Psychology and Aging
2014, Vol. 29, No. 3, 636–641

0882-7974/14/$12.00 http://dx.doi.org/10.1037/a0037452

BRIEF REPORT

The Aging Positivity Effect and Immune Function: Positivity in Recall Predicts Higher CD4 Counts and Lower CD4 Activation

Elise K. Kalokerinos, William von Hippel, and Julie D. Henry
The University of Queensland

Robert Trivers
Biosocial Research Foundation, New Brunswick, New Jersey

Older adults favor emotionally positive material over emotionally negative material in information processing. Given the potentially harmful consequences of avoiding negative information, this aging positivity effect may provide benefits that offset its costs. To test this possibility, we assessed positivity in recall and blood indicators of immune function among older adults. Greater positivity in recall predicted higher CD4 counts and lower CD4 activation 1 and 2 years later. Positivity in recall also predicted subsequent positivity in recall and recognition memory 1 year later. These data suggest that the positivity effect in information processing may play a role in healthy aging.

Keywords: positivity effect, immune functioning, emotion, aging, memory

Supplemental materials: http://dx.doi.org/10.1037/a0037452.supp

When compared with younger adults, older adults tend to favor emotionally positive material over negative material, a phenomenon that has been termed the *aging positivity effect* (Carstensen & Mikels, 2005). Because there are important costs to focusing on the positive at the expense of negative information in domains like decision making (Löckenhoff & Carstensen, 2007) and identifying untrustworthy people (Castle et al., 2012), these costs raise the question of why the positivity effect exists.

The prevailing explanation within psychology for the positivity effect is socio-emotional selectivity theory, which posits that older adults tend to perceive time as limited, and in an effort to enjoy the time they have left, pursue positive emotional goals (Carstensen, Isaacowitz, & Charles, 1999). Thus, time perspective rather than chronological age is argued to be the mechanism behind age-related positivity. In contrast to this focus on proximal causes, in the current article we propose a functional explanation for the aging positivity effect. Old age brings with it a weakening immune system and hence greater threats from cancers and life-ending diseases. One way that the aging positivity effect may offset its apparent costs is by enhancing longevity via improved immune functioning (Trivers, 2010; Trivers, 2011; von Hippel & Trivers, 2011). A large body of research has demonstrated that positive affect is associated with reduced morbidity and mortality, decreased illness symptoms, and better immune functioning, and that these associations are larger for older than younger adults (Pressman & Cohen, 2005). We suggest that positivity in information processing may have similar effects.

Aging and Immunity

In the current research we examined three sets of measures that are closely related to immune aging, or immunosenescence. First, we measured t-cell counts, including $CD4^+$ and $CD8^+$ t-cells. $CD8^+$ t-cells directly target and kill infected or cancerous cells, and $CD4^+$ t-cells act primarily as helper cells by triggering b-cells to produce antibodies. Much of the change in the immune system with age is due to alterations in the composition of t-cells (Ginaldi et al., 1999). Several studies have demonstrated a drop in the numbers of $CD4^+$ t-cells (for a review, see Pawelec et al., 2002) and $CD8^+$ t-cells with age (Effros, Cai, & Linton, 2003). Second, we measured $CD4^+$ t-cell activation using the HLA-DR activation marker, an MHC molecule expressed on activated t-cells that is increased in response to pathogen threat and with chronic viral infection (Costantino, Spooner, Ploegh, & Hafler, 2012). Older participants with increased $CD4^+$ cell HLA-DR expression demonstrate decreased responding to pathogen threat (Fahey et al., 2000), and several studies have demonstrated increased $CD4^+$ cell HLA-DR expression and activation with aging (e.g., Cossarizza et al., 1997; Rea, McNerlan, & Alexander, 1999). Finally, we measured latent Epstein-Barr virus antibodies, which are reactivated

Elise K. Kalokerinos, William von Hippel, and Julie D. Henry, School of Psychology, The University of Queensland; Robert Trivers, Biosocial Research Foundation, New Brunswick, New Jersey.

