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Brooke N. Jenkins

Chapman University, bjenkins@chapman.edu

John F. Hunter

University of California, Irvine

Marie P. Cross

University of California, Irvine

Amanda M. Acevedo

University of California, Irvine

Sarah D. Pressman

University of California, Irvine

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When is Affect Variability Bad for Health? The Association between Affect Variability and Immune Response to the Influenza Vaccination

Brooke N. Jenkins, Ph.D.¹, John F. Hunter, M.A.², Marie P. Cross, M.A.², Amanda M. Acevedo², and Sarah D. Pressman, Ph.D.²

¹Chapman University, Department of Psychology

²University of California, Irvine, Department of Psychology and Social Behavior

Abstract

Objectives—This study addresses methodological and theoretical questions about the association between affect and physical health. Specifically, we examine the role of affect variability and its interaction with mean levels of affect to predict antibody (Ab) levels in response to an influenza vaccination.

Methods—Participants (N = 83) received the vaccination and completed daily diary measures of affect four times a day for 13 days. At one and four months post-vaccination, blood was collected from the participants to assess Ab levels.

Results—Findings indicate that affect variability and its interaction with mean levels of affect predict an individual's immune response. Those high in mean positive affect (PA) who had more PA variability were more likely to have a lower Ab response in comparison to those who had high mean PA and less PA variability. Although it did not interact with mean negative affect (NA), NA variability on its own was associated with Ab response, whereby those with less NA variability mounted a more robust immune response.

Conclusion—Affect variability is related to immune response to an influenza vaccination and, in some cases, interacts with mean levels of affect. These oscillations in affective experiences are critical to consider in order to unpack the intricacies of how affect influences health. These findings suggest that future researchers should consider the important role of affect variability on physical health-relevant outcomes as well as examine the moderating effect of mean affect levels.

Keywords

affect variability; antibody response; immune function; negative affect; positive affect

Corresponding Author: Brooke N. Jenkins; Crean Hall 110 Orange, CA, (bjenkins@chapman.edu).

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Introduction

Positive affect (PA), such as feelings of joy or happiness, has been repeatedly tied to better health and physiological function [1–3], while the converse is true of negative affect (NA; e.g., feelings of sadness or anger; [4]). The majority of this research has evaluated affect in a singular fashion: by assessing mean or average levels of affect. This ignores the interesting possibility that naturally occurring *changes* in affect over time, uncaptured by averages, might also have biological relevance [5].

Fluctuations in the experience of affect over time are referred to as affect variability. This construct captures the idea that an individual who varies between extreme highs and lows on NA, for example, is starkly different from an individual with consistently moderate levels of NA. These two individuals could have the same *mean* level of NA, however, and would therefore be considered equal in many past studies about state affect and physical health (see Figure 1). Without consideration of variability, invaluable information about nuances in affect is lost. Critically, with knowledge of the interplay between transient affect and alterations in physiology (e.g., heart rate, blood pressure, immune function; [6, 7]), it seems plausible that these variability differences may have physical health-relevant consequences.

A substantial body of evidence suggests that affect variability may be associated with worse mental health (see meta analytic review [8]). For example, Gruber and colleagues [9] found that greater PA variability was associated with lower life satisfaction, worse psychosocial functioning, and greater depression and anxiety. These findings held even when controlling for mean affect, indicating that variability may predict mental health over and above mean levels of affect. In the same paper, retrospectively captured affect variability in a separate large sample showed that greater PA variability was associated with lower life satisfaction and subjective happiness. Similar to these findings, Hardy and Segerstrom [10] found that middle-aged participants with greater variability in both PA and NA experienced greater psychological distress even when controlling for each respective mean level of affect. These findings indicate that greater affect variability is detrimental to mental health.

While this evidence provides convincing support that affect variability has implications for psychological health, there is a near absence of work examining how affect variability may impact physical health-relevant outcomes. Indeed, variability in other psychological characteristics (e.g., life satisfaction, perceived control) is associated with a variety of physical health outcomes such as higher mortality risk and worse physical health, and physical health-relevant factors such as lower social support [11–13]. There is also the possibility that variability in affect has a physiologically taxing effect on the body. For example, given the known cardiovascular, immune, and hormonal alterations with even subtle affect change (e.g., [14–17]), variability may take additional energy due to repeated physical adjustments. Alternatively, one could argue that variability is healthful given that it gives activated physiological systems a break, which prevents biological exhaustion and wear and tear on these systems (e.g., [18, 19]). Although these predictions suggest affect variability may be tied to physical health, a surprising aspect of the current variability literature is the lack of inclusion of objective health-relevant biomarkers. To our knowledge, only one study has examined the association between affect variability and a health-relevant

biomarker, finding that moderate levels of PA variability were related to daily cortisol profiles that are reflective of better physiological functioning [20]. If we are to better understand the toll affect variability takes on physical health, we must continue to study objective markers of health.

One health-relevant biomarker that may be important in regard to affect variability is antibody (Ab) response to a vaccination, such as the influenza vaccine. Ab response, typically assessed via blood samples, is often used to study how psychosocial factors impact *in vivo* immune function [21–23]. Given the importance of a quick and large rise in Ab to ensure protection against virus exposure [22], vaccination response provides us with a health-relevant indicator of immune functioning. For the influenza vaccine, Ab increases one month post-vaccination represent the maximum response, while Ab levels after that time represent the extent to which the Ab increase is sustained versus declined (e.g., [24]). Critically, affect variability experienced immediately following vaccination might have physiological implications that may be associated with these Ab levels.

