Emotional Disclosure Through Writing or Speaking Modulates Latent Epstein-Barr Virus Antibody Titers

Article *in* Journal of Consulting and Clinical Psychology · March 1994
DOI: 10.1037//0022-006X.62.1.130 · Source: PubMed

5 authors, including:

Michael Antoni
University of Miami and Sylvester Comprehensive Cancer Center
438 PUBLICATIONS **15,388** CITATIONS

SEE PROFILE

Mary Fletcher
Nova Southeastern University
289 PUBLICATIONS **10,040** CITATIONS

SEE PROFILE

Neil Schneiderman
University of Miami
599 PUBLICATIONS **21,649** CITATIONS

SEE PROFILE

Journal of Consulting and Clinical Psychology
1994, Vol. 62, No. 1, 130-140

0022-006X/94/$3.00

Emotional Disclosure Through Writing or Speaking Modulates Latent Epstein-Barr Virus Antibody Titers

Brian A. Esterling, Michael H. Antoni, Mary Ann Fletcher, Scott Margulies, and Neil Schneiderman

Healthy Epstein-Barr virus (EBV) seropositive undergraduates (N = 57) completed a personality inventory, provided blood samples, and were randomly assigned to write or talk about stressful events, or to write about trivial events, during three weekly 20-min sessions, after which they provided a final blood sample. Individuals assigned to the verbal/stressful condition had significantly lower EBV antibody titers (suggesting better cellular immune control over the latent virus) after the intervention than those in the written/stressful group, who had significantly lower values than those in the written/trivial control group. Subjects assigned to the written/stressful condition expressed more negative emotional words than the verbal/stressful and control groups and more positive emotional words than the verbal/stressful group at each time point. The verbal/stressful group expressed more negative emotional words compared with the control group at baseline. Content analysis indicated that the verbal/stressful group achieved the greatest improvements in cognitive change, self-esteem, and adaptive coping strategies.

Writing or talking about stressful events allows individuals to understand upheavals in their lives. Although talking to others is a common pathway for understanding traumas, many stressful events cannot be easily discussed. For example, victims of sexual abuse or war atrocities and perpetrators of crimes or illicit acts are often hesitant to divulge their experiences because of guilt or fear of punishment. In order to avoid this guilt or punishment, they may inhibit their overt behaviors, facial expressions, and language. In addition to inhibiting behavior, individuals may attempt to inhibit conscious thoughts about the concealed information because of its aversive and unresolved nature. Therefore, even in a socially supportive environment, these individuals may be unable to confide in others about extremely upsetting events and must "work" to inhibit their behaviors, thoughts, and emotions (Pennebaker & Susman, 1988).

The expression of feelings about stressful life events is generally believed to play an important role in psychotherapy. Specifically, emotional expression may facilitate cognitive changes, such as reappraisal of an event, which may subsequently lead to adaptive behavior (Greenberg & Safran, 1987; Nichols & Efran, 1985). In recent years, accumulated evidence has suggested that inhibition of the expression of extremely personal and stressful experiences to others over a long period of time may be related to some disease processes (Pennebaker, 1985). To investigate this "affective discharge" hypothesis, Pennebaker and Beall (1986) had subjects write anonymous essays, over a period of several days, about either traumatic or trivial topics. In writing about these events, subjects were instructed to focus their thoughts on the facts, their feelings, or both surrounding a traumatic event. Writing about both their feelings and the facts surrounding the traumatic event resulted in fewer health center visits and self-reported illness in the following 6 months. Subsequent studies (Pennebaker, Colder, & Sharp, 1990; Pennebaker, Kiecolt-Glaser, & Glaser, 1988) have replicated these effects, showing decreases in negative mood and health center visits. In one of these studies, subjects classified as high emotional disclosers on a written task displayed more vigorous lymphocyte proliferative responses to mitogenic challenge—an index of immune functioning (Pennebaker et al., 1988). Moreover, subjects who were instructed to write about their feelings and the facts surrounding a stressful event showed significant increases in this index of lymphocyte proliferative responsivity. These results suggest that a person's ability to manifest an antigen-specific cellular immune response against foreign agents such as viruses or bacteria may be affected by emotional expression.

Talking about a stressful event has also been shown to have biological consequences in a number of studies. For example, Pennebaker, Barger, and Tiebout (1989) found that, among Holocaust survivors, those who evidenced lower skin conductance levels (SCL) and who used the most emotional words when they disclosed particularly traumatic war-related experiences, demonstrated the greatest improvements in health (i.e., physical symptoms and physician visits) in the year after the disclosure interview. This suggested that orally disclosing an extremely traumatic experience—even more than 40 years after its occurrence—had apparent positive health benefits. These findings have been replicated in college students who either wrote or talked into a tape recorder while SCL, blood pressure, and heart rate were continually monitored (Pennebaker, Hughes, & O'Heeron, 1987). Specifically, talking about traumatic events was associated with lower SCL and increased cardiovascular activity, and this relationship was strongest among high disclosers. Most of these studies suggest the importance of studying the

Brian A. Esterling, Michael H. Antoni, and Scott Margulies, Department of Psychology, University of Miami; Mary Ann Fletcher, Department of Medicine, University of Miami; Neil Schneiderman, Department of Psychology, Psychiatry, and Medicine, University of Miami.

Correspondence concerning this article should be addressed to Michael H. Antoni, Department of Psychology, University of Miami, P.O. Box 248185, Coral Gables, Florida 33124.

interaction between expressive style and disclosure performance.

Although these expressive factors have been hypothesized to influence immunologic function and immune-related disease processes, little is known about how they interact in affecting specific immune measures related to infectious disease. Instead of focusing on physical symptoms or physician visits, this study focused on how emotional disclosure affects the reactivation of a latent viral pathogen that is extremely prevalent in the general population, Epstein-Barr virus (EBV). Typically, primary infection with EBV, a human herpesvirus, occurs during adolescence. About 40% of patients with primary EBV infections present with infectious mononucleosis, and the remainder show no clinical signs of infection (Henle & Henle, 1982). Following infection, EBV latently infects bursal-equivalent (B-)lymphocytes resulting in a persistent infection for the life of the individual. If the virus is reactivated in these latently infected cells—possibly because of psychosocial or biological influences—viral antigens are expressed. These antigens are the products of both early virus transcription (synthesized independently of virus deoxyribonucleic acid [DNA] synthesis) and late transcription, which depends on the synthesis of new viral DNA. In either case, the memory cellular and humoral immune responses are triggered, resulting in increased antibody levels to the early antigen (EA) and late antigen, or viral capsid antigen (VCA), complexes.

Cellular immune defenses are critical both in controlling primary herpesvirus infections (including EBV) and in maintaining virus latency (Glaser & Gotlieb-Stematsky, 1982). Alternatively, an increase in EBV–VCA antibody (a humoral immune response) indicates exposure to the virus or reactivation of EBV from latently infected B-lymphocytes. Increases in these antibodies are believed also to reflect a homeostatic response to the "spillover" of excess viral antigens that are inadequately controlled by cellular immune mechanisms. Therefore, antibody titers to various EBV antigens can provide one measure of the efficiency or adequacy of the cellular immune system's control over latent virus (Glaser & Gotleib-Stematsky, 1982). For example, patients undergoing immunosuppressive therapies (e.g., cancer cytotoxic chemotherapy) or patients infected with the human immunodeficiency virus (HIV) have characteristic elevations in herpesvirus antibody titers (Esterling et al., 1992; Tuckwiller & Glaser, 1983). The increased herpesvirus antibody production in immunosuppressive conditions such as these are thought to reflect the humoral immune system's response to an increased load of opportunistic viral antigens such as EBV and cytomegalovirus (Rinaldo, Kingsley, & Lyter, 1986). In some cases, there are also severe clinical symptoms associated with the reactivation of latent virus (Harada, Bechtold, Seeley, & Purtilo, 1982; Ho, 1977; Rickinson, Moss, Pope, & Ahlberg, 1980). Thus, high levels of antibody to EBV–VCA may suggest a nonhomeostatic state in which there is a primary or secondary infection, or alternatively, reactivation of latent virus.