This research was supported by grants from the Australian Research Council and the Biosocial Research Foundation. We thank Mary-Anne Kalokerinos, Peter Hobson, Daman Langguth, Donald Lear, and Courtney Butler for their assistance in the collection and interpretation of the immunological data, and Tom Braciale for helpful discussions about the immunological data and for comments on the article.

Correspondence concerning this article should be addressed to Elise Kalokerinos, School of Psychology, The University of Queensland, St Lucia QLD, 4072, Australia. E-mail: e.kalokerinos@uq.edu.au

when the immune system is compromised (McKinnon, Weisse, Reynolds, Bowles, & Baum, 1989).

We tested positivity and immune functioning at three time points, each 1 year apart. At the initial testing, we examined the positivity effect in recall among younger and older adults, and then tested the older adults for EBV antibody titers. One year later we retested the older adults to assess whether prior positivity in recall would be associated with better immune functioning, measured via EBV antibody titers and t-cell counts and activation. We also retested recall positivity with a new set of images, as well as recognition memory for the initial images, to assess whether the positivity effect itself shows stability over time.[1] Finally, another year later we retested the older adults' immune functioning via t-cell counts and activation, to determine the stability of these measures and the reliability of our effects. We hypothesized that older adults would show an aging positivity effect, and greater positivity in recall would be associated with better immune functioning as indicated by higher t-cell counts, lower t-cell activation, and fewer EBV antibody titers.

Method

Participants

At Time 1, 57 older adults (M_{age} = 71.84 years, SD_{age} = 5.18 years, range: 65–90; 37 women) were recruited through a community registry and paid for their participation. Fifty younger adults (M_{age} = 18.88 years, SD_{age} = 1.65 years, range: 17–25; 36 women) were undergraduates who participated in exchange for course credit. Forty-six of the older participants returned for blood sampling at Time 2, and 45 returned at Time 3. At Time 2, the Mini-Mental State Examination was administered to screen for cognitive impairment (Folstein, Folstein, & McHugh, 1975). All participants met the cut-off score of 27 points. No participants self-reported any diagnosed cognitive impairment or diagnoses of autoimmune disease.

Measures

The PANAS (Watson, Clark, & Tellegen, 1988) was used to assess positive (Time 1 α = .92; Time 2 α = .90) and negative affect (Time 1 α = .71; Time 2 α = .85) at Times 1 and 2. The Future Time Perspective Scale (Carstensen & Lang, 1996) was used at Time 1 to assess the role of time perspective in the positivity effect and immune functioning, due to the integral role it plays in socio-emotional selectivity theory (α = .80). Frailty was also included as a potential control variable because of its important role in the health of older adults, and was measured using self-reported unintentional weight loss, exhaustion, and physical activity levels as per Fried et al. (2001).[2]

Latent EBV reactivation was assessed at Time 1 by testing for levels of EBV VCA-IgG, the antibody response to the combination of viral proteins that compose the EBV virus coat (Fagundes et al., 2012). We also tested for IgM antibodies to determine if participants had experienced a recent EBV viral infection, which would render the use of EBV IgG to measure latent reactivation inappropriate. Serum samples were stored at −20 °C until tested with the DiaSorin Liaison XL, an automated testing platform that uses indirect chemiluminescense immunoassay technology, and assayed using a p18 synthetic peptide.

We assayed two t-cell markers at Times 2 and 3: $CD4^+$ and $CD8^+$. We also assessed the percentage of $CD4^+$ t-cells activated as reflected in HLA-DR marker up-regulation. Blood was collected into lithium heparin anticoagulant and tested within 72 hr of collection. Cells were stained with monoclonal antibodies (CD3 PerCP, CD4 APC, CD8 Pacific Blue) and the activation marker tested (HLA-DR Fitc; Becton Dickinson), and CD45 V450 was used to distinguish leukocytes; 100 μl of heparin-treated whole blood was added to 20 μl of each of the prepared monoclonal cocktails, and these were mixed and incubated at room temperature for 15 min in the dark, lysed,[3] centrifuged, aspirated, and fixed, then run on a BD Facs Canto II. Full blood counts were also performed.