In addition to the limitation of the lack of health-relevant biomarkers, previous affect variability and health research has also not included interaction terms between affect variability and mean levels of affect. This may be important because variability may have different implications based on mean levels [25]. For example, an individual with high mean PA may benefit from low variability because he or she would experience consistently high levels of PA. On the other hand, an individual low on mean PA may benefit from high variability because he or she could at least experience *some* instances of high PA, which could provide temporary benefits. However, this also means that he or she will be experiencing instances of extremely low PA when he or she drops far below his or her already low PA level. For NA, similar instances could occur. Individuals with high mean NA may benefit from high variability because this provides “breaks” in NA (when they drop below their usually high NA levels), while those low in mean NA may benefit from low variability so that they stay consistently low on NA. As noted by these examples, the combination of these potential interaction effects may have profound effects on how affect influences health. Although affect papers have not tested this interaction, one study investigated the interaction between variability and mean levels of life satisfaction (which is only moderately correlated with affect [26]) and showed that greater variability was associated with an increase in mortality risk, especially for those with low mean life satisfaction [11].

The goal of the present study is to examine how affect variability is associated with Ab response to an influenza vaccination. This study fills important gaps in the literature by employing a fine-grained methodology to assess affective experiences, measuring a novel healthrelevant biomarker that provides rich information about immunocompetence, and examining previously unexplored interaction effects. Affect variability was measured using the common standard deviation approach (similar to the methods used by the papers reviewed above). This method is advantageous in that it represents affect variability with a single value that is widely used and understood [27–29]. We interacted mean affect with affect variability to uncover whether affect variability has different implications for physical health at different levels of mean affect.

Method

Participants

Participants included 83 undergraduate students ($M_{age} = 18.29$; $SD_{age} = 0.90$; 44% male). Sixty-six percent were Caucasian, 24% were Asian, and 10% were other or mixed ethnicity. Participants were eligible for participation if they were healthy (i.e., no chronic or acute illnesses), were not on a regular medication regimen (with the exception of birth control), had never been vaccinated for influenza, and were not pregnant or breastfeeding. Participants were compensated \$120. All study procedures were approved by the university Institutional Review Board.

Procedures

Participants were run in two cohorts across the fall in consecutive years. Participation in the study lasted for four months. Participants first completed baseline measures and then completed daily diaries four times a day for 13 consecutive days. Data were collected on a handheld computer which alerted participants to complete questionnaires one hour after their wake time and then three, eight, and 10 hours later. On day three, participants received the flu vaccination at a university flu clinic. Before receiving the vaccination, blood was collected to measure Ab levels. At one and four months post-vaccination, blood was again collected from the participants to assess Ab levels.

Measures

Daily affect—Affect was assessed with a checklist of 12 adjectives adapted from the State Adjective Questionnaire [30, 31]. Participants reported how much each adjective represented their current affect at each of the diary entries. NA was assessed with the items jittery, nervous, unhappy, and sad. PA was assessed with the items active, intense, enthusiastic, lively, happy, cheerful, relaxed, and calm. NA and PA items were rated on a scale from 0 (Not at all) to 4 (Extremely). Cronbach's alphas for NA ranged from .56 to .84 and Cronbach's alphas for PA ranged from .68 to .85 across the four time points over 13 days.

Affect variability and mean affect—For the purposes of examining how affect variability after the vaccination influenced Ab response, only those days on or after the flu vaccine (i.e., days 3 through 13) were used to create the affect variability values¹. Therefore, adjective items were averaged over each of the 44 time points (11 days* 4 assessments) to create a NA and PA mean value (split-half reliabilities were 0.91 and 0.90, respectively). Then, standard deviations over the 44 time points were calculated for NA and PA (split-half reliabilities were 0.67 and 0.68, respectively; reliability estimates reflect similar ones reported in previous affect variability literature [32, 33]). These calculations resulted in the following variables used for analyses: NA mean (NA_{MEAN}), PA mean (PA_{MEAN}), NA standard deviation (NA_{SD}), and PA standard deviation (PA_{SD}).

Antibody response to vaccination—Ab levels were assessed using venipuncture blood sampling. A 20-mL sample of blood was collected immediately before the immunization

¹We used only these days because past research has shown that psychological variables are more influential after the vaccine [42].

occurred (i.e., baseline) and at one and four month follow-up appointments. The Fluzone vaccine consisted of three antigens: A/New Caledonia, A/Panama, and B/Yamanashi or B/Victoria (B/Victoria was substituted for B/Yamanashi in the second year of data collection). Because previous literature has found psychological associations with changes in Ab levels with A viruses [21] and past work with this data set has found associations with only the A/New Caledonia virus [34], for the purposes of this study, only the A/New Caledonia virus was considered.

Ab titers were quantified using a standard hemagglutination inhibition protocol. To quantify the volume of a participant's Ab level, his or her serum was diluted with various saline concentrations and then added to a red blood cell culture that contained influenza. The titer is the reciprocal of the highest dilution at which a person's serum continues to prevent red cells from clumping. Thus, higher titer values indicate greater volumes of antibodies to the vaccine component. All samples were run in duplicate as well as a nonantigen control, and all time points for each participant were run in the same assay contemporaneously. The antigen used to check Ab levels for the A/New Caledonia virus was A/New Caledonia/20/99 with a hemagglutination titer of 1024 used at four hemagglutinating units (HAU)/25 μ L. The A/New Caledonia was obtained from the World Health Organization collaborating center.

