

Do Adverse Life Events Trigger Atypical Symptoms?

TO THE EDITOR: In their study published in the October 2007 issue of the *Journal*, Matthew C. Keller, Ph.D., et al. examined adverse life events and individual symptoms of depression. However, it appeared that “atypical” symptoms, such as overeating and hypersomnia, were disproportionately associated with no particular adverse events (1). This contrasts a significant body of research suggesting that atypical symptoms are highly reactive to external conditions and triggers. Two key examples are overeating and hypersomnia in the context of either seasonal affective disorder driven by environmental light conditions (2) or Columbia group atypical depression, in which the trigger is usually an otherwise minor interpersonal event (3).

Given that atypical episodes are characteristically driven by the physical or social environment, it is a thought-provoking speculation as to why a large proportion of subjects reported no cause for their atypical symptoms in the study conducted by Dr. Keller et al. One major issue that was not discussed by the authors is that seasonality and decreased light availability were not considered in the overall study design. Seasonality and decreased light availability are particularly pertinent to overeating and oversleeping in the context of seasonal affective disorder and its milder variants, which occur in a majority of individuals at the same geographic latitude as Virginia (4). The authors might wish to consider separate analysis of data limited to the Spring/Summer versus Fall/Winter periods to assess these important seasonal effects.

A second factor to consider is that on average, atypical symptoms and their environmental triggers may be less salient to individuals compared with melancholic symptoms and their associated triggers. For example, in the case of weight loss and anhedonia secondary to a major loss, both the symptoms and life event stand out as markedly different compared with normal experience as a result of their severity, ego-dystonic nature, and relatively clear, delineated time course. In contrast, in the case of overeating episodes or fatigue triggered by minor interpersonal slights, for example, neither the symptom nor its external trigger will consistently stand out as markedly different compared with normal experience. Over time, the signal-to-noise ratio for atypical symptoms and associated triggers would thus be very low relative to melancholic symptoms. The end result would be an underestimation of the importance of external events in the causation of atypical relative to melancholic symptoms.

Thus, while the overall conclusion that different adverse life events are associated with distinct depressive symptoms is strongly supported in the article by Dr. Keller et al., the specific finding that atypical symptoms are often associated with no adverse life events does not reflect the actual importance of environmental factors in triggering this symptom cluster. The conclusion implied by the data as currently presented—i.e., that melancholic symptoms are more influenced by external circumstances than atypical symptoms—may ultimately reflect more of a measurement and reporting issue than actual clinical reality.

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Dr. Levitan reports no competing interests.

This letter (doi: 10.1176/appi.ajp.2008.07121958) was accepted for publication in January 2008.

Dr. Keller Replies

TO THE EDITOR: We welcome the opportunity to respond to Dr. Levitan's thoughtful letter regarding our article. He has expressed that our finding that atypical symptoms were elevated among those subjects who could think of no specific cause of their dysphoric episodes (the “nothing” adverse life event) is at odds with previous research showing that atypical symptoms are highly reactive to external conditions. According to DSM-IV, atypical symptoms are mood reactivity (the opposite of our measure of anhedonia), hypersomnia, appetite gain, leaden paralysis (related to our measure of psychomotor retardation), and rejection sensitivity (unmeasured). Contrary to Dr. Levitan's claim, however, appetite gain, hypersomnia, anhedonia, and psychomotor retardation all vary significantly across adverse life events in both the between- (Figure 2 in the article) and within- (Figure 1 in the article) person samples. This result was maintained in a reanalysis of our data in which we excluded the “nothing” adverse life event, indicating that these five atypical symptoms are indeed sensitive to external conditions. Moreover, the most consistent symptoms for the “nothing” adverse life event (Table 2 in the article) are fatigue and appetite gain, with little (relative to other adverse life events) appetite loss, trouble concentrating, or sadness (“blues”). It should be noted therefore that appetite gain is the only atypical symptom in this cluster. Indeed, the symptom pattern following the “stress” adverse life event was more consistent with atypical depression than the symptom pattern following the “nothing” adverse life event. Thus, our results do not show that atypical symptoms are unresponsive to external conditions.