The 24 images used for the recall task at Time 1 were from Charles, Mather, and Carstensen (2003), and originally drawn from the International Affective Picture System (IAPS: Lang, Bradley, & Cuthbert, 1999). The recall task at Time 2 relied on a novel set of images, which were drawn from the IAPS. As per Time 1, we used eight positive, eight negative, and eight neutral images, half of which were social and half nonsocial. Forty-eight images were used for the recognition task: The original images used in the Time 1 recall task, and 24 matched distracters from Charles et al. (2003).

Procedure

Time 1. Participants provided demographic information and completed the PANAS. They were then told that they would be viewing a series of images on the computer screen, and to watch them as they would a TV. Participants watched the images, which were displayed consecutively for 2 s each in a random order, and then completed a 15-min filler task. After this interval they were asked to free recall the images that they had seen earlier, and then to complete the Future Time Perspective Scale. Within 2 months of this session, older adults returned to provide a blood sample. We only collected blood samples from older adults, as younger adults did not show a positivity effect.

Time 2. Eleven to 13 months after Time 1, older adults returned to the lab and completed the PANAS, after which they viewed 24 new images. After a 15-min filler task they free recalled

[1] It is important to note that at the second point in time older adults would have been aware of the memory component of the positivity task. As a consequence, the positivity effect itself is less likely to emerge (Löckenhoff & Carstensen, 2007; Reed & Carstensen, 2012), and it is also possible that memory goals might cloud the relationship with any positivity effects that emerge at Time 1.

[2] Several additional control measures were included but did not change the nature of the relationships and hence are not reported. For additional details, contact the first author.

[3] A lab error resulted in different lysing methods being used at Time 2 (ammonium chloride) and Time 3 (FACS lyse). The lysing method used at Time 3 results in less cell loss because there are fewer washing steps, which means this lysing method results in more accurate cell counts (Kalina et al., 2012). The cell loss with the method used at Time 2 appeared to target CD8 cells in particular, as there was a lower mean for CD8 counts at Time 2 than Time 3. Follow-up tests using the two lysing methods on a set of test samples confirmed this preferential loss of CD8. As a result, we do not report results with Time 2 CD8 counts (although there was no relationship between this variable and our variables of interest).

these images. They then completed the recognition task for the Time 1 images, in which they were presented with the images and asked to identify whether they had seen them at Time 1. Participants then completed the Mini Mental State Exam (Folstein et al., 1975). Within 2 months of this session, they provided a blood sample.

Time 3. Eleven to 13 months after their Time 2 blood testing session, older adults returned to provide a final blood sample.

Results

For descriptive statistics and intercorrelations among the variables, see Table S1 in the online supplemental materials.

Memory Analyses

A mixed-model ANOVA on recall scores revealed the predicted interaction between valence and age group on recall scores at Time 1, $F(1, 105) = 5.50$, $p = .021$. Simple effects analyses indicated that older adults recalled a greater number of positive ($M = 3.00$, $SD = 1.56$) than negative images ($M = 2.19$, $SD = 1.30$), $F(1, 55) = 16.23$, $p < .001$, whereas no such effect emerged among younger adults (positive $M = 2.78$, $SD = 1.27$; negative $M = 2.66$, $SD = 1.04$), $F(1, 49) = 0.32$, $p = .576$. Older adults recalled significantly fewer negative images than younger adults, $F(1, 105) = 4.12$, $p = .045$, but there was no significant difference between age groups in memory for positive images, $F(1, 105) = 0.63$, $p = .429$. At Time 2, a paired-samples t test showed no difference in recall of positive ($M = 2.63$, $SD = 1.51$) and negative images ($M = 2.61$, $SD = 1.27$), $t(45) = .08$, $p = .935$.

