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Commentary—Pre- and Posttrauma Predictors of Posttraumatic Stress Disorder Symptom Severity: Reply to van der Velden and van der Knaap (2017)

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Utilizing data from a longitudinal study spanning five decades and thirteen waves of data collection, our study (Ogle, Rubin, & Siegler, 2016) examined the most extensive set of factors associated with posttraumatic stress disorder (PTSD) in a single sample of participants to determine their independent and combined utility in accounting for PTSD symptom severity. Our primary prediction model included 15 pre- and post-trauma constructs identified as PTSD risk factors in three meta-analyses (Brewin, Andrews, & Valentine, 2000; Orth & Weiland, 2006; Ozer, Best, Lipsey, & Weiss, 2003), and 12 individual difference and behavioral health factors that have received substantial empirical support since the PTSD meta-analyses were published. This model explained 56% of the variance in DSM-IV TR PTSD symptom severity; 10 of the 12 constructs that explained unique variance were measured after the traumatic event. In contrast, an analysis restricted to a subset of 15 predictors measured before the target trauma occurred explained only 16% of variance in PTSD symptom severity. Based on these results, we concluded that, “individual difference measures assessed after the target trauma occurred, including insecure attachment, depressive symptoms, and a lower perceived ability to cope with stress, in addition to measures related to the current trauma memory, including its self-rated severity, centrality to identity, frequency of involuntary recall, and the strength of visceral reactions to the memory, emerged as strong statistical predictors of PTSD symptom severity, whereas pre-trauma factors, such as socio-demographic characteristics and parental history of psychopathology, did not (p. 279).” As we noted in Ogle et al., our findings are consistent with results of the PTSD meta-analyses in showing that factors that follow a trauma better account for variance in PTSD symptom severity than characteristics of the individual or their life history that precede the traumatic event.

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In contrast to our study, the commentary (van der Velden & van der Knaap, 2016) analyzed data from a two-year longitudinal study of PTSD symptoms from recent traumas that occurred in the past year. Five predictors were included in the commentary's analytic model: three demographic variables (age, gender, education) and pre-trauma measures of mental health problems and emotional stability. Although no theoretical rationale or empirical support was provided for the selection of these predictors, each predictor tested has low predictive power in the published PTSD meta-analyses and combined explained only 13 to 17% of the variance in PTSD symptom severity in the commentary's analyses.

Despite critical differences between the two studies in research design, study characteristics (e.g., length of longitudinal study, time since the trauma), and number and kind of predictors, the commentary erroneously claims that our findings and conclusion regarding the role of post-trauma predictors in PTSD symptom severity should be rejected based on the results of their study. The commentary's extrapolation so far beyond the range of its own data is scientifically unsound. We next discuss the central points of the commentary's critique and present additional empirical support for our original conclusion.

The commentary wrongly claims that the results of our study are invalid based on the method we used to test content overlap. The best way to test for content overlap is to directly remove it, and we did so in our study using a commonly utilized method (e.g., Krause, Kaltman, Goodman, & Dutton, 2008; Lommen, Engelhard, van de Schoot, & van den Hout, 2014; Orth, Cahill, Foa, & Maercker, 2008). When the primary analysis in our study (Table 1) was reanalyzed to predict the PTSD Checklist (PCL, Weathers, Litz, Huska, & Keane, 1994) score with the items measuring the B1, B4, and B5 DSM-IV-TR (American Psychiatric Association, 2000) PTSD symptoms removed due to their potential overlap with measures of the phenomenological properties of the current trauma memory, the R^2 decreased from .56 to .52. With the exception of self-reported DSM-IV-TR A2, all predictors that explained unique variance in the original model were retained, and the magnitude of their coefficients changed $< |.04|$. In addition, 9 of the 11 predictors that explained unique variance were definitively post-trauma factors, as defined by their assessment after the trauma occurred for all participants in the sample. Moreover, when we tested for the effects of content overlap using the method suggested by the commentary, our results did not change substantially. Specifically, removing the properties of the current trauma memory from the model as predictors rather than from the predicted PCL score decreased the R^2 to .50. Compared to our original results, voluntary rehearsal, sleep disturbances, and previous traumas explained unique variance in PTSD symptom severity, the magnitude of the coefficients for all other predictors changed $< |.04|$, and 9 of the 13 significant predictors were definitively post-trauma factors.