A recently completed meta-analytic study of 24 human psychoimmunological "stressor" studies conducted across the world revealed that among 20 immunological variables tested, antibody titers against EBV were found to be the most consistent and significant correlate of psychosocial stressors (Van Rood, Bogaards, Goulmy, & van Houwelingen, in press). In a series of widely cited studies, elevated EBV antibodies have been associated with a variety of psychological stressors, including academic stress (Glaser et al., 1987), divorce (Kiecolt-Glaser, Kennedy, et al., 1988), and caring for a family member with Alzheimer's disease (Kiecolt-Glaser, Dura, Speicher, Trask, & Glaser, 1991). More recent work provides evidence that the reactivation of latent EBV in medical students at the time of examinations is specific to certain viral proteins (Glaser et al., 1991) and that lymphocyte proliferative responses to several EBV antigens are impaired during examination periods (Glaser et al., in press).

Despite a growing body of evidence supporting the influence of psychosocial stressors on immunological control over EBV, only a handful of studies have investigated the role that individual differences in coping response may play in explaining these relationships. Previously, we found that the emotional expression and interpersonal coping style evidenced by healthy people dealing with stressful traumatic experiences were related to their EBV antibody titers (Esterling, Antoni, Kumar, & Schneiderman, 1990). Specifically, we found that subjects who abstained from disclosing emotional material on a laboratory writing task had elevated EBV antibody titers. Similarly, subjects who displayed elevations repressive interpersonal styles according to personality test scores had higher EBV antibody titers than those displaying more emotionally expressive interpersonal styles.

In the current study, we first sought to replicate the interpersonal coping style data from our previous study suggesting higher EBV reactivation in subjects classified as repressors. The present study was also designed to address the issue of whether experimentally manipulating emotional expression can modulate EBV antibody titers. Finally, we examined whether EBV antibody responses following verbal and written disclosure of stressful events would be different and how seriousness of the disclosed event, cognitive changes, self-esteem improvements, and discussion of adaptive coping strategies relate to changes in EBV antibody titers observed over the course of the disclosure intervention.

Method

Subjects and Exclusion Criteria

Subjects between the ages of 17 and 22 (M = 18.4 years, SE_M = 0.14) were recruited from the first-year undergraduate class at a large southeastern university. Subjects participated to fulfill the requirements of an introductory psychology course. Several exclusionary criteria (e.g., medication use and certain potentially immunomodulatory behaviors) were used to reduce extraneous sources of variance in immunological measurements. Subjects were excluded from the study if they reported current use of any prescription medications, recreational drug use, heavy cigarette smoking (>1 pack/week), excessive alcohol intake (>10 drinks/week), that they were pregnant, or that they were currently undergoing psychotherapy. Other exclusion criteria included psychiatric diagnosis, medical illness within the previous 3 months, or self-reported history of chronic mental or physical illness or skin reactive diseases that might contribute to immunological dysregulation (e.g., eczema). Subjects were required to be free of symptoms that might indicate an active upper respiratory infection or other infection at the time of the study. Seventy-two of 75 (39 male and 33 female) subjects met these criteria. All 72 subjects completed the assessment protocol, although of

the original 72 only 57 were determined to be EBV seropositive. Thus, data from only these 57 subjects were used in study analyses. All 57 subjects completed all sessions of the intervention and blood donation.

Procedure

Before participation in the study, all subjects signed an informed consent form that explained the nature of the study. All those meeting the study criteria completed a battery of measures evaluating their physical status and several potential behavioral immunomodulatory confounds (all measures are described in more detail below).

Subjects were randomly assigned to one of the following conditions: written disclosure of stressful events, verbal disclosure of stressful events, or a trivial writing condition. In each of the first two conditions, subjects were asked to recall and focus on a stressful event that had happened to them and that they had not disclosed to many people. It was emphasized that they should choose a topic that they felt was highly stressful or traumatic or about which they felt very guilty. They were then asked either to write an essay (n = 21) or to speak into a tape recorder (n = 17) about the event as if they were writing (or speaking) to someone they could trust. In the trivial written condition (n = 19), subjects simply wrote about trivial topics that were assigned to them (i.e., the contents of their bedroom closets, their bedrooms and their cars). Subjects completed three weekly sessions in which they received the same instructions. Subjects in each intervention condition had the option to discuss the same stressful event as in the previous week or to choose a different stressful event that they had recently encountered. Approximately 20 min/session was spent in each condition.

Blood for immunological assessment was collected between 1:00 p.m. and 4:00 p.m. 1 week before the initial intervention session and at the same time of day 1 week following the last intervention session. All subjects were debriefed en masse once the entire study was completed.

Psychosocial Assessments

Psychometric measures. At baseline, all subjects completed the Millon Behavioral Health Inventory (MBHI; Millon, Green, & Meagher, 1982), which assesses individual differences in interpersonal coping styles. This inventory is composed of 150 true–false forced choice items, is computer scored, and yields base rate scores pertaining to eight basic coping styles: Introversive, Inhibited, Cooperative, Sociable, Confident, Forceful, Respectful, and Sensitive. These eight basic coping styles are based on the personality theory of Millon. The MBHI subscale scores have a test–retest reliability over 4.5 months ranging from 0.77 to 0.88 with a mean of 0.82 (Millon, 1969; Millon et al., 1982).

As in our previous work (Esterling et al., 1990), subjects were classified into one of three personality groups, depending on their elevations on each of the eight coping scales, consonant with the theory underlying the instrument. One group was designated as repressors, a second group as sensitizers, and a third comprised subjects with neither personality style (NP). The MBHI high-point criteria used for creating these groupings are described in detail in a previous publication (Esterling et al., 1990). Twenty-six subjects met the criteria for sensitizers (elevations on the Inhibited, Forceful, or Sensitive scales), 13 were classified as repressors (elevations on the Introversive, Cooperative, or Respectful scales), and 18 were classified in the NP group (elevations on the Sociable or Confident scales).

To test the validity of our classification method for personality, we compared the base rate elevations for each of the MBHI scales between groups designated as repressors and sensitizers at baseline. Consistent with the test authors, we found that scores on the MBHI Introversive, $F(1, 45) = 28.56$, $p < .001$; Cooperative, $F(1, 45) = 8.87$, $p < .01$; and Respectful $F(1, 45) = 13.82$, $p < .001$, style scales were significantly higher for repressors, compared with sensitizers. Further, scores on MBHI Avoidant, $F(1, 45) = 8.81$, $p < .01$; Forceful, $F(1, 45) = 12.06$, $p < .001$; and Sensitive, $F(1, 45) = 60.87$, $p < .001$, scales were significantly higher for sensitizers, compared with repressors.

Subjects classified as repressors ostensibly have an inner need to deny negative feelings to themselves and others, tend to appear content in the face of problems, and may attempt to please others with self-sacrificing behaviors (Millon et al., 1982). Individuals classified as sensitizers ostensibly come across to others as overbearing, aggressive, rivalrous, and confident; tend to have a low level of frustration tolerance; and are quick to express their negative feelings.