We used signal detection to assess Time 2 recognition memory for Time 1 images. The average proportion of hits (correct identification of images shown at Time 1 as having been seen before) and false alarms (incorrect identification of images not shown at Time 1 as having been seen before) were calculated separately for positive and negative images. The hit and false alarm rates were then used to derive sensitivity (d') and bias (β) statistics separately for the positive and negative images. Participants tended to respond conservatively, meaning that many of our participants had hit and false alarm rates of 0. This is not surprising, given that older adults generally tend to be more conservative than younger adults on recognition tasks (Craik, 1969; Silverman, 1963) and they had not seen the images in a year. Nevertheless, scores of 0 in hit and false alarm rates are problematic for signal detection analyses, because these analyses require the calculation of a z-score, and the corresponding z-score for 0 is infinity. To counter this problem, we added 0.5 to the number of hits and false alarms, and 1 to the number of signal trials and noise trials (as in Hautus, 1995). Paired-samples t tests revealed that the difference between the sensitivity (d') for positive ($M = .08$, $SD = .58$) and negative images ($M = .32$, $SD = .63$) approached significance, $t(45) = 1.95$, $p = .058$, indicating that participants showed a trend toward greater recognition of *negative* than positive images. A significant difference was evident between the bias (β) for positive ($M = 1.10$, $SD = .58$) and negative images ($M = 1.47$, $SD = .92$), $t(45) = 2.13$, $p = .039$, indicating that participants showed a greater bias toward reporting recognition of negative than positive images.

Despite the fact that a positivity effect did not emerge in recall or recognition at Time 2, it remains possible that participants who recalled more positive images at Time 1 were also more likely to recall and recognize positive images at Time 2. To test this possibility, we simultaneously regressed positive and negative recall at Time 2 on positive and negative recall at Time 1. Recall of positive images at Time 1 predicted recall of positive images at Time 2 ($\beta = .47$, $p = .003$), but recall of negative images at Time 1 did not ($\beta = .10$, $p = .49$). In contrast, recall of negative images at Time 1 predicted recall of negative images at Time 2 ($\beta = .58$, $p < .001$), but recall of positive images at Time 1 did not ($\beta = .03$, $p = .835$). The (d') scores for positive and negative images at Time 2 were similarly regressed on positive and negative image recall at Time 1. Positive images recalled at Time 1 did not predict d' for positive images at Time 2 ($\beta = .31$, $p = .069$), nor did negative images recalled at Time 1 ($\beta = -.14$, $p = .39$). Negative images recalled at Time 1 predicted d' for negative images at Time 2 ($\beta = .43$, $p = .011$), but positive images recalled at Time 1 did not ($\beta = -.04$, $p = .815$).

Positivity Effect and Immunity

To test whether Time 1 image recall was associated with Time 1 EBV antibody titers, we regressed $\log_{10}$ transformed Time 1 EBV VCA IgG antibody titers simultaneously on Time 1 positive and negative image recall. Neither positive ($\beta = .003$, $p = .99$) nor negative ($\beta = .10$, $p = .57$) images recalled predicted EBV antibody titers at Time 1. We then ran the same analysis with Time 2 EBV VCA IgG, both with and without Time 1 EBV as a predictor in the analyses. In neither of these analyses did positive or negative images recalled at Time 1 predict EBV antibody titers at Time 2 (all ps $> .40$).

To assess the relationship between Time 1 image recall and Time 2 and Time 3 t-cell counts and activation, we conducted a series of regressions predicting CD4 count and HLA-DR$^+$ activated CD4 cells.[4] First we regressed the Time 2 immune variables onto Time 1 positive and negative recall. Consistent with predictions, Time 1 positive recall was positively associated with Time 2 CD4 counts ($\beta = .34$, $p = .045$) and negatively associated with Time 2 CD4 activation ($\beta = -.49$, $p = .003$). There were no associations with negative recall. Then we regressed the Time 3 immune variables onto Time 1 positive and negative recall. Consistent with predictions, Time 1 positive image recall was positively associated with Time 3 CD4 counts ($\beta = .38$, $p = .025$) and negatively associated with Time 3 CD4 activation ($\beta = -.48$, $p = .003$). Time 1 negative image recall was not associated with Time 3 CD4 counts ($\beta = -.13$, $p = .435$), although it was positively associated with Time 3 CD4 activation ($\beta = .43$, $p = .008$).

Next, we conducted path analyses with recall and CD4 counts and HLA-DR$^+$ activated CD4 cells to determine the pattern of relationships across all three time-points. To test whether the Time 2 variables mediated the relationship between the Time 1 and Time 3 variables, we used bias-corrected bootstrapping with 5,000 resamples to generate estimates and 95% confidence intervals (CIs) of indirect effects. Figure 1 depicts the interrelationships between recall and CD4 percentage activation at HLA-DR across all three study time-points. There was a significant negative relationship

[4] One participant was a univariate outlier on this variable at Times 2 and 3, and thus was excluded from analyses using this variable (results were not substantively different when this participant was included).

