to increased distress on the next. This bias occurs because the Harmful Support Model does not take the adversity causal process into account.[10]

In the third column, the parameter values for the Third-Variable Adversity Model were .4 for β_2, .3 for β_3, and .8 for α_2. We wondered how feasible the data were that were generated with these parameter values. The largest mean distress for a simulated subject was 2.26, and the maximum probability of support was .69; these values are feasible. However, the values we used for the effects of the adverse event on support and distress were larger. Expressed as an effect size, for example, the effect of an adverse event on distress was nearly 1.0, which is larger than what Cohen (1988) calls a large effect.[11]

Discussion

We generated sets of data that follow the Third-Variable Adversity Model. In these data sets, distress was determined by previous day's distress and the occurrence of adverse events on concurrent and previous days, and support was determined by previous day's support, the occurrence of a concurrent adverse event, and the occurrence of an adverse event on the previous day. We analyzed the data with the Harmful Support Model, in which today's distress is predicted by previous distress and previous support and is misspecified in that it did not consider the concurrent adverse event. A spurious association between today's distress and yesterday's support was not always observed when the misspecified model was used but it was observed for one of the three sets of parameter values that we reported in Table 2.

These analyses illustrate that a spurious association due to an omitted variable can result from analyses of daily diary data using multilevel analysis but that the magnitude of the causal effect of the omitted variable must be relatively large. The simulated data did appear to be realistic in that the distributions of distress and support levels were similar to those reported by Bolger et al. (2000) and others in the literature. However, the adverse event had to increase negative mood by .4, which is about as large as one within-person standard deviation. It is almost as large as the effect of changing one scale point on the previous day's negative mood.[12] We only consid-

ered such large effects of an adverse event because only at this level was bias apparent.

In studies of actual daily distress, the size of the effect of daily stressful life events appears to be much less than the value that we assumed above. Gleason et al. (2003), for example, found that the effect of concurrent stressful events on distress was only .057, an effect one-sixth of the size of our simulated effect. In addition, Seidman (2004) found that daily stressful events had no significant relationship to distress, suggesting that our simulated effect of support was large as well. In fact, there is a long history of stressful life event research that suggests that the impact of events is subtle (Rabkin & Struening, 1976).

GENERAL DISCUSSION

Although daily diary methods are well suited for looking at psychological processes over time, the analysis of diary data requires multilevel statistical models that can be challenging to evaluate critically. We used statistical simulation methods to examine two alternative explanations for the counterintuitive but empirically supported association between the receipt of support and increased distress. In the first model, the Reverse Causation Model, we considered the possibility that rather than support increasing distress, distress may increase support. In this model, levels of distress on one day influenced support on that day, possibly making it appear that support increases distress. In the model examining a stressor as a third variable, the Third-Variable Adversity Model, we considered a situation in which the occurrence of an adverse event increases both support and distress, leading to a spurious association between the two variables. For this model, we examined an event that has an effect on support and distress on the day it occurs and then a lesser effect on the day after it occurs.

Only one of the alternative models produced data that incorrectly suggested that support is costly when analyzed using a statistical model (the Harmful Support Model) that is similar to that assumed by Bolger et al. (2000). Although the Third-Variable Adversity Model yielded simulated data that appeared to be realistic, the size of the effects of the omitted adversity variable on both distress and support had to be set to be very large for a spurious association between today's distress and yesterday's support to be observed. The size of this effect is quite large compared to other types of effects of daily variables such as minor stressors (e.g., Gleason et al., 2003; Seidman, 2004).

When considering the results of both of these simulation studies, it is important to note that the data sets we generated had very large sample sizes: 1,000. We used these large samples to obtain stable estimates so that the presence and magnitude of potential spurious associations could be evaluated without regard to sampling variability. The important effects that we observed in analyses had standard errors of around .01 for effects approximately .03 to .04. In analysis of data simulated under the Third-Variable Adversity Model for which we consistently found a spurious association between today's distress and yesterday's support, the effect of support on distress was approximately .03, an effect that is about one fifth of the size of the effect of support on distress that was reported by Bolger et al. (2000). To produce a spurious association of the same magnitude as that found by Bolger et al. (2000), the effect of the adverse event on support and distress would have to be even larger than the one used in our final simulation.

