Confronting Traumatic Experience and Immunocompetence: A Reply to Neale, Cox, Valdimarsdottir, and Stone

James W. Pennebaker Department of Psychology, Southern Methodist University

Janice K. Kiecolt-Glaser
Department of Psychiatry and Comprehensive Cancer Center
The Ohio State University College of Medicine

Ronald Glaser
Department of Medical Microbiology and Immunology
and Comprehensive Cancer Center
The Ohio State University College of Medicine

In their comment, Neale, Cox, Valdimarsdottir, and Stone (1988) argued that one should not conclude, based on our findings, that there is any positive effect of a brief psychotherapeutic writing treatment on immunocompetence. We disagree. Our experimental technique of having subjects write about the most traumatic experiences of their lives has now been shown to reduce health center visits for illness in three studies in two laboratories. In the Pennebaker, Kiecolt-Glaser, and Glaser (1988) study, we found that those subjects who wrote about traumatic experiences showed an improvement in one immunological assay (blastogenesis with the mitogen phytohemagglutinin) relative to control subjects who wrote about trivial topics. Furthermore, theoretically meaningful internal analyses demonstrated similar effects with another mitogen, concanavalin A. Although it is not possible to pinpoint the exact physiological links, consideration of all of the data indicate that confronting traumatic experience is, in all probability, physically beneficial.

We are writing this response to address questions raised by Neale. Cox. Valdimarsdottir, and Stone (1988). To paraphrase Neale et al.'s arguments: "Immune function cannot be concluded from blastogenesis. If it could be concluded, we are not sure that the effects reported by Pennebaker, Kiecolt-Glaser, and Glaser (1988) are statistically strong enough. If they are strong enough, they are probably artifactual. If they are not artifactual, then their immune effects are not longlasting enough. Even if they are longlasting enough, nothing can be concluded from them." We will briefly respond to these points.

Is Blastogenesis Related to Other Aspects of Immunity and Health?

Blastogenesis, the most commonly used functional assay in the clinical immunological literature, has long been a standard for determining the reactivity of cellular elements of the immune system (Fletcher, Baron, Ashman, Fischl. & Klimas, 1987). Briefly, it is a measure of the proliferation of lymphocytes when they are exposed to a foreign substance, a mitogen.

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Correspondence concerning this article should be addressed to any of the three authors: James W. Pennebaker, Department of Psychology, Southern Methodist University, Dallas, Texas 75275; Janice K. Kiecolt-Glaser, Department of Psychiatry, The Ohio State University College of Medicine, Columbus, Ohio 43210: or Ronald Glaser, Department of Medical Microbiology and Immunology, The Ohio State University College of Medicine, Columbus, Ohio 43210.

This proliferative response (in vitro) is thought to measure the ability of lymphocytes to replicate in vivo when they are exposed to a foreign invader such as a bacteria or virus.

Blastogenesis is one of the few immunological assays that has been reliably associated with relevant health parameters. Decreased lymphocyte proliferation reflects suppression of normal immune responses in a variety of immunodeficiency conditions, including acquired immune deficiency syndrome (AIDS) and AIDS-related complex (ARC) (Fletcher et al., 1987); less severe illnesses (e.g., Cogen, Stevens, Cohen-Cole, Kirk, & Freeman, 1982; Lumio, Welin, Hirvonen, & Weber, 1983), and even normal aging (Roberts-Thompson, Whittingham, Youngchaiyud, & Mackay, 1974). Finally, between-groups differences in blastogenesis have regularly paralleled differences in other immune functions across a large number of studies (Cogen et al., 1982; Fletcher et al., 1987; Glaser et al., 1985; Kiecolt-Glaser et al., 1987; Roberts-Thompson et al., 1974).

Are the Effects Statistically Real?

Contrary to the speculations made by Neale et al., there were, in fact, no initial differences between groups on any of the immune measures. Ironically, the covariance analyses suggested by Neale et al. yielded F values that were even more significant than our originally reported repeated-measures analyses of variance (ANOVAS). For covariance, p=.03 for the phytohemagglutinin (PHA) condition main effect and p=.10 for the concanavalin (ConA) condition by mitogen concentration interaction.

Are the Results Artifactual?

Our means for the blastogenesis data indicated that there was a decrease in the PHA response of lymphocytes from control COMMENTS 639

subjects, whereas lymphocytes from the experimental group showed a small increase. Ideally, of course, we would have preferred to see no change in the control group and an impressive enhancement of PHA reactivity in the experimental group. Why, then, did we get the pattern of effects that we did? We suggested the possibility of seasonal variation or of some unknown technical artifact. A recent article may provide another explanation. Fletcher et al. (1987) showed that the effect of sample storage time on lymphocyte proliferation could be far more profound than previously thought and that significant reductions in proliferation occur as sample storage times increase. To perform the assays for our collaborative study, blood samples that were drawn at the same time each evening were shipped overnight from Dallas to Columbus. The second set of blood samples was assayed about 4 hr later than the other two samples because of a delay in package delivery. Thus, the delay could account for the somewhat lower proliferative response at the second sample point.

More important, we doubt that the between-groups differences were artifactual because (a) the pattern of means was in the predicted direction, (b) the internal analyses dictated by a theoretically relevant individual difference found consistent effects for ConA, and (c) health center visits for illness came out in the same direction as the blastogenesis data.

Do the Immune Changes Persist After the Four Days of Writing?

Why were the maximal immune differences obtained on the final day of writing rather than during the 6-week follow-up? It would be nice to have found significant correlations between immunocompetence at Time 1 and illness incidence at Time 2. Unfortunately, the immune system is very complex and must be viewed probabilistically.

Consider two people under stress with impaired immune function. Whether they become ill depends on many variables including exposure to an infectious agent, previous history with the particular agent, and fluctuations in immunocompetence related to a variety of psychological, physiological, and genetic influences. Furthermore, if the individuals become ill simultaneously, one may recover quickly and the other quite slowly. Indeed, it will be difficult to predict the status of their respective immune systems 6 weeks in the future. In short, the state of the field of immunology does not allow us to pinpoint the direct causal link between immune change and subsequent illness episodes.

Can We Conclude That Our Writing Technique Positively Affects Immunocompetence?

Unlike Neale et al., we are cautiously optimistic. Three separate studies in two laboratories have now found that writing about traumatic experience is associated with improvements in physical health (Murray, Lamnin, & Carver, 1987; Pennebaker

& Beall, 1986; Pennebaker, Kiecolt-Glaser, & Glaser, 1988). Data now suggest that this technique may affect immunocompetence in a positive way. Although the results from our various studies are promising, we strongly encourage researchers to replicate and explore the objective health effects of confronting traumatic experiences under a variety of conditions.

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¹ In a recently completed study, Murray, Lamnin, and Carver (1987) compared the effects of brief psychotherapy with those of writing about traumatic experience or about trivial events. Although follow-up data could not be collected for many of their subjects, the number of days restricted due to illness in the 6 months following the study were lower for those writing about traumas (1.5 days) than for those writing about superficial topics (7.0 days), t(21) = 1.63, p = .059 (one-tailed test using the overall mean-square error term). We are indebted to Edward J. Murray for providing us with a copy of his raw data.