Dr. Levitan's second point is that certain adverse life events and certain corresponding symptoms may be less salient than others, leading to a weakening of certain adverse-life-event-symptom relationships and to an under-reporting of some adverse life events. This is a valid critique. Our study design depended on participants' own attributions of what caused their dysphoric episodes. To the degree that these were incorrect, our results may inaccurately reflect the true

adverse-life-event-symptom correspondences. As noted by Dr. Levitan, such misattributions seem more likely to occur for amorphous adverse life events, such as the “nothing” adverse life event, than for clearly delineated events with specific onset times (e.g., deaths, romantic breakups, failures, conflicts, scares). Indeed, the “nothing” adverse life event is probably a mixed bag of causes, including both truly endogenous, unperceivable causes (e.g., vascular dysfunction, biamine dysregulation) and external causes that are difficult to perceive (e.g., changes in the season, diet). Therefore, in agreement with Dr. Levitan, we feel that it is important to remember that participants’ causal attributions may have sometimes been incorrect and that this is probably especially the case in dysphoric episodes, for which participants could not determine a cause.

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Dr. Keller's disclosures accompany the original article.

This letter (doi: 10.1176/appi.ajp.2008.07121958r) was accepted for publication in January 2008.

How “Supportive” Is Internet-Based Supportive Psychotherapy?

TO THE EDITOR: In their article, published in the November 2007 issue of the *Journal*, Brett T. Litz, Ph.D., et al. presented thought-provoking preliminary data on Internet-assisted, cognitive behavioral self-management of posttraumatic stress disorder (PTSD) symptoms (1). In a report that emphasized technology and downplayed human contact, however, it might have been helpful to clarify certain details pertaining to the control intervention. A randomized study is only as credible as its control intervention, which raises conundrums. What exactly is Internet supportive counseling—the control condition—in this trial? Furthermore, how much therapist contact did subjects actually receive?

One imagines that supportive counseling would require affective mirroring and interpersonal warmth. Although the study design included a 2-hour initial meeting between the subject and therapist and allowed “periodic and ad lib study therapist contact via e-mail and telephone” (1, p. 1677), it was not clear how much direct human contact and loving kindness the supportive counseling patients received. Although therapists were “instructed to be empathic and validating” (1, p. 1681), e-mail in particular can obscure affect. The fact that patients read about stress and its management and wrote about “daily nontrauma-related concerns and hassles” (1, p. 1681) does not actually explain how the treatment was supportive. The authors described data on the frequency of Internet sessions but not on the background e-mail and phone contacts. It may have been helpful if they had commented further on how frequent, how long, and how supportive the interpersonal contacts were in each cell.

Training good supportive therapists requires a great deal of work (2). Although the article emphasized the study web site, it omitted any description of the training and prior experience of the therapists involved. Did these same therapists back up both the cognitive and supportive web sites? If so, could this have introduced allegiance bias (3) into the study?

Were attempts made to monitor therapist adherence to the respective treatments?

Finally, the authors described their cognitive web site at length, but relatively little about its supportive counterpart was mentioned. What features of the latter make it “supportive”?

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Dr. Markowitz receives royalty payments for psychotherapy-oriented books.

This letter (doi: 10.1176/appi.ajp.2007.07121853) was accepted for publication in December 2007.

Dr. Litz Replies

TO THE EDITOR: We appreciate Dr. Markowitz’s queries pertaining to the role of interpersonal contact in our Internet-based program. He raises a number of questions about our article, which he states “emphasized technology and downplayed human contact.” It is important to note that our self-management cognitive behavioral therapy (CBT) *intentionally* reduces the role of human contact with the objective that more people will receive the care they need. The model is germane because many people 1) are reluctant to seek traditional services, 2) live in remote regions where expert care is unavailable, and 3) are unable to access services because the demand exceeds the resources. In an ideal world, there would be no barriers to care, but it is imperative to recognize the sobering reality that most survivors of trauma do not receive evidence-based mental health services (1). Telehealth therapies may be less efficacious because they do not provide intensive human connection and oversight, but there is an unequivocal public health need to overcome barriers to care through alternative methods of therapy delivery.

Dr. Markowitz suggests that a supportive counseling program should provide “interpersonal warmth.” Our supportive counseling program followed previous psychotherapy trials by ensuring that it 1) did not contain active CBT skills and 2) involved the same therapist contact time (2). The issue concerning the telephone and e-mail contacts with patients in the respective conditions is an important one, and our analyses indicate that there were no significant differences between patients in the two conditions in terms of the total number or length of phone calls or e-mail messages. It should also be noted that the supportive counseling program resulted in a pre-/posttreatment effect size of 1.1, which is actually larger than most supportive counseling programs offered in traditional therapy formats (3). This suggests that the supportive counseling program was a change agent and provided