Before we discuss additional alternative explanations for the support-distress link, it is important to address the distinction between enacted support as perceived by the recipient and actual, objectively measured, enacted support. Diary studies showing a support-distress link have relied on the recipient's perception of the occurrence of a supportive event, and the goal of the current research was to examine whether this type of data can lead to a spurious association between support and distress. There is experimental evidence that at least some kinds of experimentally manipulated, enacted support can increase distress. In a laboratory study where undergraduates had to perform a stressful task, Amarel and Bolger (2005) found that obvious support attempts by a confederate led to increased anxiety.

The current simulation studies obviously did not consider all possible alternative explanations for the association between support and distress. For example, one reviewer pointed out that an association between support on day t and distress on day $t + 1$ could be induced by the following situation: Suppose distress on day t was immediately reduced by support on that day, but on the subsequent day (a nonsupport day) distress returned to its usual level. The return to the baseline could be associated with the lagged support event in a way that might support the Harmful Support Model. This alternative is possible and could be investigated using the methodology we have described here.

It is also possible that a stressful event could extend more than the 1 or 2 days considered in our models or that the relative size of its effects on support and distress could differ more than it did in our simulated data. With regard to the issue of reverse causation, one also could imagine a situation in which support is affected by not only that day's distress but earlier days' distress as well. However, we believe it is unlikely that considering these possibilities would lead to different conclusions than those that we can draw from our current simulations.[13] Readers who remain interested in alternative models are invited to modify the syntax we provide and to explore additional variations of these models.

Although we motivated our alternative models with two rather specific examples in this article, possible sources of spuriousness tend to come in these two varieties (Cohen et al., 2003, Chap. 3). Other examples, although conceptually different from those we tested, would be modeled similarly to the two that we chose to examine. Any third variable that simultaneously increases both support and distress would be modeled in the way that we modeled the occurrence of an adverse event. Therefore, we can generalize our results to any model that considers a third variable as a possible source of a spurious association between support and distress.

In addition, our research group has carried out analyses of empirical data that help to rule out other intervening variables that could potentially produce a spurious association between support and distress. In particular, Shrout, Herman, and Bolger (in press) have examined the buildup of stress due to the approach of a major stressful event, the effects of smaller transient troubles and tensions, and the effect of weekend breaks from the stress process. None of these adjustments changed the impression that received support was linked to worse mood on the next day.

It is also important to note that although the current simulation studies focused on day-to-day support transactions and mood measures, they could just as easily be focused on other time increments. In our discussion of the simulations, we treated each unit of time as 1 day, but we could have made that unit 1 hour, 1 week, or 1 year and our modeling of the process would remain unchanged.

There is no doubt that interpersonal support is a much more complex phenomenon than these two alternative models would suggest. For example, the amount or type of support provided, the nature of the stressful event, and characteristics of the provider or recipient could all affect the effectiveness or harmfulness of the support. The goal of the current research was to understand a circumscribed question: how the Harmful Support Model works with support as a binary variable. These same simulation methods could be used to examine continuous, rather than binary, support events or to manipulate other aspects of the supportive interaction.

The current simulation studies provide some evidence that the link between enacted support one day and increased distress on the following day is not a spurious association. Although the current studies do not completely rule out all possible causes of a spurious association, they lend support to the notion that there is a causal relationship between these two variables. In addition, recent research by Amarel and Bolger (2005) has provided experimental evidence for this causal relationship.

The practical implications of possible negative effects of enacted support are considerable for researchers who study interactions among intimate partners and for counselors who help couples resolve relationship difficulties. If enacted support can have negative effects on the recipient, it is important to determine when these effects are most likely to occur and how they can be prevented. Cutrona's (1990) emphasis on the importance of matching the nature of the offered support to the recipient's needs might diminish or eliminate the negative effects that have been reported in the literature. Gleason et al. (2003) offered another perspective on support harm reduction. They showed that if recipients of support can reciprocate, then the apparent negative effects of support are eliminated. Both of these perspectives suggest the importance of emphasizing dyadic dynamics rather than focusing on the individual when encouraging one partner to provide support to the other.