Behavioral measures. All tape recordings of the experimental sessions were transcribed verbatim and verified by having a second judge listen to the tape while reading the transcription. Once all disclosures from the sessions were converted to a written format, each essay was read independently by two judges who were unaware of the other data in the study, as well as of each other's ratings. In scoring the essays, the number of emotional words was computed in line with previous research (Esterling et al., 1990; Pennebaker et al., 1988). Ratings of the percentages of total emotional words were averaged across the judges at each time point and served as the index of emotional expressiveness on this behavioral task. A word was scored as emotional if it satisfied two criteria: The word had to describe a human feeling (e.g., she *loves* her mother), and it could not describe a physical action (e.g., they went to make *love*). The difference between the two types of words is that any word describing a human emotion can have a more intense (or less intense) emotional word substituted in its place. In the first example, "she *loves* her mother" can be converted into "she *likes* her mother" and still make sense. This is not true in the second statement, "they went to make *love*," because the referent of the emotional word *love* is the act of having sex. Thus, the word *loves* in the first example would have been scored, and the word *love* in the second example would not have been scored. Two emotional expression indices were computed with respect to valence (positive and negative emotional word use).

Content ratings were also conducted on all written and verbal disclosures, using a form modified from Murray, Lamnin, and Carver (1989) and Donnelly and Murray (1991). After the essays were read for emotional word content, all essays and verbal transcriptions were read again and were independently and subjectively rated on a 7-point scale (1 = *none*, 4 = *somewhat*, and 7 = *very much*) by the aforementioned two judges according to evidence of positive *cognitive appraisal change* (e.g., were alternative explanations discussed and to what degree, or was there evidence of better understanding of the problem and to what degree?), *self-esteem improvements* (e.g., was there evidence that the subject felt better about self, or less down on self, and to what degree?), and degree to which *adaptive coping strategies* were discussed (e.g., was there evidence that the subject expressed feelings to people, became more assertive, or took more interpersonal risks?). These dimensions were scored as high only if there was explicit evidence in the essay regarding any particular dimension (e.g., if the subject provided behavioral examples). Low scores were given if there was no explicit evidence in the essay or if the dimension was addressed only implicitly (e.g., if the subject wanted to express anger to an ex-boyfriend without actually providing an example of where or how this would be done). In addition, the seriousness of event was rated on each subject's essay or tape transcription. This dimension was scored as high if the subject felt that the event was stressful to them. This item was scored independent of the objective nature of the stressful event. For example, failing an exam was scored high for someone who provided examples of not being able to achieve life goals but low for someone who approached the topic casually. The two judges' scores were averaged for each rating index at each time point.

In addition, five subjects were randomly selected, and all three sessions for each were independently scored by an advanced clinical psychology graduate student trained to check interrater reliability for each

of the above ratings (number of emotional words and content analysis). A Pearson correlation was calculated for each of the three sessions with each rating. A composite reliability coefficient for each rating was computed by averaging the three obtained weekly correlations. Average interrater reliability coefficients for the six ratings ranged from 0.78 to 0.95 (all $ps < .01$).

Self-report control measures. Subjects were asked to describe any infectious disease symptoms, particularly symptoms associated with respiratory tract infections, that they had experienced within the previous 1 and 6 months, respectively. Consistent with previous methodological suggestions in behavioral immunological research (Kiecolt-Glaser & Glaser, 1988), we also collected self-report data on the amount of restful sleep (over the previous week), and the amount of hard physical (sports and work-related) activity, average kilocaloric intake, and recreational substance use over the previous 6 months. In addition, each subject's lean body mass was calculated according to Metropolitan Life Insurance actuarial tables, which take into account body weight and frame size in determining the subject's deviation from his or her ideal weight (i.e., the weight that maximizes life expectancy).

Viral Serology

Blood samples were collected from all subjects and the serum fractions were evaluated for immunoglobulin-G (IgG) antibodies against the VCA and EA components of EBV. We used reagents supplied as kits (Organon Teknika, number 9100-11) and used the indirect immunofluorescence method (IFA). The procedure followed was in accordance with Henle and Henle (1982) and is described in more detail in a previous study (Esterling et al., 1990). Antibody titers to EBV-VCA and EBV-EA were determined by the highest dilution of serum able to demonstrate IFA-positive cells. Antibody titers to EBV-EA were examined to determine whether the high levels of EBV-VCA antibodies in some subjects were due to the result of a primary infection. Data are presented as the mean $\log_2$ transformation of the highest dilution where immunofluorescence was detected. As mentioned previously, only 57 study subjects were determined to be EBV seropositive.

Results

Control Measures

Before interpreting intervention-associated immunological changes, it was important that we investigate self-reported confounding variables that might account for the variance observed in the antibody outcome data. To test whether sleep, physical activity, caloric intake, alcohol use, or lean body mass could account (uniquely or in combination) for immune data variance, we performed a stepwise multiple regression. It was determined that none of these variables contributed a significant amount of the variance in baseline EBV-VCA titer variability, all $Fs < 1$. No significant effects were found when subject gender was included as an additional variable, so data for both sexes were combined. In addition to testing for direct effects of the confounding variables (e.g., sleep or lean body mass) on EBV-VCA titers, it was determined individually using analysis of variance (ANOVA) that none of these variables differed among groups classified by either personality cluster or by treatment condition, all $Fs < 1$ (Table 1). Finally, chi-square analysis determined that the groups did not differ with respect to personality group classification, $\chi^2(4) = 7.9$, which was nonsignificant.

As a further control, antibody titers to EBV-EA were assessed to determine if any of the subjects were recovering from a primary EBV viral infection. Subjects in such a recovery phase might show EBV-VCA levels that are not representative of latent virus reactivation and more indicative of a primary response to initial viral challenge (Sumaya, 1986). It was found that all subjects had antibody titers to EBV-EA in the normal range (i.e., a $\log_2$ dilution factor of 3.32, which corresponds to a dilution factor of 1:10), suggesting that the sample was EBV-VCA seropositive yet not undergoing primary viral infection (Roberts, 1989).

Behavioral Measures

Content analysis. As in previous studies using similar paradigms, the content of the essays suggested that subjects uniformly disclosed highly personal and upsetting experiences, often powerful and poignant. The three stressful experiences discussed most frequently in the stressful conditions were death of a relative or friend, divorce of parents, and break-up of a romantic relationship. The frequency of these themes did not differ between the written/stressful and verbal/stressful disclosure groups. Further, the written/stressful and verbal/stressful groups did not differ in the ratings of seriousness of the event disclosed ($M = 5.1$, $SE_M = 0.23$, and $M = 4.6$, $SE_M = 0.26$, respectively), and both groups reported events rated as more serious than those reported by the control group ($M = 1.0$, $SE_M = 0.0$), $F(2, 54) = 106.48$, $p < .0001$, which did not differ across time, $F < 1$.

In addition to the seriousness of the events, the verbal/stressful group had significantly greater evidence of cognitive change compared with the written/stressful group, and both groups displayed more cognitive change compared with the control group, $F(2, 54) = 15.35$, $p < .0001$. The disclosures of the verbal/stressful group were also rated significantly higher on self-esteem improvements compared with those of the written/stressful and control groups, which did not differ from each other, $F(2, 54) = 8.17$, $p < .001$. Finally, the verbal/stressful group's disclosures were rated as displaying significantly more adaptive coping strategies than those of the written/stressful and control groups, which again did not differ from each other, $F(2, 54) = 14.69$, $p < .0001$. There was no Group × Time interaction on any of these variables, $Fs < 1$.