Statistical Analysis

The dependent variable in all analyses was Ab level at either one or four months post-vaccination. The independent variables of interest were NA_{SD} and PA_{SD} and their interactions with NA_{MEAN} and PA_{MEAN} , respectively. NA_{MEAN} , PA_{MEAN} , NA_{SD} , and PA_{SD} were all centered to allow for ease of their interpretations in interaction terms. An interaction term between NA_{SD} and NA_{MEAN} allowed for a test of whether different values of variability in NA had differential implications for Ab response at different levels of mean NA. The same was true for the interaction between PA_{SD} and PA_{MEAN} . Additionally, before interaction terms were added into the model, models with only the main effects were tested.

Ordinary least squares regression models [35] were used to investigate the effect of the independent variables on Ab level at one and four months post-vaccination. Due to problems of non-constant error variance that was likely due to substantial negative skewness of the outcome variable that could not be alleviated using transformations, robust standard errors were used in all ordinary least squares regression models. Individuals who started with baseline levels at maximum level ($N = 5$) were excluded from the analyses, as this would not allow us to see a change in Ab level.

Consistent with previous research (e.g., [34]), standard control variables were included in analyses: baseline Ab level (i.e., immediately before the vaccination occurred), study cohort, sex, and ethnicity (Caucasian = 1; other = 0).

A power analysis revealed that for a linear regression with 7 predictors (e.g., NA_{SD} , NA_{MEAN} , $NA_{SD} * NA_{MEAN}$, baseline Ab level, female, Caucasian, and cohort), 80 participants would be required to detect a medium to large effect size with 80% power and an alpha error probability of 0.05 [36].

Results

Descriptive Statistics

NA_{MEAN} and PA_{MEAN} were significantly different in average value ($t(82) = 8.40, p < .001$, 95% CI of the difference [0.54, 0.88]), while NA_{SD} and PA_{SD} averages were closer in value but still significantly different ($t(82) = 2.33, p = .011$, 95% CI of the difference [0.01, 0.08]; see Table 1). NA_{MEAN} and PA_{MEAN} were negatively correlated ($r = -0.31, p = .004$), but NA_{SD} and PA_{SD} were positively correlated ($r = 0.50, p < .001$; see Table 1). In other words, those with greater NA variability also experienced greater PA variability. Although NA_{MEAN} was positively correlated with NA_{SD} ($r = 0.63, p < .001$), PA_{MEAN} was not correlated with PA_{SD} ($r = 0.16, p = .140$).

Affect Variability and Antibody Response

NA_{SD} was a predictor of Ab level at one and four months post vaccination (see Tables 2 and 3; one month: $b = -574.31, SE = 271.41, p = .038$, 95% CI [-1,115.35, -33.27]; four months: $b = -692.48, SE = 274.86, p = .014$, 95% CI [-1,240.54, -144.42]) when placed in a model not controlling for NA_{MEAN}. This indicated that individuals who were lower in NA variability were more likely to exhibit higher levels of Ab post-vaccination. However, after controlling for NA_{MEAN}, NA_{SD} was not significantly associated with Ab level at one month (see Table 2; $b = -485.84, SE = 341.59, p = .159$, 95% CI [-1,166.96, 195.28]) and marginally associated with Ab level at four months post-vaccination (see Table 3; $b = -660.30, SE = 374.67, p = .082$, 95% CI [-1,407.55, 86.96]). Additionally, there was no significant interaction between NA_{SD} and NA_{MEAN} (see Tables 2 and 3; one month: $b = -201.65, SE = 681.71, p = .768$, 95% CI [-1,561.28, 1,157.98]; four months: $b = 64.43, SE = 696.26, p = .927$, 95% CI [-1,324.58, 1,453.44]).

PA_{SD} was marginally associated with Ab levels at one and four months post-vaccination (see Tables 2 and 3; one month: $b = -456.76, SE = 270.85, p = .096$, 95% CI [-996.70, 83.18]; four months: $b = -533.23, SE = 269.33, p = .052$, 95% CI [-1,070.26, 3.79]). When controlling for PA_{MEAN}, this association became non-significant at one month (see Table 2; $b = -422.03, SE = 265.01, p = .116$, 95% CI [-950.45, 106.39]) and stayed marginally significant at four months post vaccination (see Table 3; $b = -515.75, SE = 267.27, p = .058$, 95% CI [-1,048.80, 17.30]). However, PA_{SD} did significantly interact with PA_{MEAN} to predict Ab levels at both time points (see Tables 2 and 3; one month: $b = -2,070.98, SE = 604.88, p = .001$, 95% CI [-3,277.37, -864.59]; four months: $b = -1,653.02, SE = 508.10, p = .002$, 95% CI [-2,666.66, -639.38]). Specifically, at high PA_{MEAN}, individuals with higher levels of PA_{SD} (i.e., more variability) were more likely to have lower Ab levels at follow-up (see Figures 2 and 3). This is evidenced by the negative slope of PA_{SD} at high PA_{MEAN} (one month: $dy/dx = -1,510.03, SE = 339.70, p < .001$, 95% CI [-2,187.54, -832.52]; four months: $dy/dx = -1,379.26, SE = 270.44, p < .001$, 95% CI [-1,918.77, -839.75]). In contrast, when examining the simple effects of the slope of PA_{SD} at low PA_{MEAN}, the slope was not significantly different from zero (one month: $dy/dx = 602.36, SE = 421.98, p = .158$, 95% CI [-239.26, 1,443.99]; four months: $dy/dx = 306.82, SE = 413.56, p = .461$, 95% CI [-518.20, 1,131.84]), indicating that PA_{SD} had no effect on Ab levels for individuals with lower PA_{MEAN}.