In the two analyses described above, the majority of the predictors that explained unique variance in PTSD symptom severity were definitively post-trauma factors. Given that the remaining predictors could not be easily classified as pre- or post-trauma and that none of the predictors was a definitive pre-trauma factor, we conducted an additional analysis to test the explanatory power of pre-trauma compared to post-trauma measures. In this analysis, the 8 post-trauma trauma memory-related measures from our original primary model were added to our original prospective analysis (Table 2) which contained only pre-trauma

measures. Of the seven predictors that explained unique variance in this model, three were pre-trauma measures (standardized coefficients, +.14 depressive symptoms, +.09 neuroticism, +.06 previous traumas) and four were post-trauma measures (standardized coefficients, +.23 physical reactions, +.21 involuntary recall, +.20 event centrality, +.19 self-rated event severity). Adding the post-trauma memory-related predictors to the model increased the R^2 from .16 in the original prospective model to .53.

Collectively results from these three analyses demonstrate that the explanatory power of our original primary model was not the result of methodological artifact produced by content overlap. Predictors related to the phenomenological properties of the current trauma memory accounted for a small percentage of the total explained variance. Moreover, the pattern of our results was consistent irrespective of the statistical method used to assess content overlap, including removing the content-overlap items entirely from the model. These findings challenge the commentary's claim that our study rested on "the crucial assumption that eliminating a group of items from a PTSD scale that share content with predictors of PTSD assessed simultaneously, prevented that results were driven by potential content overlap." Contrary to this claim, the additional analyses presented here indicate that our conclusion regarding the role of content overlap in our results was an empirically supported statement, not an assumption. Furthermore, results from a more stringent comparison of definitively pre-trauma versus definitively post-trauma factors were consistent with our original findings in showing that post-trauma factors better account for PTSD symptom severity compared to pre-trauma factors.

Support for the commentary's call to reject our results was based on a set of analyses in which each of the three symptom-cluster subscales of the commentary's PTSD measure was removed from the total score and used to predict the sum of the remaining two subscales. When each subscale was added as a predictor to the commentary's regression models, the R^2 increased from between .13 and .17 to between .54 and .66. Removing one part of a measure with a Cronbach's alpha of .94 and using it to predict the sum of the other two parts accomplishes nothing more than demonstrate the strong psychometric properties of the measure. Using one group of items to predict the sum of the other two should yield high correlations. This finding is neither novel nor surprising; it follows from classical test theory and is unrelated to either the commentary's or our conclusions. Removing groups of items from a highly internally consistent scale in random fashion or to reflect certain content should not change the relations between the sum of the remaining items and other measures beyond the reduction expected from the Spearman-Brown Prophecy Formula. In fact, our past work with the PCL published in this journal (Berntsen & Rubin, 2015) demonstrated that removing conceptually overlapping items (symptom clusters) has little effect on analyses predicting the PCL. Thus, we view this analysis as a test of the psychometric properties of the commentary's PTSD measure, rather than a statistically defensible test of content overlap. Furthermore, we contend that the magnitude of the increase in the R^2 occurred because of the low predictive power of the commentary's measures; the 1/3 to 2/3 prediction had little to compete with among the set of measures with low predictive power in the multiple regression.

Based on the flawed logic of these findings, the commentary extrapolated that content overlap between the PCL items corresponding to the B1, B4, and B5 DSM-IV-TR symptoms and the predictors related to phenomenological properties of the current trauma memory similarly inflated the R^2 of our primary prediction model. The implication is that the R^2 of our model would decrease by a similar magnitude if the trauma memory characteristics were removed as predictors. Contrary to this prediction, results from the analysis presented above showed that removing the content-overlap items from our model as predictors had little impact on our results.

The commentary further wrongly claims that the results of our study should be rejected based on our use of cross-sectional data for some predictors. We noted the general concern regarding the use of cross-sectional data to draw conclusions about causality in our study, and acknowledged the limitations of the cross-sectional data in our specific dataset in the description and justification of our prospective analysis which included only pre-trauma predictors. Empirically, however, our use of cross-sectional data does not appear to have a major impact on the overall explanatory power of our primary prediction model. Data for subjective physical health, coping ability, and social support, 3 of the 13 cross-sectional predictors included in our primary model, were available from earlier waves of the longitudinal study from which our data were drawn. When we substituted measures of physical health, coping ability, and social support collected an average of two years prior to the assessment of PTSD symptoms for their cross-sectional counterparts in our regression model, the total R^2 was not diminished compared to the original model (.55 vs. .56). Compared to our original results, coping ability no longer explained unique variance. The magnitude of the remaining coefficients changed $< |.03|$.