Emotional expression. As a manipulation check of the experimental conditions, we compared the degree of emotional disclosure displayed by subjects in each of the experimental groups. The intervention groups differed significantly with respect to total words expressed during the sessions, $F(2, 54) = 69.71$, $p < .0001$. Specifically, subjects in the verbal/stressful condition expressed significantly more words ($M = 1250.1$ words, $SE_M = 25.3$) per session compared with the written ($M = 288.9$ words, $SE_M = 3.0$) and control ($M = 217.1$ words, $SE_M = 3.0$) groups, which did not differ from each other. As a result, percentages of emotional words were computed to control for the different numbers of total words expressed.

A 3 × 3 (Intervention Group × Time Point) split-plot factorial ANOVA showed significant group differences for total, $F(2, 54) = 22.11$, $p < .001$; negative, $F(2, 54) = 42.12$, $p < .001$; and positive, $F(2, 54) = 6.57$, $p < .01$, emotional words, although there was no significant change over time in any group, all $Fs <$

Table 1
Immunological Confounds Between the Intervention Groups

Variable	Trivial/control		Written/stressful		Verbal/stressful		Total	
	M	SE_M	*M*	SE_M	*M*	SE_M	*M*	SE_M
Age	18.3	0.22	18.7	0.26	18.3	0.24	18.4	0.14
Calories/day	1,773.7	185.2	1,719.0	106.4	1,782.4	170.7	1,756.1	87.5
Alcohol use/week (drinks)	1.4	0.54	3.8	1.3	4.2	2.5	3.2	0.90
Physical activity/week (hr)	2.4	0.57	2.8	0.59	2.4	0.62	2.6	0.34
Sleep/day (hr)	7.9	0.35	7.4	0.38	7.5	0.34	7.6	0.21
Lean body mass	102.8	3.4	102.6	2.8	105.8	4.0	103.6	1.9

1. Tukey post hoc tests ($p < .05$) determined that the written/stressful group expressed significantly more total and negative emotional words than the verbal/stressful and control groups and significantly more positive emotional words than the verbal/stressful group at each time point (Figure 1). The verbal/stressful group expressed significantly more negative emotional words compared with the control group at Week 1. All other comparisons yielded nonsignificant differences.

EBV-VCA Antibody Titers

Personality correlates. A one-way ANOVA showed significant differences on pre-intervention EBV antibody titers among the three personality classifications, $F(2, 54) = 4.10$, $p < .05$. These tests were followed up by Tukey post hoc tests, which revealed that repressors had significantly higher levels of EBV antibody titers than the NP and sensitizer groups, which did not differ from each other (Figure 2).

Experimental effects. At baseline, subjects randomly assigned to each group were equivalent in EBV-VCA antibody levels, $F < 1$. A 3 × 2 (Intervention Group × Pre- and Postintervention) split-plot factorial repeated measures ANOVA revealed a significant Group × Time interaction for EBV-VCA antibody titers, $F(2, 54) = 11.73$, $p < .001$ (Figure 3). A priori planned-comparison contrasts revealed no significant changes in EBV-VCA titers within the control group, $F(1, 18) = 2.82$, *ns*. This was in contrast to the written/stressful group, $F(1, 20) = 12.31$, $p < .01$, and the verbal/stressful group, $F(1, 16) = 17.69$, $p < .001$, which showed significant decreases in levels of antibody titers to EBV-VCA over the course of the 4-week observation period.

After 3 weeks of participation in the disclosure intervention, significant differences between the groups resulted, $F(2, 54) = 10.20$, $p < .001$. Tukey post hoc tests ($p < .05$) at the postintervention time point confirmed that subjects in the control condition ($M = 7.53$, $SE_M = 0.27$) had significantly higher antibody titers to EBV-VCA than those in the written/stressful

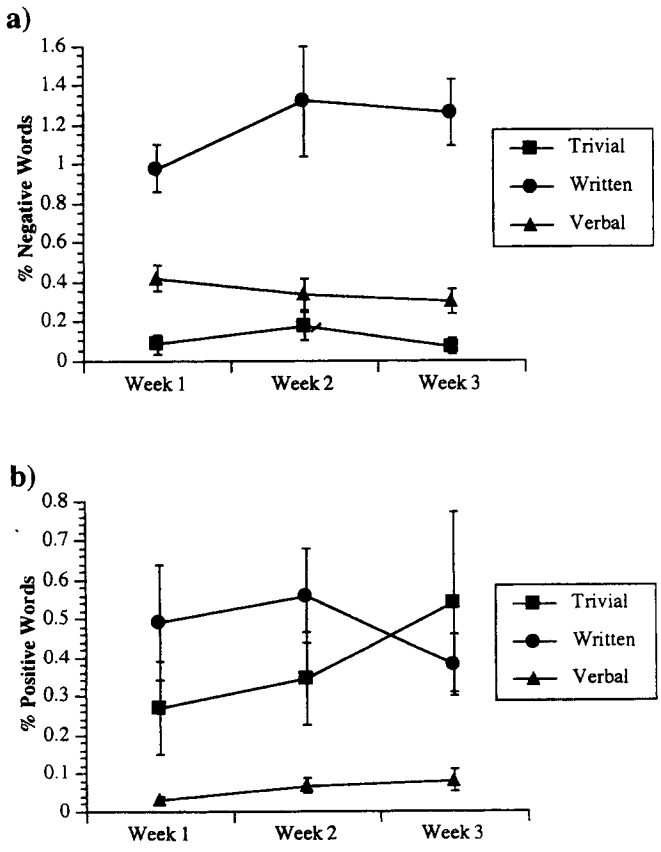

Figure 1. Mean ($\pm SE_M$) percentage of (a) negative and (b) positive words expressed across time.

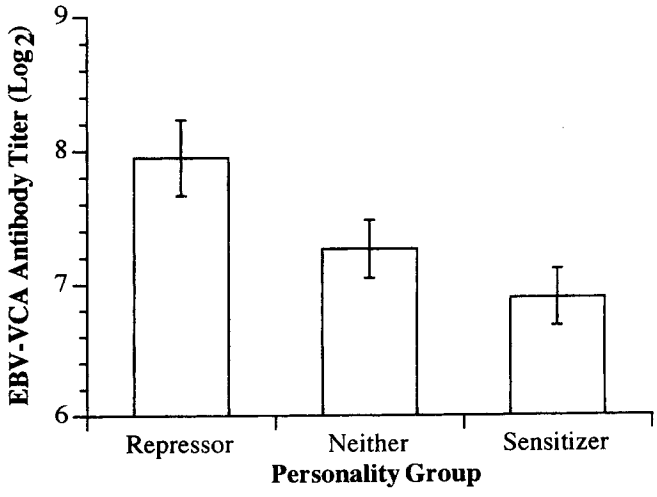

Figure 2. Mean ($\pm SE_M$) Epstein-Barr virus-viral capsid antigen (EBV-VCA) antibody titer values for personality groups.

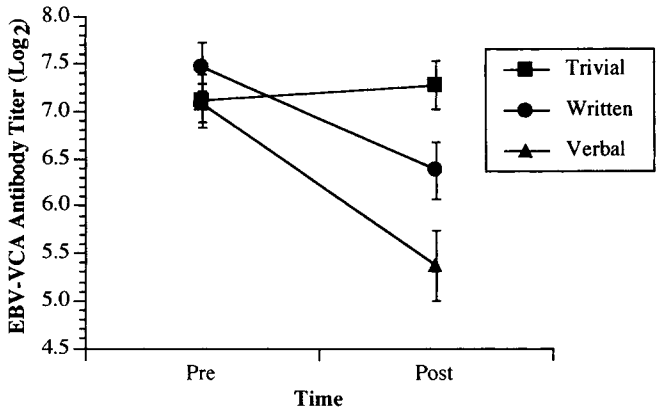

Figure 3. Mean ($\pm SE_M$) Epstein-Barr virus–viral capsid antigen (EBV–VCA) antibody titer values at pre- and postintervention time points.

condition (M = 6.42, SE_M = 0.29), whose titers were significantly higher than observed for the verbal/stressful condition (M = 5.48, SE_M = 0.38).