Interestingly, when the NA and PA variables were placed in the same model, the findings for the interaction between PA_{SD} and PA_{MEAN} (see Tables 2 and 3; one month: $b = -2,255.78$, $SE = 654.36$, $p = .001$, 95% CI $[-3,561.88, -949.67]$; four months: $b = -1,882.19$, $SE = 643.69$, $p = .005$, 95% CI $[-3,167.36, -597.01]$) as well as the main effect of NA_{SD} (see Tables 2 and 3; one month: $b = -762.24$, $SE = 383.19$, $p = .051$, 95% CI $[-1,527.09, 2.62]$; four months: $b = -909.15$, $SE = 448.09$, $p = .047$, 95% CI $[-1,803.80, -14.51]$) held².

Discussion

Our findings indicate that affect variability, and the interaction between that variability and mean levels of affect, significantly influence immunocompetence in response to the flu vaccination. NA variability, along with the interaction between PA_{MEAN} and PA variability, predicted Ab levels following a vaccination, and these findings held when they were all placed in the same model (i.e., model 9 in Tables 2 and 3). These results suggest that affect variability may have important implications for physical health, especially in the context of the immune system.

The results regarding PA are nuanced and depend on the interaction between PA mean and variability. Individuals high in PA_{MEAN} who had low variability (consistently stayed at their high PA level as opposed to drastically bouncing up and down around it) had a robust immune response. However, if an individual had a high PA_{MEAN} , high PA variability was detrimental for mounting a large Ab response. It is possible that for an individual with high PA variability, the negative ramifications of dropping far below a mean level are not offset by the peaks of being above a mean level. In contrast, for individuals with low levels of PA_{MEAN} , variability in PA had no effects on Ab levels (see Figures 2 and 3). These PA findings are consistent with some of the current affect variability and physiological findings (e.g., [20] Study 1).

High NA variability was associated with lower Ab levels (see model 1 in Tables 2 and 3). However, when interpreting the meaning of these results, it is important to consider the connection between mean levels and variability. Standard deviations can be confounded with mean levels (e.g., [37]), and indeed, NA_{MEAN} and NA_{SD} were highly correlated in this study (see Table 1). Therefore, it was important to control for NA_{MEAN} when determining the effect of NA_{SD} on Ab levels. When controlling for NA_{MEAN} , the main effect of NA_{SD} became nonsignificant at one month and marginal at four months post vaccination. This could imply that NA_{MEAN} was an important factor driving the association between NA_{SD} and Ab levels. However, the coefficient of NA_{SD} did not dramatically reduce from model 1 without the mean level control (one month: -574.31 ; four months: -692.48) to model 3 with the mean level control (one month: -485.84 ; four months: -660.30), suggesting that maybe with more participants, this effect would have remained significant. Nevertheless, given the high correlation between mean level and standard deviation, we tested our models with the

²Past studies have looked at other variables including loneliness, social network, and stress related to vaccine response (including studies that have used this data set). When considering these psychosocial variables, the pattern of results, especially the interaction terms of interest, was unaffected. The only exceptions to this were that PA_{SD} became significant as a main effect and NA_{SD} became non-significant in some models. This non-significance may reflect the substantial overlap in NA_{SD} with loneliness ($r = 0.47$, $p < .001$) and stress ($r = 0.52$, $p < .001$).

alternative variability indicator of the mean square successive differences (MSSD). We found no main effect of NA_{MSSD} in any of the models. Interestingly, NA_{MEAN} and NA_{MSSD} were not significantly correlated ($r = 0.16, p = 0.16$), in contrast to the high correlation between NA_{MEAN} and NA_{SD} ($r = 0.63, p < .001$). This may suggest that even when NA variability is not confounded with mean levels, NA variability has little effect on biomarkers (at least in healthy college samples). With regard to PA, PA_{MEAN} and PA_{SD} were not confounded (see Table 1), which may explain why there were interactive effects of PA_{MEAN} and PA_{SD} on Ab levels. It is interesting that NA variability is related to NA_{MEAN} levels while PA variability is not related to PA_{MEAN} levels. Indeed, many studies only find effects with PA variability as opposed to NA variability (e.g., [9,20]), providing evidence that these two valence types should be examined as separate variables as opposed to combining them into one variability variable.

Why is affect variability associated with Ab levels? One explanation may be connected to the influence of affect variability on affective processes. High affect variability may be indicative of poor affect regulation, which has implications for health [38, 39]. Additionally, affect variability is a key factor in neuroticism (e.g., [40]), and high levels of neuroticism have been shown to negatively influence immune response [41]. Alternatively, affect variability might be related to health through its influence on health behaviors. For example, it is possible that affect variability impacts sleep, eating behaviors, and/or the use of alcohol and tobacco (all of which have been shown to affect immunity [22, 42]). Yet another explanation could be that affect variability may alter the concentration of circulating stress-related hormones (e.g., cortisol, epinephrine, norepinephrine), which can bind to and alter the function of immune cells involved in the Ab response [43]. These possible pathways are likely interrelated and may all contribute in some way to how affect variability gets “under the skin” to influence Ab responses to vaccines. Thus, future research should consider these potential pathways in the affect variability and Ab response relationship to determine *how* affect variability influences immune responses.