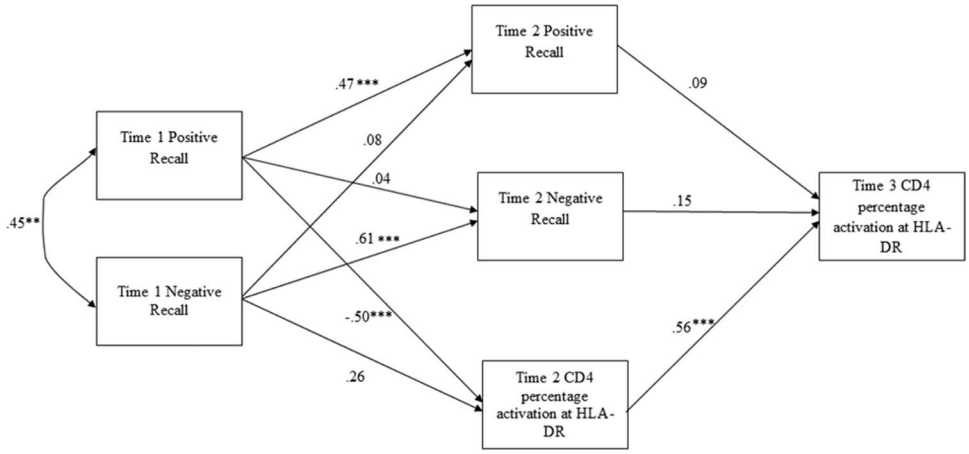

Figure 1. Path model depicting the interrelationships between recall and CD4 percentage activation at HLA-DR across all three study time-points. Numbers are standardized regression coefficients. * $p < .05$. ** $p < .01$. *** $p < .001$.

between Time 1 positive recall and Time 2 CD4 activation, and Time 2 CD4 activation predicted Time 3 CD4 activation, but there was no residual direct relationship between the recall variables and Time 3 CD4 activation. Bootstrapping analyses revealed that the relationship between Time 1 positive recall and Time 3 CD4 activation was mediated through Time 2 CD4 activation (IE = −.76, *SE* = .31, 95% CI [−1.43, −.26], $p = .001$), but not through Time 2 positive recall (IE = .12, *SE* = .16, 95% CI [−.15, .48], $p = .346$) or negative recall (IE = .02, *SE* = .09, 95% CI [−.11, .24], $p = .508$).

Figure 2 depicts the relationships between recall and CD4 counts at all three study time-points. As with CD4 percentage activation at HLA-DR, there was a significant relationship between Time 1 positive recall and Time 2 CD4 count, and Time 2 CD4 count predicted Time 3 CD4 count, but there was no residual direct relationship between the recall variables and Time 3 CD4 count. Bootstrapping analyses revealed that the relationship between Time 1 positive recall and Time 3 CD4 count was mediated through Time 2 CD4 count (IE = .06, *SE* = .03, 95% CI [.02, .13], $p = .004$), but not Time 2 positive recall (IE = .02, *SE* = .01, 95% CI [−.01, .05], $p = .164$) or negative recall (IE = .00, *SE* = .00, 95% CI [−.00, .01], $p = .598$).

The PANAS, the Future Time Perspective Scale, and frailty were uncorrelated with the memory measures, EBV measures, or CD4 counts and activation (all *p*s > .05). CD8 counts at Time 3 were uncorrelated with the memory measures.