Finally, experimental condition (dummy coded using three vectors), personality style (dummy coded using three vectors), degree of emotional expression at Week 3 (negative and positive emotional words), and emotional content (cognitive reappraisal, enhanced self-esteem, adaptive coping, and seriousness of event) were entered in a hierarchical multiple regression model to predict changes in EBV antibody titers. The overall model with all predictors was significant, $F(10, 46) = 5.05$, $p < .0001$, and accounted for 53.2% of the variance in EBV antibody titer change (Table 2). As expected, experimental group assignment was the most significant predictor of EBV antibody change. In addition, personality style significantly predicted EBV antibody titer change, with subjects classified as sensitizers having greater decreases in antibody titers over the course of the intervention. Increases in the number of expressed negative emotional words over the intervention significantly predicted greater decreases in EBV antibody titer level, although positive emotional word changes did not significantly predict EBV antibody titer change. Finally, greater cognitive change, enhanced self-esteem, and seriousness of event predicted greater decreases in EBV antibody titers, whereas adaptive coping strategies were not related to EBV antibody change.

Discussion

Previously, we found that subjects who abstained from disclosing emotional material on a laboratory task, or who were psychometrically classified as having a repressive interpersonal style, had higher EBV antibody titers, suggesting poorer immunological control of latent EBV (Esterling et al., 1990). We reasoned in the present study that by manipulating emotional expression through different mechanisms of arousal (i.e., speaking vs. writing), antibody titers to EBV might be modulated downward. Specifically, we found that participation in either a written or a verbal emotional disclosure intervention significantly decreased EBV antibody titers over a 4-week observation period. Although equivalent at baseline, individuals assigned to the verbal/stressful group showed significantly lower antibody titer values after the intervention than those in the written/stressful group, who had significantly lower values than those in the control group. Further, it was found that subjects assigned to the written/stressful group expressed significantly more total and negative emotional words than the verbal/stressful and control groups and significantly more positive emotional words than the verbal/stressful group at each time point. The verbal/stressful group expressed significantly more negative emotional words compared with the control group at Week 1. Further, the verbal/stressful group was rated higher in cognitive change, self-esteem improvements, and adaptive coping strategies as compared with the other groups. Finally, results confirmed our previous research, which suggested that individuals classified as having a repressive personality style have significantly higher EBV antibody titers compared with sensitizers (Esterling et al., 1990).

Compared with previous EBV seroepidemiological studies, which have shown characteristic EBV–VCA IgG geometric means of the raw titers ranging from 1:10 to 1:80 ($\log_2$ = 3.32

Table 2
Hierarchical Multiple Regression of Change in Epstein-Barr Virus Antibody Titer

Variable	β	R	R^2	R^2 Change	F	dfs	p
Step 1							
Experimental group	−0.36***	0.53	0.28	0.28	10.09	2, 54	.0001
Step 2							
Interpersonal coping style	−0.24*	0.58	0.33	0.05	6.31	4, 52	.001
Step 3: Degree of emotional expression							
Negative words	−0.33*	0.63	0.39	0.06	5.29	6, 50	.001
Positive words	0.14						
Step 4: Emotional content							
Cognitive change	−0.33*	0.73	0.53	0.14	5.05	10, 46	.0001
Self-esteem	−0.38*						
Coping	−0.21						
Seriousness	−0.48**						

* $p < .05$. ** $p < .01$. *** $p < .001$.

and 6.32, respectively) in normal adult populations (Henle & Henle, 1982), our sample appeared to have slightly elevated means at baseline but fall far short of the serologic pattern documented in patients who are immunologically compromised because of immunosuppressive diseases, cancers such as Burkitt's lymphoma or nasopharyngeal carcinoma, or therapies that directly suppress the immune system (Tuckwiller & Glaser, 1983). Elevated antibody titers in healthy individuals not suffering from these conditions are thought to occur in response to enhanced expression of latent virus facilitated by change, or dysfunction of, different thymus-derived (T)-lymphocyte subpopulations (Henle & Henle, 1982; Klein et al., 1981; Rickinson et al., 1981). Unfortunately, because all of this work has involved in vitro assays, it is difficult to determine with certainty just how the viral genome is latently controlled and how it subsequently becomes reactivated. Whatever the mechanism, the elevated EBV-VCA antibody titers noted in our subjects suggest that there was some compromise in their viral antibody response, which approached normal values as a function of participating in the written/stressful or verbal/stressful intervention conditions.

It is not surprising that healthy first-year undergraduates demonstrated elevated antibody titers to EBV at this time of year. There is much accumulated evidence that EBV is sensitive to psychological phenomena (e.g., academic stress, marital disruption, caregiving, personality), as demonstrated in previous studies in our laboratory and others (Esterling, Antoni, Kumar, & Schneiderman, 1993; Esterling et al., 1990, 1992; Glaser, Kiecolt-Glaser, Speicher, & Holliday, 1985; Glaser et al., in press; 1991). Although we did not measure stress explicitly, the subjects may have been experiencing stress because of separation from family and friends, moving to a new environment (many of the students who attend the university are not from the neighboring area), increased expectations and pressure with respect to academic achievement, and so on. Collectively, these events could have been stressful enough to reactivate EBV to a degree where antibody titers would be elevated. In fact, many of the topics chosen focused on these types of events (e.g., failing an exam, moving away from a boyfriend or girlfriend, loneliness due to difficulty making friends, etc.).

A final limitation of this study involves the interpretations of the antibody data. However, because only one type of antibody was evaluated, it is possible that the relationships found were due to an overall change in antibody levels (i.e., polyclonal B cell activation secreting IgG) and were not specific to EBV-VCA. Although the possibility that B cells were polyclonally stimulated cannot be ruled out, in two previous studies we found no correlation between levels of antibody to EBV and Forrsman antibody, a marker of polyclonal B cell activation (Esterling et al., 1992, 1993).

Talking Versus Writing

In an effort to understand the processes that differ between speaking and writing about stressful events, attention has focused on cognitive appraisal. Murray et al. (1989) compared brief psychotherapy with written expression in a population comparable to the one used in this study. They found that written expression of emotion was quite effective in temporarily arousing negative affect, but not in changing feelings about traumatic events, although some self-generated cognitive changes did occur. In contrast, verbal expression aroused less negative affect, but resulted in much more cognitive reappraisal and a dramatic shift to positive affect, as well as a basic change in attitude about stressful events. The latter findings appear to have been replicated in the present study, where we found that the verbal/stressful group showed higher ratings for cognitive change, self-esteem improvements, and adaptive coping strategies compared with the written/stressful and control groups. Further, recent work from our lab suggests that changes in the way stressful events are cognitively processed during a disclosure manipulation are also predictive of decreases in EBV-VCA antibody titers over a similar 1-month period (Lutgendorf, Antoni, Kumar, & Schneiderman, in press). Further, we found that greater expression of negative affect words, as well as greater cognitive reappraisal, self-esteem, and seriousness of event chosen predicted decreases in EBV-VCA antibody titers over the 4-week observation period. Therefore, ventilation of negative feelings may be the important event in written disclosure, whereas cognitive reappraisal, enhanced self-esteem, and generation of adaptive coping strategies may be achieved through verbal expression.