This study has several potential limitations. First, although we used a common metric of affect variability, namely, the standard deviation approach, there are also other methods (e.g., insufficient variations, adjusted squared successive difference scores, core affect variability; [40, 44–47]) that were not considered. However, we chose the standard deviation approach since it is easy to understand and the most commonly used technique, in addition to the fact that different variability measures often lead to similar results (e.g., [9, 46], our results reported above with MSSD). A second limitation was that we did not include the effect of affect variability before the vaccination. Because past research has shown that psychological variables are more influential after the vaccine [42], we used only the days on or after the flu vaccination to calculate variability³. Third, we only considered one virus. This decision was based on previous findings that psychosocial variables influence responses to only some virus strains [24, 41]. Although it is difficult to say whether certain viruses are more sensitive to psychosocial factors, it has been suggested that there may be differences in strain

³Furthermore, we felt that the two days before the vaccination would not provide sufficient data because participants were still new to self-reporting and that two days might not be sufficient to capture a large enough sample to reflect variability before the vaccination. Therefore, the first two days were considered to be a diary run-in.

novelty and the participant's previous exposure to the strain that could explain the differing findings in the literature [48].

Fourth, statistical significance does not translate to clinical significance. In our sample, all but 5 participants at one month and all but 6 participants at four months post vaccination had Ab levels above 40 titers, which is considered the clinically protective level [49, 50]. Similar vaccine studies using young adult samples have shown that the majority of the sample are protected against the virus after vaccination (e.g., [34, 51]). Nevertheless, there was sufficient titer variability within our sample to see statistically significant associations with affect variability and mean levels. Additionally, we included pre-vaccine levels as a control variable in all analyses (as suggested by [49]). Therefore, our findings still speak to how affect variability may influence immune responses to vaccines in young, healthy individuals.

This study emphasizes the importance of assessing the effects of affect variability in addition to mean levels of affect on an objective health-relevant biomarker. Mean levels of affect do not explain the whole story about how an individual may mount an immune response. Previous research may have overlooked important intricacies about the influence of affect because variability was not assessed. In our study, if we only considered mean levels of PA or NA, then we would have found no influence of affect on Ab response. This would have resulted in an incomplete understanding of the effects of affect on vaccination response. The results of this study should encourage future researchers to consider affect variability in addition to mean levels of affect and point to the possibility that it may have unique effects on other physical health-relevant parameters. Assessing vacillations in affect will help paint a more vivid picture of how our affective experiences influence physical health.

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Highlights

- Affect variability has important implications for immune response.
- Negative affect variability is related to lower antibody production.
- Positive affect variability and mean levels interact to influence antibody production.
- Future researchers should consider how affect variability influences health.

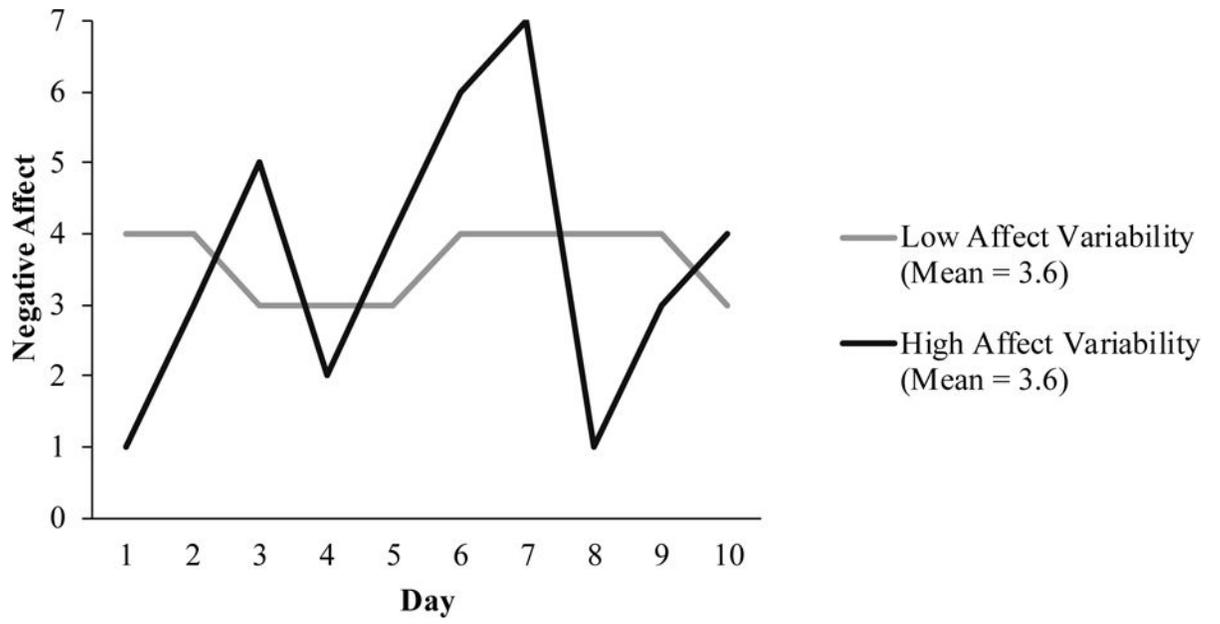


Figure 1. Two individuals with the same mean level of negative affect but different negative affect variability.