Our analyses suggested that the inclusion of the autoregression effect for distress helped account for both reverse causal processes and small effects of missing variables. Including the autoregression effect, however, cannot be recommended unconditionally. If the longitudinal process is a latent growth process rather than a dynamic change process, the autoregression effect can appear important and misleading (Rogosa & Willett, 1985). Care should always be taken to specify the alternative models and to study them critically.

We recommend that critical evaluation of alternative models, using simulation methods, can commence even before empirical data is collected. In conjunction with a careful review of the literature, statistical simulation studies can pit competing theories against each other to highlight what kinds of empirical studies are likely to make a difference. In addition, because one knows the causal model underlying simulated data, it is possible to examine the potential threat of spurious associations by analyzing simulated data with models that are known to be misspecified. This approach is likely to be especially useful when theories are represented by complex statistical models, such as the social relations model (e.g., Cook, 1994).

NOTES

1. Statistical bias means an estimate of a parameter is systematically different from the desired value. That is, even if the sample were made to be very large, the estimate and the parameter value would be different. Statistical bias can be quantified. When it exists, it can be subtle or large.

2. The methods we use are technically generalizations of repeated-measures mixed models from ANOVA (see Diggle, Heagrty, Liang, & Zeger, 2002; Littell, Milliken, Stroup, & Wolfinger, 1996). Similar to repeated-measures ANOVA, our analysis recognizes that subjects are "randomly selected" for study and that the influences on the repeated measures can be construed to be the same (fixed) for all subjects.

Unlike traditional ANOVA methods, the repeated measures are fit by an explicit regression model rather than being expressed as a set of within-person planned contrasts.

3. One reviewer wondered why we did not simulate this model with lagged rather than concurrent distress leading to support. If we used a lagged model to simulate the data, we would be looking for a spurious association between distress (on day $t + 1$) and support (on day t) that is caused by elevated distress 2 days prior (on day $t - 1$) to the dependent distress in the Harmful Support Model (on day $t + 1$).

4. We also carried out 100 simulations using a sample size of 100, yielding similar results to those described here. For the final simulation shown in Table 1, we obtained a significant effect of support for 2 of these 100 simulations.

5. Logistic regression was used because the outcome is binary. We wanted the likelihood of a support event to depend on an additive combination of previous support and current distress and the log odds of support is well suited for such linear models (see Cohen, Cohen, West, & Aiken, 2003). The log odds can be related in reasonable ways to probabilities. For example, on days when there is a 50% chance of a support event occurring, the odds are 1 to 1, and the logit (S_{it}) = 0.

6. We chose not to include random effects for these coefficients because it did not affect our results and only added noise to the data and unnecessary complexity to the model.

7. By analyzing the simulated data with a simple model predicting today's distress with yesterday's support without adjusting for yesterday's distress we were able to demonstrate that the data simulated under the Reverse Causation Model did show an association between distress and previous support. This is analogous to a manipulation check. With the initial (most realistic) values of the parameters, the estimate for the effect size of support is 0.064 ($p < .001$), suggesting that distress is .06 points higher following a supported day relative to an unsupported day. Although this is a small effect, it is completely spurious in that the Reverse Causation Model (Equations 2a and 2b) contains no effect of support on distress.

8. We also carried out 100 simulations using a sample size of 100, yielding similar results to those described here. For the final simulation shown in Table 2, we obtained a significant effect of support for 30 of these 100 simulations.

9. As in Study 1, we initially checked that the Adversity Model induced an association between distress on day t and support on day $t - 1$. When estimated using a simple model that defined distress on day t as the outcome and support ($t - 1$) as the explanatory variable, we found that all sets of parameter values under the Adversity Model produced significant associations, which lead to bias in the simplest misspecified model.

10. When the simulated adversity variable (the cause of the potential spurious association) is entered into the analysis model, the bias disappears even for the third column of Table 2. Adjusting for the cause of a potential spurious association can eliminate any bias.

11. As in Study 1, the estimates of the intercept mean and variance, as well as the residual variance, are very similar to the numbers used to generate the data.

12. The simulations were constructed so that the variation of the negative mood was similar to that reported by Bolger, Zuckerman, and Kessler (2000). Their mood measure was based on the POMS (Lorr & McNair, 1971) and it varied from 0 to 4.

13. Unpublished analyses of diary data have shown that longer lags have little or no effect on support or distress.

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