Our data support other research that has investigated the effects of written expression on health care utilization and physical symptoms. For example, Greenberg and Stone (in press) found that subjects who disclosed more severe traumas reported fewer physical symptoms in the months following the study, compared with both subjects reporting low-severity traumas and those in a control group, independent of previous disclosure history. In addition, Pennebaker and Beall (1986) showed that writing emotionally about stressful events decreased health center visits in the 6 months following the study. Our data suggest one immunological pathway by which emotional expression may contribute to the health findings noted in these studies.

Emotional Experience Versus Emotional Expression

In examining the interactions between emotional arousal and immune function, the literature suggests that immune function and health are modulated differently depending on whether individuals are subjected to emotionally arousing situations or have an appropriate outlet for the expression of those emotions. Several studies have found that writing about stressful events leads to increased experience and expression of emotion, particularly anxiety (Pennebaker & Beall, 1986; Pennebaker et al., 1987, 1988). This increased experience of anxiety has further been correlated with decreased health center visits, improvement in cardiovascular health indices, and increases in mitogen responsiveness, a marker of cellular proliferation in response to a novel antigen (Pennebaker & Beall, 1986; Pennebaker & O'Heeron, 1984; Pennebaker et al., 1987, 1988, 1989).

However, there also exist studies in which heightened trait-anxiety is related to lower levels of monocytes (an immune cell) and increases in endogenous hormones such as cortisol, prolactin, testosterone, epinephrine, norepinephrine, and thyrotropin-releasing hormone, all of which play a role in reduced cellular control over latent EBV (Demyttenaere, Nijs, Evers-Kie-

booms, & Koninckx, 1989; Herbert, Moore, de la Riva, & Watts, 1986; Kasvikis et al., 1988; Laakmann, Hinz, & Neuhauser, 1986; Salmon et al., 1989). Moreover, anxiety-provoking events (e.g., medical school examination, marital disruption, or antibody testing for human immunodeficiency virus [HIV-1]) have also been associated with decrements in markers of cellular immune functioning, including reactivation of latent EBV (Antoni et al., 1990; Glaser et al., 1985, 1991, 1987; Kiecolt-Glaser et al., 1987, 1988). One team found that examination stress and marital disruption were associated with increased EBV antibody titers and transformation of B-lymphocytes, as well as decreased antigen-specific memory T-cell response, as measured by the ability of peripheral blood T-lymphocytes to respond to EBV-specific polypeptides (Glaser et al., 1985, 1987, 1991). In other work, anticipatory anxiety levels and intrusive thoughts were related to higher plasma cortisol levels and lower lymphocyte proliferative responses to mitogenic stimulation (Antoni et al., 1990, 1991; Ironson et al., 1990; McKinnon, Weisse, Reynolds, Bowles, & Baum, 1989).

Therefore, these and previous data suggest that the experience of an emotion (e.g., anxiety) does not by itself have a beneficial effect on the immune response against latent viruses such as EBV; in fact, the opposite seems to be the case. On the one hand, experiencing heightened emotion without the opportunity to release or express it in a constructive manner may be detrimental to various components of the cellular immune system. On the other hand, if with heightened emotion there is an easily accessible and comfortable route of discharge (e.g., writing or talking about the stressful event), disclosure may have beneficial effects in modulating the orchestration of effective communication within the viral surveillance system with respect to EBV. In fact, the distinction between emotional experience and expression may help to explain the relatively low or nonexistent correlations between most measures of negative affect, neuroticism, and anxiety with traditional health outcome measures. Specifically, chronically highly anxious individuals may be at highest risk for health decrements, but may also benefit most from expressive therapies, compared with less anxious individuals or ones who are not in touch with their emotional world (i.e., repressors).

Personality Correlates

The present study confirms our previous findings (Esterling et al., 1990) suggesting that subjects who ostensibly have an inner need to deny negative feelings to themselves and others and who appear content in the face of problems (i.e., repressors) have greater reactivation of latent EBV compared with those subjects who have low levels of frustration tolerance and who are quick to express their negative feelings (i.e., sensitizers). Further, the fact that we did not find an interaction between personality classification and group assignment suggests that both writing and speaking about stressful events have beneficial physiological effects, independent of personality factors related to emotional proclivities.

It has been hypothesized that "physiological work" is required to inhibit ongoing behavior, thoughts, or feelings (Fowles, 1980; Gray, 1975). Specifically, immediately following a stressful event, behavioral (and perhaps emotional) inhibition is accompanied by increased sympathetic nervous activity (Fowles, 1980) and increased sympathetic firing in the septal and hippocampal regions of the brain (Gray, 1975). It is plausible that, if this behavior pattern continues, the work of inhibition may serve as a low-level chronic stressor that has the potential of long-term dysregulation of viral pathogens. If this hypothesis is correct, it may explain the immunological differences noted in those individuals who freely express negative emotion in response to stressful events versus those individuals who express less affect following similar events.

Previously, we found that subjects high in defensive coping or anxiety may have an increased risk for reactivation of latent EBV among EBV seropositive, but healthy, individuals (Esterling et al., 1993). This, combined with previous data, suggests that a large number of psychological variables at least correlate with EBV antibody titers. Previously, we found that subjects classified as low in expressed emotion, or who had high scores on the repressor scales of the MBHI (i.e., our repressors), were correctly classified as repressors 35% of the time (i.e., high defensive–low anxious) using the construct originally proposed by Weinberger, Schwartz, and Davidson (1979). In contrast, 47% of the previously classified repressors were classified as high defensive–high anxious according to Weinberger et al. (1979). In addition, 28% of the previously classified sensitizers were classified as repressors using Weinberger et al.'s criteria. Finally, the fact that the anxiety-by-defensiveness interaction accounted for such a small proportion of the variance in EBV titers raised questions about whether these two measures should be combined to form artificial constructs of repression in investigations of health correlates (e.g., Weinberger et al., 1979). The fact that we did not find that the interaction between anxiety and defensiveness contributed to EBV–VCA variance in our prior work, and a very different pattern of results was obtained when such a classification system was used, draws light into the potential problems with the current standard classification system for repressive coping. Therefore, how one defines repression remains a very important concept when interpreting, understanding, and comparing data across laboratories.

Methodological Issues

In the present design, subjects assigned to the written/stressful group expressed significantly more negative and positive emotional words compared with the verbal/stressful and control groups. The written group apparently expressed more emotion than the verbal group. However, some aspects of our method limit such conclusions. For example, there appears to have been a difference in investment in the task. Specifically, subjects assigned to the trivial task wrote fairly short essays compared with the other groups. Further, the verbal/stressful group spoke on average five times as many words as the written/stressful group, principally because it is easier to talk than to write and easier to go into much greater detail when speaking about everything regarding a stressful event. Therefore, the percentages of emotional words expressed by the verbal group may be lower than expected because their emotional expression was "diluted" by other thoughts and expressions about the stressful event (i.e., greater detail was expressed). Because the verbal group as a whole expressed many more total words compared

with the other groups, and because of their potentially greater involvement in the task, their overall percentage scores may have been lower. Although involvement was not coded, it may provide some insight into the immunological differences noted between the verbal and written/stressful conditions. Other recent work in our lab suggests that greater involvement in a verbal disclosure task predicts greater decrements in EBV-VCA antibody titers over a similar 4-week period (Lutgendorf et al., in press).