In addition, our use of cross-sectional data for predictors related to the current trauma memory was ideal in that it allowed us to examine key aspects of mnemonic theories of PTSD. Specifically, according to the autobiographical memory theory (AMT) of PTSD (Ogle et al., 2016; Rubin, Berntsen, & Bohni, 2008; Rubin, Dennis, & Beckham 2011), particular characteristics of the current trauma memory, including the intensity of individuals' emotional and physical reactions to memories of their trauma, the frequency of involuntary and voluntary rehearsal, and the centrality of the current memory to the person's identity, promote the development and maintenance of PTSD. Cross-sectional assessments of these variables were therefore a strength of our study in that they permitted us to test key predictions of a theoretical model of PTSD. Inclusion of these measures also allowed us to compare the predictive utility of the central mechanisms of the AMT to other constructs identified as PTSD risk factors in the published meta-analyses.

The commentary also critiqued our study based on our examination of post-trauma rather than pre-trauma social support. We view this criticism as further evidence of the commentary's misinterpretation of the purpose of our study. Social support was included in our analyses based on the strength of empirical evidence for social support as a PTSD risk factor in the published meta-analyses; social support emerged as the first and second strongest predictor of PTSD in Brewin et al. (2000) (weighted $r = -.40$) and Ozer et al. (2003) (weighted $r = -.28$), respectively. In the 15 studies that measured social support in one or both meta-analyses, all assessments of social support were collected post-trauma. We

agree that additional prospective studies would advance our understanding of the role of social support in the development of PTSD symptoms. However, we do not view our decision to examine post-trauma social support as a weakness of our study; our study was designed to test constructs identified as PTSD risk factors in the published meta-analyses and the meta-analyses uniformly examined post-trauma social support.

In addition, we contend that the commentary espouses an overly-simplified theoretical view of pre- versus post-trauma measures. Consider gender and social support as examples. Although gender is the prototypical example of a factor that does not change over time as a function of trauma exposure, its effects pre- and post-trauma can vary substantially. Similarly, social support can also vary over time, with some traumas (e.g., death of loved one) eliminating existing social support. Moreover, the kinds of social support one needs to cope with trauma are often not the same kinds that are needed pre-trauma.

The commentary's call for our results to be rejected is also troubling due to its implication for clinical psychological science. Clinical treatment for PTSD is typically aimed at the modification of post-trauma factors to reduce symptoms, in part because pre-trauma factors, such as demographic characteristics of the individual and family history of psychopathology, are in the past or unchangeable. For example, two of the most effective therapies for PTSD, prolonged exposure (PE) and cognitive processing therapy (CPT), and the theoretical models from which they are derived (i.e., emotional processing theory and social cognitive theory, respectively) focus uniquely on post-trauma factors (U.S. Department of Veterans Affairs, 2016). Specifically, CPT is aimed at changing thoughts and beliefs about the trauma and their effects on emotions and behavior, whereas PE focuses on modifying affective responses to thoughts and situations that remind the individual of the trauma. Thus, the commentary's call for our results to be rejected in favor of its results which tested only pre-trauma factors is counter to the most successful therapeutic treatments for PTSD.

In conclusion, the commentary's call to reject our results is based on a statistically flawed argument. Its critique of the strength of post-trauma predictors in our analysis is neither empirically nor theoretically defensible. In response to the commentary, we provided additional empirical support for our original conclusion regarding the central role of post-trauma factors in accounting for PTSD symptom severity. Specifically, our results demonstrate that the trauma memory characteristics for which content overlap with the PCL was a concern accounted for a relatively small percentage of the total variance explained in our model. In addition, we show that the explanatory power of the model was not significantly diminished after accounting for potential content overlap, and that our basic results did not change with the statistical method used to assess content overlap. Collectively these analyses demonstrate that the statement the commentary erroneously refers to as the "crucial assumption" of our study is not an assumption but rather an empirically supported conclusion. Although we welcome critical analysis of our work aimed at advancing the scientific understanding of PTSD, we view the commentary as misguided.

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