Discussion

The current findings demonstrate that positivity in memory is associated with fewer indicators of immunosenescence, suggesting that the aging positivity effect may play a role in healthy aging. At initial testing older but not younger adults recalled more positive than negative images, replicating the aging positivity effect (Reed, Chan, & Mikels, 2014). Although there was no relationship between positivity and EBV antibody titers and no relationship

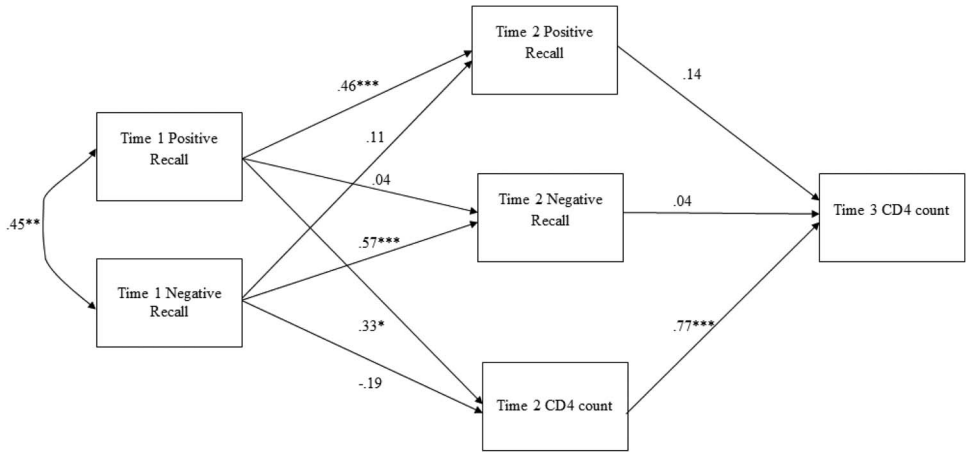

Figure 2. Path model depicting the interrelationships between recall and CD4 counts across all three study time-points. Numbers are standardized regression coefficients. * $p < .05$. ** $p < .01$. *** $p < .001$.

between positivity and CD8 counts, greater positivity in recall predicted higher CD4 counts and lower CD4 activation at HLA-DR for older adults 1 and 2 years later. The relationship CD4 counts and activation after 1 year. The higher CD4 counts and lower CD4 activation associated with positive recall suggest that older adults who show a positivity effect may be able to more effectively cope with pathogen challenges.

The second finding of note was that the positivity effect was stable across a 1-year period. Positivity in recall at initial testing predicted positivity in recall of new material and to a lesser extent recognition of the original images 1 year later. Given the considerable variability among older adults in the degree to which they show the positivity effect (Isaacowitz & Blanchard-Fields, 2012), this stability suggests that intraindividual variability may indeed represent meaningful variance. From the perspective of the current research, the intraindividual stability of the positivity effect raises the possibility that older adults could use positivity as a long-term health maintenance strategy. Nevertheless, the positivity effect itself only emerged in recall at the initial testing session.

We did not examine mediating mechanisms in any depth in the current research, but there are several potential mechanisms through which positivity could lead to improved immune function. The most obvious possibility is that positivity is an emotion up-regulation strategy that leads to increased positive affect, which in turn is associated with better immune function (for a discussion of the link between positive affect and immunity, see Marsland, Pressman, & Cohen, 2007). Although this seems like a plausible account, research on the positivity effect has failed to establish positive affect as an outcome of positivity in information processing (Isaacowitz & Blanchard-Fields, 2012). Indeed, in our study there was no evidence that positive or negative mood mediated the relationship between positivity in memory and enhanced immune functioning. Nevertheless, the current study did not provide a clear test of positive affect as a mediating variable. First, participants only completed the affect measure before the recall task. A test of affect both before and after the recall of positive and negative images would allow us to determine if participants successfully used memory positivity to regulate their emotions, which could be the basis of the positivity/immune function relationship. Second, understanding the relationships between memory positivity, positive affect, and health will require an understanding of the time frame of these relationships: It may be that any affective consequences of memory positivity are temporally offset from the recall event itself. Thus, the role of positive affect remains unclear.

Positive affect is not the only variable that may explain the link between positivity in information processing and immune outcomes. There are a range of other psychological variables that are known to be tied to both emotional processes and immune outcomes, for example stress and coping (Segerstrom & Miller, 2004), optimism (Rasmussen, Scheier, & Greenhouse, 2009), and social relationships (Cohen, 2004). These variables may help to explain the link between memory positivity and immune function: A person who focuses on positive information and avoids negative information may be better able to cope with stressful situations, may take a more positive long-term outlook on life, and may maintain positive social interactions, and hence reap the immune benefits. The current research provides evidence that the aging positivity effect is linked to better immune functioning, but future research will be necessary to clarify the mechanisms that underlie this relationship.