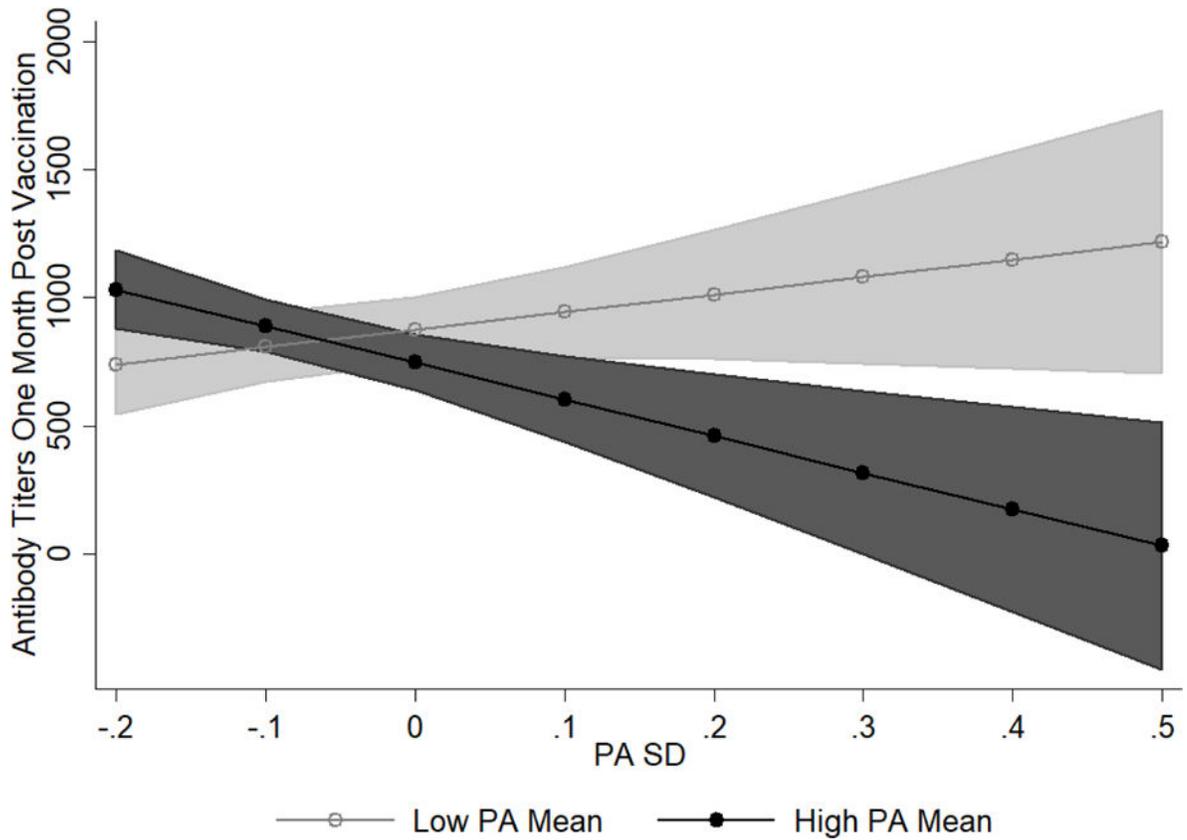


Figure 2. PA_{SD} and PA_{MEAN} interaction on antibody (Ab) titers at one month post-vaccination. Low PA_{MEAN} is one SD below the mean on PA_{MEAN} while high PA_{MEAN} is one SD above the mean on PA_{MEAN} . Lines represent adjusted predictions. Shaded regions are the 95% confidence intervals around the predictions. Regions of PA_{MEAN} (low vs. high) that do not overlap are significantly different from one another in terms of the predicted Ab level. Possible predicted values are between 0 and 1024 titers. However, confidence intervals exceed this range.