Second, it is possible that time since occurrence of the stressful event disclosed may have had an impact on our results. For example, recent stressful events that the subjects chose to discuss, as compared with events in the more distant past, may be related differently to EBV reactivation, resulting in an interaction of therapy intervention and time of stressful event experience. This is especially salient in light of other work in our laboratory that has shown that subjects who disclosed older and more troublesome events had higher levels of antibody to EBV, suggesting greater viral reactivation (Lutgendorf et al., in press). Unfortunately, these data were not collected, and this question cannot be addressed with this sample.

Clinical Implications

In this study, we show that both writing and speaking about stressful events have a beneficial effect on the immune response to EBV, and that the act of writing about nonstressful events is inconsequential. This suggests that the act of writing per se is not enough to cause immunological changes, at least as measured in the present study. Verbalizing the event may produce a deeper awareness of the emotional issues surrounding the stressor, because people get to rehearse a behavior that more realistically captures the ways in which they are accustomed to ventilating their feelings and frustrations. Of course, the possibility remains that the mere act of talking—even about trivial matters—might bring about immunological changes similar to those seen in this study. Future work is needed to investigate this hypothesis.

Because we and others have shown that verbal or written expression of stressful events creates an initial upsurge of negative mood (Donnelly & Murray, 1991), questions regarding the practical uses of such methods have been raised. For example, although verbal or written expression seems to have a therapeutic effect, the negative affect experienced in the initial session may lead individuals to drop out of therapy before a therapeutic effect is observed. In our study we minimized our attrition rate by providing an incentive of undergraduate course credit. In the clinical application of such techniques, motivating individuals to participate and persist would be very important. One method, as suggested by several authors (L'Abate, 1991; Phillips & Wiener, 1966), might include combining written homework with expressive psychotherapy. Indeed, verbal homework might work even better than written homework when physical, as well as emotional, health is a goal of therapy.

Conclusion

The findings of this study may be important for healthy individuals, as well as for high-risk and HIV-1 seropositive populations. EBV antibody titers may serve as a marker for viral surveillance, in general, of other pathogens, leading to the idea that psychosocial stress may place a burden on the host's immune system, which may have consequences for other diseases. Because psychological interventions have been shown here and elsewhere to be effective in modulating immune system control over EBV and other herpesviruses (Esterling et al., 1992), they may provide an adjunctive treatment that could be offered over and above traditional medical treatments. Further, because of the close interacting relationship between EBV and life-threatening viruses such as HIV, EBV reactivation may have serious consequences, such as increased HIV viral load or increased viral transport within the host (Roncella et al., 1990; Rosenberg & Fauci, 1991). One remaining question that needs further investigation is whether psychosocial variables can influence latent herpes virus infections that might have clinical significance in people diagnosed with AIDS or some cancers. Because herpesvirus infections are important in HIV-related clinical conditions (e.g., cytomegalovirus retinitis, herpes zoster infections, EBV, and herpes simplex virus infections), the present findings may provide an important impetus for considering the role of psychosocial factors when developing treatment strategies for these conditions.

References

Antoni, M. H., August, S., LaPerriere, A., Baggett, L., Klimas, N., Ironson, G., Schneiderman, N., & Fletcher, M. A. (1990). Psychological and neuroendocrine measures related to functional immune changes in anticipation of HIV-1 serostatus notification. *Psychosomatic Medicine, 52,* 496–510.

Antoni, M. H., Schneiderman, N., Klimas, N., LaPerriere, A., Ironson, G., & Fletcher, M. A. (1991). Disparities in psychological, neuroendocrine, and immunologic patterns in asymptomatic HIV-1 seropositive and seronegative gay men. *Biological Psychiatry, 29,* 1023–1041.

Demyttenaere, K., Nijs, P., Evers-Kiebooms, G., & Koninckx, P. R. (1989). The effect of a specific emotional stressor on prolactin, cortisol, and testosterone concentrations in women varies with their trait anxiety. *Fertility and Sterility, 52,* 942–948.

Donnelly, D. A., & Murray, E. J. (1991). Emotional changes in written essays and therapy interviews. *Journal of Social and Clinical Psychology, 10,* 334–350.

Esterling, B. A., Antoni, M. H., Kumar, M., & Schneiderman, N. (1990). Emotional repression, stress disclosure responses and Epstein-Barr viral capsid antigen titers. *Psychosomatic Medicine, 52,* 397–410.

Esterling, B. A., Antoni, M. H., Kumar, M., & Schneiderman, N. (1993). Defensiveness, trait anxiety and Epstein-Barr viral capsid antigen titers in healthy college students. *Health Psychology, 12,* 133–140.

Esterling, B. A., Antoni, M. H., Schneiderman, N., Carver, C. S., LaPerriere, A., Ironson, G., Klimas, N. G., & Fletcher, M. A. (1992). Psychosocial modulation of antibody to Epstein-Barr viral capsid antigen and human herpesvirus type-6 in HIV-1-infected and at-risk gay men. *Psychosomatic Medicine, 54,* 354–371.

Fowles, D. C. (1980). The three arousal model: Implications of Gray's two-factor theory for heart rate, electrodermal activity and psychopathy. *Psychophysiology, 17,* 87–104.

Glaser, R., & Gotlieb-Stematsky, T. (1982). *Human herpesvirus infections: Clinical aspects.* New York: Marcel Dekker.

Glaser, R., Kiecolt-Glaser, J. K., Speicher, C. E., & Holliday, J. E. (1985). Stress, loneliness, and changes in herpesvirus latency. *Journal of Behavioral Medicine, 8,* 249–260.

Glaser, R., Pearson, G. R., Bonneau, R. H., Esterling, B. A., Atkinson, C. A., & Kiecolt-Glaser, J. K. (in press). Stress and the memory T-cell response to the Epstein-Barr virus. *Health Psychology.*

Glaser, R., Pearson, G. R., Jones, J. F., Hillhouse, J., Kennedy, S., Mao, H., & Kiecolt-Glaser, J. K. (1991). Stress-related activation of Epstein-Barr virus. *Brain, Behavior, and Immunity, 5,* 219–232.

Glaser, R., Rice, J., Sheridan, J., Fertal, R., Stout, J., Speicher, C., Pinshy, D., Kotur, M., Post, A., Beck, M., & Kiecolt-Glaser, J. K. (1987). Stress-related immune suppression: Health implications. *Brain, Behavior, and Immunity, 1,* 7–20.

Gray, J. A. (1975). *Elements of a two-process theory of learning.* New York: Academic Press.

Greenberg, L. S., & Safran, J. D. (1987). *Emotion in psychotherapy.* New York: Guilford Press.

Greenberg, M. A., & Stone, A. A. (in press). Emotional disclosure about traumas and health: Effects of previous disclosure and trauma severity. *Journal of Personality and Social Psychology.*

Harada, S., Bechtold, T., Seeley, J. K., & Purtilo, D. T. (1982). Cell-mediated immunity to Epstein-Barr virus (EBV) and natural killer (NK)-cell activity in the X-linked lymphoproliferative syndrome. *International Journal of Cancer, 30,* 734–744.

Henle, W., & Henle, G. (1982). Epstein-Barr virus and infectious mononucleosis. In R. Glaser and T. Gottleib-Stematsky (Eds.), *Human herpesvirus infections: Clinical aspects.* New York: Marcel Dekker.

Herbert, J., Moore, G. F., de la Riva, C., & Watts, F. N. (1986). Endocrine responses and examination anxiety. *Biological Psychology, 22,* 215–226.

Ho, M. (1977). Virus infections after transplantation in man. Brief review. *Archives of Virology, 55,* 1–24.