References

Carstensen, L. L., Isaacowitz, D. M., & Charles, S. T. (1999). Taking time seriously: A theory of socioemotional selectivity. *American Psychologist, 54,* 165–181. doi:10.1037/0003-066X.54.3.165

Carstensen, L. L., & Lang, F. R. (1996). *Future time perspective scale*. Unpublished manuscript, Stanford University.

Carstensen, L. L., & Mikels, J. A. (2005). At the intersection of emotion and cognition: Aging and the positivity effect. *Current Directions in Psychological Science, 14,* 117–121. doi:10.1111/j.0963-7214.2005.00348.x

Castle, E., Eisenberger, N. I., Seeman, T. E., Moons, W. G., Boggero, I. A., Grinblatt, M. S., & Taylor, S. E. (2012). Neural and behavioral bases of age differences in perceptions of trust. *Proceedings of the National Academy of Sciences of the United States of America, 109,* 20848–20852. doi:10.1073/pnas.1218518109

Charles, S. T., Mather, M., & Carstensen, L. L. (2003). Aging and emotional memory: The forgettable nature of negative images for older adults. *Journal of Experimental Psychology: General, 132,* 310–324. doi:10.1037/0096-3445.132.2.310

Cohen, S. (2004). Social relationships and health. *American Psychologist, 59,* 676–684. doi:10.1037/0003-066X.59.8.676

Cossarizza, A., Ortolani, C., Monti, D., & Franceschi, C. (1997). Cytometric analysis of immunosenescence. *Cytometry, 27,* 297–313. doi:10.1002/(SICI)1097-0320(19970401)27:4<297::AID-CYTO1>3.0.CO;2-A

Costantino, C. M., Spooner, E., Ploegh, H. L., & Hafler, D. A. (2012). Class II MHC self-antigen presentation in human B and T lymphocytes. *PLoS ONE, 7,* e29805. doi:10.1371/journal.pone.0029805

Craik, F. (1969). Applications of signal detection theory to studies of aging. *Interdisciplinary Topics in Gerontology, 4,* 147–157.

Effros, R. B., Cai, Z., & Linton, P.-J. (2003). CD8 t cells and aging. *Critical Reviews in Immunology, 23,* 45–65. doi:10.1615/CritRevImmunol.v23.i12.30

Fagundes, C. P., Bennett, J. M., Alfano, C. M., Glaser, R., Povoski, S. P., Lipari, A. M., . . . Kiecolt-Glaser, J. K. (2012). Social support and socioeconomic status interact to predict Epstein-Barr virus latency in women awaiting diagnosis or newly diagnosed with breast cancer. *Health Psychology, 31,* 11–19. doi:10.1037/a0025599

Fahey, J. L., Schnelle, J. F., Boscardin, J., Thomas, J. K., Gorre, M. E., Aziz, N., . . . Nishanian, P. (2000). Distinct categories of immunologic changes in frail elderly. *Mechanisms of Ageing and Development, 115,* 1–20. doi:10.1016/S0047-6374(00)00094-4

Folstein, M. F., Folstein, S. E., & McHugh, P. R. (1975). "Mini-mental state:" A practical method for grading the cognitive state of patients for the clinician. *Journal of Psychiatric Research, 12,* 189–198. doi:10.1016/0022-3956(75)90026-6

Fried, L. P., Tangen, C. M., Walston, J., Newman, A. B., Hirsch, C., Gottdiener, J., . . . McBurnie, M. (2001). Frailty in older adults: Evidence for a phenotype. *Journal of Gerontology A: Medical Sciences, 56,* 146–156. doi:10.1093/gerona/56.3.M146

Ginaldi, L., De Martinis, M., D'Ostilio, A., Marini, L., Loreto, M. F., Martorelli, V., & Quaglino, D. (1999). The immune system in the elderly: II. Specific cellular immunity. *Immunologic Research, 20,* 109–115. doi:10.1007/BF02786467