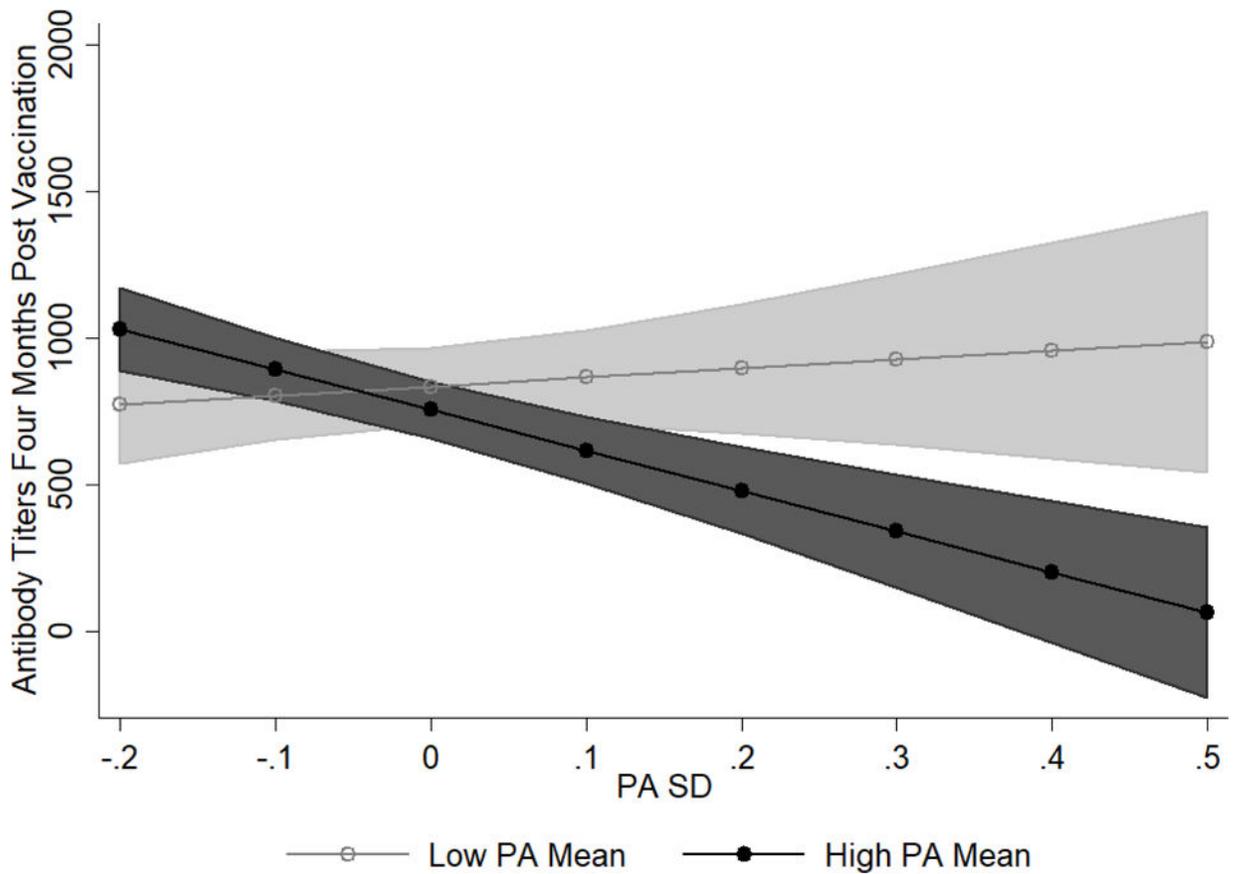


Figure 3. PA_{SD} and PA_{MEAN} interaction on antibody (Ab) titers at four months post-vaccination. Low PA_{MEAN} is one SD below the mean on PA_{MEAN} while high PA_{MEAN} is one SD above the mean on PA_{MEAN} . Lines represent adjusted predictions. Shaded regions are the 95% confidence intervals around the predictions. Regions of PA_{MEAN} (low vs. high) that do not overlap are significantly different from one another in terms of the predicted Ab level. Possible predicted values are between 0 and 1024 titers. However, confidence intervals exceed this range.

Table 1

Descriptive Statistics of Affect Mean and Standard Deviation Measures.

Measure	Mean	SD	2	3	4
1. NA _{MEAN}	0.81	0.43	-0.31 ^{**} (-0.50, -0.10)	0.63 ^{**} (0.48, 0.75)	0.11 (-0.11, 0.32)
2. PA _{MEAN}	1.52	0.51		-0.31 ^{**} (-0.49, -0.10)	0.16 (-0.06, 0.37)
3. NA _{SD}	0.46	.17			0.50 ^{**} (0.32, 0.64)
4. PA _{SD}	0.50	.16			

Note. NA = Negative Affect; PA = Positive Affect; SD = Standard Deviation. The last three columns are the correlation matrix of the variables. 95% confidence intervals are in parentheses.

^{**} $p < 0.01$;

^{*} $p < 0.05$

Table 2

Standard Deviation and Mean of NA and PA Predicting Antibody Level at 1 Month Post-Vaccination.

	NA					PA			NA and PA
	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7	Model 8	Model 9
NA _{SD}	-574.31 [*] (-1,115.35, -33.27) 271.41		-485.84 (-1,166.95, 195.28) 341.59	-460.24 (-1,181.29, 260.82) 361.53					-762.24 [†] (-1,527.09, 2.62) 383.19
NA _{MEAN}		-164.19 (-367.84, 39.45) 102.16	-54.36 (-321.46, 212.74) 133.96	-68.03 (-337.04, 200.98) 134.88					-27.04 (-276.62, 222.54) 125.04
NA _{SD} [*]				-201.65 (-1,561.28, 1,157.98) 681.71					-204.02 (-1,307.20, 899.16) 552.69
NA _{MEAN}									
PA _{SD}					-456.76 [†] (-996.70, 83.18) 270.85		-422.03 (-950.45, 106.39) 265.01	-453.83 [†] (-906.75, -0.92) 227.09	4.80 (-660.25, 669.86) 333.19
PA _{MEAN}						-74.76 (-259.76, 110.24) 92.80	-52.56 (-228.7, 123.66) 88.38	-118.26 (-289.98, 53.45) 86.10	-223.91 [*] (-399.12, -48.70) 87.78
PA _{SD} [*]									
PA _{MEAN}								-2,070.98 ^{**} (-3,277.37, -864.59) 604.88	-2,255.77 ^{**} (-3,561.88, -949.67) 654.36
Baseline Ab Level	0.32 (-1.51, 2.15) 0.92	0.42 (-1.43, 2.28) 0.93	0.29 (-1.59, 2.16) 0.94	0.34 (-1.61, 2.29) 0.98	0.64 (-1.02, 2.30) 0.83	0.86 (-0.68, 2.40) 0.77	0.76 (-0.76, 2.28) 0.76	0.84 (-0.46, 2.13) 0.65	0.63 (-0.85, 2.10) 0.74
Female (1 = female)	26.28 (-136.03, 188.58) 81.42	15.88 (-147.79, 179.54) 82.10	27.34 (-137.77, 192.44) 82.80	27.60 (-137.79, 193.00) 82.93	12.34 (-146.96, 171.65) 79.91	-9.39 (-169.63, 150.85) 80.38	4.28 (-153.33, 161.89) 79.05	43.98 (-107.45, 195.40) 75.92	58.64 (-96.97, 214.25) 77.96
Caucasian (1 = Caucasian)	163.04 [†] (-12.62, 338.71) 88.12	146.68 [†] (-26.76, 320.11) 87.00	163.67 [†] (-13.31, 340.66) 88.76	158.17 [†] (-29.68, 346.02) 94.18	149.42 [†] (-23.76, 322.60) 86.87	127.07 (-47.43, 301.56) 87.53	146.59 (-29.68, 322.86) 88.40	88.26 (-80.44, 256.96) 84.58	102.64 (-64.23, 269.50) 83.60
Cohort	-334.34 ^{**} (-523.50, -145.17) 94.89	-347.37 ^{**} (-540.75, -153.99) 97.01	-336.17 ^{**} (-526.99, -145.35) 95.70	-335.36 ^{**} (-528.35, -142.37) 96.76	-328.58 ^{**} (-524.23, -132.92) 98.15	-339.11 ^{**} (-538.28, -139.93) 99.91	-323.58 ^{**} (-521.84, -125.33) 99.43	-312.62 ^{**} (-505.06, -120.19) 96.48	-299.16 ^{**} (-461.92, -136.41) 81.54
Constant	749.47 ^{**}	802.09 ^{**}	757.46 ^{**}	769.29 ^{**}	771.96 ^{**}	800.79 ^{**}	772.91 ^{**}	807.53 ^{**}	777.89 ^{**}