Ironson, G., LaPerriere, A., Antoni, M., O'Hearn, P., Schneiderman, N., Klimas, N., & Fletcher, M. A. (1990). Changes in immune and psychological measures as a function of anticipation and reaction to news of HIV-1 antibody status. *Psychosomatic Medicine, 52,* 247–270.

Kasvikis, Y. G., Basoglu, M., Monteiro, W., Marks, I. M., Lelliott, P., & Noshirvani, H. (1988). Urinary cortisol during exposure in obsessive-compulsive ritualizers. *Psychiatry Research, 23,* 131–135.

Kiecolt-Glaser, J. K., Dura, J. R., Speicher, C. E., Trask, J., & Glaser, R. (1991). Spousal caregivers of dementia victims: Longitudinal changes in immunity and health. *Psychosomatic Medicine, 53,* 345–362.

Kiecolt-Glaser, J. K., Fisher, L. D., Ogrocki, P., Stout, J. C., Speicher, C. E., & Glaser, R. (1987). Marital quality, marital disruption, and immune function. *Psychosomatic Medicine, 49,* 13–34.

Kiecolt-Glaser, J. K., & Glaser, R. (1988). Methodological issues in behavioral immunology research with humans. *Brain, Behavior, and Immunity, 2,* 67–78.

Kiecolt-Glaser, J. K., Kennedy, S., Malkoff, S., Fisher, L., Speicher, C. E., & Glaser, R. (1988). Marital discord and immunity in males. *Psychosomatic Medicine, 50,* 213–229.

Klein, E., Ernberg, I., Masucci, M. G., Szigeti, R., Wu, Y. T., Masucci, G., & Svedmyr, R. (1981). T-cell response to B-cells and Epstein-Barr virus antigens in infectious mononucleosis. *Cancer Research, 41,* 4210–4215.

Laakmann, G., Hinz, A., & Neuhauser, H. (1986). New neuroendocrine aspects of depression and anxiety. *Clinical Neuropharmacology, 9,* 245–247.

L'Abate, L. (1991). The use of writing in psychotherapy. *American Journal of Psychotherapy, 45,* 87–98.

Lutgendorf, S. K., Antoni, M. H., Kumar, M., & Schneiderman, N. (in press). Changes in cognitive coping strategies predict EBV-antibody titer change following a stressor disclosure induction. *Journal of Psychosomatic Research.*

McKinnon, W., Weisse, C. S., Reynolds, C. P., Bowles, C. A., & Baum, A. (1989). Chronic stress, leukocyte subpopulations, and humoral response to latent viruses. *Health Psychology, 8,* 389–402.

Millon, T. (1969). *Modern psychopathology: A biosocial approach to maladaptive learning and functioning.* Philadelphia: W. B. Saunders.

Millon, T., Green, C. J., & Meagher, R. B. (1982). *Millon Behavioral Health Inventory.* Minneapolis, MN: Interpretive Scoring Systems.

Murray, E. J., Lamnin, A. D., & Carver, C. S. (1989). Emotional expression in written essays and psychotherapy. *Journal of Social and Clinical Psychology, 7,* 414–429.

Nichols, M. P., & Efran, J. S. (1985). Catharsis is psychotherapy: A new perspective. *Psychotherapy, 22,* 46–58.

Pennebaker, J. W. (1985). Traumatic experience and psychosomatic disease: Exploring the roles of behavior inhibition, obsession, and confiding. *Canadian Psychology, 26,* 82–95.

Pennebaker, J. W., Barger, S. D., & Tiebout, J. (1989). Disclosure of traumas and health among Holocaust survivors. *Psychosomatic Medicine, 51,* 577–589.

Pennebaker, J. W., & Beall, S. (1986). Confronting a traumatic event: Toward an understanding of inhibition and disease. *Journal of Abnormal Psychology, 95,* 274–281.

Pennebaker, J. W., Colder, M., & Sharp, L. K. (1990). Accelerating the coping process. *Journal of Personality and Social Psychology, 58,* 528–537.

Pennebaker, J. W., Hughes, C. F., & O'Heeron, R. C. (1987). The psychophysiology of confession: Linking inhibitory and psychosomatic processes. *Journal of Personality and Social Psychology, 52,* 781–793.

Pennebaker, J. W., Kiecolt-Glaser, J., & Glaser, R. (1988). Disclosure of traumas and immune function: Health implications for psychotherapy. *Journal of Consulting and Clinical Psychology, 56,* 239–245.

Pennebaker, J. W., & O'Heeron, R. C. (1984). Confiding in others and illness rate among spouses of suicide and accidental-death victims. *Journal of Abnormal Psychology, 93,* 473–476.

Pennebaker, J. W., & Susman, J. R. (1988). Disclosure of traumas and psychosomatic processes. *Social Science and Medicine, 26,* 327–332.

Phillips, E. L., & Wiener, D. N. (1966). *Short term psychotherapy and structured behavior change.* New York: McGraw-Hill.

Rickinson, A. B., Moss, D. J., Pope, J. H., & Ahlberg, N. (1980). Long-term T-cell-mediated immunity to Epstein-Barr virus in man. IV. Development of T-cell memory in convalescent infectious mononucleosis patients. *International Journal of Cancer, 25,* 59–65.

Rickinson, A. B., Moss, D. J., Wallace, L. E., Rowe, M., Misko, I. S., Epstein, M. A., & Pope, J. H. (1981). Long-term T-cell-mediated immunity to Epstein-Barr virus. *Cancer Research, 41,* 4216–4221.

Rinaldo, C. R., Kingsley, L. W., & Lyter, D. W. (1986). Association of HTLV-III with Epstein-Barr virus infection and abnormalities of T lymphocytes in homosexual men. *Journal of Infectious Diseases, 154,* 556–561.

Roberts, G. H. (1989). The many faces of Epstein-Barr virus. *Diagnostic and Clinical Testing, 27,* 16–21.

Roncella, S., Caretto, P., Forni, G., Cutrona, G., Verde, A., Ramarli, D., de-Celle, P. F., Foa, R., Sessarego, M., & Pistoia, V. (1990). Assessment of the potential malignancy of Epstein-Barr virus infected B cells in AIDS-related disorders. *Cancer Detection and Prevention, 14,* 325–329.

Rosenberg, Z. F., & Fauci, A. S. (1991). Activation of latent HIV infection. *Journal of the NIH Research, 2,* 41–45.

Salmon, P., Pearce, S., Smith, C. C., Manyande, A., Heys, A., Peters, N., & Rashid, J. (1989). Anxiety, type A personality and endocrine responses to surgery. *British Journal of Clinical Psychology, 28,* 279–280.

Sumaya, C. V. (1986). Epstein-Barr virus serologic testing: Diagnostic indications and interpretations. *Pediatric Infectious Diseases, 5,* 337–342.

Tuckwiller, L. S., & Glaser, R. (1983). Epstein-Barr virus and nasopha-

ryngeal carcinoma. In Reznick-Schuller (Ed.), *Comparative respiratory tract carcinogenesis* (pp. 171–185). Cleveland, OH: CRC Press.

Van Rood, Y. R., Bogaards, M., Goulmy, E., & van Houwelingen, H. C. (in press). The effects of stress and relaxation on the in vitro immune response in man: A meta-analytic study. *Journal of Behavioral Medicine.*

Weinberger, D. A., Schwartz, G. E., & Davidson, R. J. (1979). Low-anxious, high-anxious, and repressive coping styles: Psychometric patterns and behavioral and physiological responses to stress. *Journal of Abnormal Psychology, 88,* 369–380.

Received December 28, 1992
Revision received April 13, 1993
Accepted April 20, 1993 ■