Hautus, M. J. (1995). Corrections for extreme proportions and their biasing effects on estimated values of d′. *Behavior Research Methods, Instruments, & Computers, 27,* 46–51. doi:10.3758/BF03203619

Isaacowitz, D. M., & Blanchard-Fields, F. (2012). Linking process and outcome in the study of emotion and aging. *Perspectives on Psychological Science, 7,* 3–17. doi:10.1177/1745691611424750

Kalina, T., Flores-Montero, J., Van Der Velden, V., Martin-Ayuso, M., Böttcher, S., Ritgen, M., . . . Mendonça, A. (2012). EuroFlow standardization of flow cytometer instrument settings and immunophenotyping protocols. *Leukemia, 26,* 1986–2010. doi:10.1038/leu.2012.122

Lang, P. J., Bradley, M. M., & Cuthbert, B. N. (1999). *International affective picture system (IAPS): Instruction manual and affective ratings*. Gainesville, FL: The Center for Research in Psychophysiology, University of Florida.

Löckenhoff, C. E., & Carstensen, L. L. (2007). Aging, emotion, and health-related decision strategies: Motivational manipulations can reduce age differences. *Psychology and Aging, 22,* 134–146. doi:10.1037/0882-7974.22.1.134

Marsland, A. L., Pressman, S. D., & Cohen, S. (2007). Positive affect and immune function. In R. Ader (Ed.), *Psychoneuroimmunology* (pp. 761–779). Philadelphia, PA: Elsevier. doi:10.1016/B978-012088576-3/50042-3

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. doi:10.1037/0278-6133.8.4.389

Pawelec, G., Barnett, Y., Forsey, R., Frasca, D., Globerson, A., McLeod, J., . . . Gupta, S. (2002). T cells and aging, January 2002 update. *Frontiers in Bioscience, 7,* d1056–d1183. doi:10.2741/Pawelec

Pressman, S. D., & Cohen, S. (2005). Does positive affect influence health? *Psychological Bulletin, 131,* 925–971. doi:10.1037/0033-2909.131.6.925

Rasmussen, H., Scheier, M., & Greenhouse, J. (2009). Optimism and physical Health: A meta-analytic review. *Annals of Behavioral Medicine, 37,* 239–256. doi:10.1007/s12160-009-9111-x

Rea, I. M., McNerlan, S. E., & Alexander, H. D. (1999). CD69, CD25, and HLA-DR activation antigen expression on CD3+ lymphocytes and relationship to serum TNF-α, IFN-γ, and sIL-2R levels in aging. *Experimental Gerontology, 34,* 79–93. doi:10.1016/S0531-5565(98)00058-8

Reed, A. E., & Carstensen, L. L. (2012). The theory behind the age-related positivity effect. *Frontiers in Psychology, 3,* 339. doi:10.3389/fpsyg.2012.00339

Reed, A. E., Chan, L., & Mikels, J. A. (2014). Meta-analysis of the age-related positivity effect: Age differences in preferences for positive over negative information. *Psychology and Aging, 29,* 1–15. doi:10.1037/a0035194

Segerstrom, S. C. & Miller, G. E. (2004). Psychological stress and the human immune system: A meta-analytic study of 30 years of inquiry. *Psychological Bulletin, 130,* 601–630. doi:10.1037/0033-2909.130.4.601

Silverman, I. (1963). Age and the tendency to withhold response. *Journal of Gerontology, 18,* 372–375. doi:10.1093/geronj/18.4.372

Trivers, R. (2010). Deceit and self-deception. In P. M. Kappeler & J. Silk (Eds.), *Mind the Gap* (pp. 373–393). Berlin: Springer.

Trivers, R. (2011). *The folly of fools: The logic of deceit and self-deception in human life*. Philadelphia, PA: Basic Books.

von Hippel, W., & Trivers, R. (2011). The evolution and psychology of self-deception. *Behavioral and Brain Sciences, 34,* 1–16. doi:10.1017/S0140525X10001354

Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. *Journal of Personality and Social Psychology, 54,* 1063–1070. doi:10.1037/0022-3514.54.6.1063

Received June 20, 2013
Revision received May 30, 2014
Accepted June 5, 2014 ■