Note. Robust 95% confidence intervals in parentheses; Robust standard errors below confidence intervals; NA = Negative Affect; PA = Positive Affect; SD = Standard Deviation; Ab = Antibody.

**
p < 0.01

*
p < 0.05

$p < .01$

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Table 3

Standard Deviation and Mean of NA and PA Predicting Antibody Level at 4 Months Post-Vaccination.

	NA				PA				NA and PA
	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7	Model 8	Model 9
NA _{SD}	-692.48* (-1,240.54, -144.42) 274.86		-660.30[†] (-1,407.55, 86.96) 374.67	-668.69[†] (-1,436.65, 99.28) 384.96					-909.15* (-1,803.80, -14.51) 448.09
NA _{MEAN}		-173.45 (-381.26, 34.35) 104.22	-20.14 (-321.67, 281.38) 151.18	-15.58 (-325.40, 294.25) 155.31					17.26 (-274.40, 308.91) 146.08
NA _{SD} [*]				64.43 (-1,324.58, 1,453.44) 696.26					71.52 (-1,063.81, 1,206.86) 568.64
PA _{SD}					-533.23[†] (-1,070.26, 3.79) 269.33		-515.75[†] (-1,048.80, 17.30) 267.27	-536.22* (-1,003.79, -68.64) 234.38	-26.88 (-681.64, 627.89) 327.95
PA _{MEAN}					-54.67 (-232.01, 122.67) 88.94		-29.75 (-197.40, 137.90) 84.06	-78.48 (-237.20, 80.24) 79.56	-191.49* (-343.80, -39.19) 76.28
PA _{SD} [*]									-1,882.18** (-2,666.66, -639.38) 643.69
PA _{MEAN}									
Baseline Ab Level	1.22[†] (-0.13, 2.58) 0.68	1.39[†] (-0.00, 2.79) 0.70	1.21[†] (-0.17, 2.59) 0.69	1.19 (-0.25, 2.64) 0.72	1.60* (0.31, 2.89) 0.65	1.79** (0.46, 3.11) 0.67	1.66* (0.34, 2.99) 0.67	1.72* (0.40, 3.05) 0.66	1.44* (0.01, 2.87) 0.71
Female (1 = female)	-20.07 (-186.37, 146.23) 83.40	-36.75 (-206.29, 132.79) 85.03	-19.96 (-187.97, 148.04) 84.24	-20.00 (-189.30, 149.30) 84.86	-34.31 (-199.98, 131.37) 83.09	-55.73 (-222.35, 110.89) 83.56	-38.24 (-200.47, 123.98) 81.34	-8.12 (-169.26, 153.02) 80.77	6.46 (-155.95, 168.86) 81.34
Caucasian (1 = Caucasian)	120.24 (-59.23, 299.71) 90.01	100.75 (-78.64, 280.14) 89.97	120.99 (-59.30, 301.28) 90.40	122.65 (-71.10, 316.40) 97.12	98.43 (-78.78, 275.64) 88.87	73.89 (-110.07, 257.86) 92.26	96.04 (-87.39, 279.47) 91.97	52.33 (-133.17, 237.83) 92.98	76.06 (-112.06, 264.17) 94.22
Cohort	-297.24** (-496.86, -97.61) 100.12	-315.96** (-521.00, -110.92) 102.83	-298.38** (-499.62, -97.13) 100.90	-298.54** (-501.93, -95.15) 101.95	-286.52** (-491.93, -81.12) 103.01	-303.61** (-513.96, -93.26) 105.49	-282.74** (-490.26, -75.22) 104.05	-277.59** (-483.26, -71.92) 103.10	-265.54** (-464.85, -66.22) 99.83
Constant	741.47**	803.87**	744.18**	740.42**	771.58**	805.79**	772.31**	799.21**	746.51**

Note. Robust 95% confidence intervals in parentheses; Robust standard errors below confidence intervals; NA = Negative Affect; PA = Positive Affect; SD = Standard Deviation; Ab = Antibody.

**
p < 0.01

*
p < 0.05

$p < .01$

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