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UNIVERSITY OF CALIFORNIA, IRVINE

Affect Variability is Constantly Important: Implications for Health

DISSERTATION

To be submitted in partial satisfaction of the requirements for the degree of

DOCTOR OF PHILOSOPHY

in Psychology and Social Behavior

by

Brooke N. Jenkins, M.S.

Dissertation Committee:

Associate Professor Sarah D. Pressman, Ph.D., Chair

Associate Professor Michelle A. Fortier, Ph.D.

Professor Karen S. Rook, Ph.D.

Professor Susan T. Charles, Ph.D.

2017



## TABLE OF CONTENTS

	Page
ACKNOWLEDGEMENTS	iv
CURRICULUM VITAE	vii
ABSTRACT OF THE DISSERTATION	xix
CHAPTER 1: Introduction	1
Figure 1: Two individuals with the same mean level of negative affect but different negative affect variability	3
Figure 2: Two individuals with different patterns of affect variability	3
References	6
CHAPTER 2: Methodological Issues of Affect Variability and a Brief Review of the Affect Variability and Health Literature	8
Measuring Self-Reported State Affect	9
Calculating Affect Variability	10
Table 1: Methods to assess affect variability	11
Other Methodological Issues Associated with Affect Variability	24
Figure 1. Two individuals with the same mean negative affect but with different standard deviations	27
Affect Variability and Health and Health-relevant Outcomes	28
Table 2: Summary of health and health-relevant factors associated with affect variability	29
Conclusion	33
References	35
CHAPTER 3: When is Affect Variability Bad for Health? The Association between Affect Variability and Immune Response to the Influenza Vaccination	40
Abstract	41
Introduction	42
Figure 1: Two individuals with the same mean level of negative affect but different negative affect variability	43
Method	45
Results	49
Table 1: Descriptive statistics of affect mean and standard deviation measures	50
Table 2: Standard deviation and mean of negative affect and positive affect predicting antibody level at one month post-vaccination	52
Table 3: Standard deviation and mean of negative affect and positive affect predicting antibody level at four months post-vaccination	53
Figure 2: $PA_{MEAN}$ and $PA_{SD}$ interaction on antibody (Ab) levels at one month post-vaccination	55
Figure 3: $PA_{MEAN}$ and $PA_{SD}$ interaction on antibody (Ab) levels at four months post-vaccination	56

Discussion	57
Acknowledgements	60
References	61
CHAPTER 4: Affect Variability and Predictability: Using Recurrence Quantification Analysis to Better Understand How the Dynamics of Affect Relate to Health	66
Abstract	67
Introduction	68
Figure 1: Two individuals with different predictability of affect variability	70
Study 1	73
Table 1: Data simulation parameters and predicted results	74
Figure 2: Data analysis for recurrence quantification analysis	76
Figure 3: Mean, standard deviation, percent recurrence (%REC), and percent determinism (%DET) values by group	79
Table 2: Mean, standard deviation, and recurrence quantification measures by group	80
Study 2	84
Table 3: Mean, standard deviation, and pearson's correlation of affect metrics	89
Table 4: Variability metrics of negative affect predicting depressive symptoms	92
Table 5: Variability metrics of positive affect predicting depressive symptoms	93
Table 6: Variability metrics of negative affect predicting negative well-being	94
Table 7: Variability metrics of positive affect predicting negative well-being	95
Table 8: Variability metrics of negative affect predicting somatic symptoms	96
Table 9: Variability metrics of positive affect predicting somatic symptoms	97
Figure 4: Interaction between $NA_{MEAN}$ and $NA_{SD}$ predicting depressive symptoms and negative well-being	98
Figure 5: Interaction between $PA_{MEAN}$ and $PA_{SD}$ predicting depressive symptoms	99
Figure 6: Interaction between $NA_{MEAN}$ and $NA_{%REC}$ predicting depressive symptoms and somatic symptoms	100
Figure 7: Interaction between $NA_{MEAN}$ and $NA_{%DET}$ predicting depressive and somatic symptoms	101
Figure 8: Interaction between $NA_{SD}$ and $NA_{%REC}$ predicting negative well-being	102
Figure 9: Interaction between $PA_{SD}$ and $PA_{%REC}$ predicting depressive symptoms	102
Figure 10: Interaction between $NA_{%DET}$ and $NA_{SD}$ predicting depressive symptoms and negative well-being	103
Figure 11: Interaction between $PA_{%DET}$ and $PA_{SD}$ predicting depressive symptoms and somatic symptoms	104
Figure 12: Three-way interaction between $PA_{MEAN}$ , $PA_{SD}$ , and $PA_{%DET}$ predicting negative well-being and somatic symptoms	105
General Discussion	110
Acknowledgements	112
References	113
CHAPTER 5: Epilogue	116
Figure 1: Two individuals with the same mean negative affect but with different standard deviations	119
References	123

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and I am looking forward to also calling you a colleague as I begin at Chapman. Dr. Kain, thank you for developing me into the researcher I am today. Throughout my time at UC Irvine, you have pushed me to succeed beyond what I could have imagined. I look forward to our continual collaboration.

It has been an absolute joy to be a graduate student in the Department of Psychology and Social Behavior (PSB) at UC Irvine. The faculty and staff make the department shine and excel. I would like to thank Susan Charles, Liz Martin, Roxy Silver, Nick Scurich, Ray Novaco, Wendy Goldberg, Doug Granger, Paul Piff, Linda Levine, and Ilona Yim, who all supported me in my academic development. Susan, thank you for serving on my dissertation committee. It has been a great pleasure learning from you both in and outside of the classroom. Liz, Roxy, Nick, Ray, Wendy, Doug, and Paul, thank you for your support in my job search. Your feedback and encouragement allowed me to succeed. Linda and Ilona, thank you for inspiration in the classroom. I would also like to thank the department staff, including Diane, Allison, Toni, and Claudia, for creating a positive atmosphere that has made this department so special. I would especially like to thank Claudia for all her support as Graduate Program Coordinator. Claudia, your outgoing and warm personality and your passion for student success always lifts my spirits.

The friendships I have made throughout graduate school will forever be remembered. Amanda Acevedo, Marie Cross, and Johnny Hunter, thank you for being my support network both in school and life. Thank you for not only sharing an office with me but sharing this entire journey with me. We have had so much fun together and created so many memories – I look forward to the memories to come! Thank you to my cohort, Adam, Becky, Jason, Karina, Lauren, Nicky, and Rachel, and thank you to all the other graduate students at UC Irvine, Alex, Alina, Becca, Christie, Connor, Cortney, Desi, Dmitry, Emily H., Emily U., Eric, Jared, Joanna, Kate, Kevin, Mayan, Melody, and Sean, whom I am honored to call my friends. Thank you to

Eva, Ariana, Robert, Marla, and Haydee, my friends from the Center on Stress and Health, who always brighten my day. Eva, thank you for all that you do. You are truly amazing.

Thank you to my family for their continual support in my life and career goals. I would like to thank my little sister, Samantha, and my parents, Becky and Tony Gentle. Thank you, Mom and Dad, for your continual support throughout my academic journey. You have always supported my decisions and your continual support has given me confidence in the decisions I have made. Samantha, thank you for being such a fun-loving sister whom I respect and admire. Your work as a teacher has truly been influential in my career path.

Without the support of my husband, Nick Jenkins, I would not be where I am today. Nick, thank you for motivating me to persist and succeed throughout my graduate studies. Thank you for encouraging me in my research and career pursuits. Thank you for listening to my presentations and giving me feedback, reading my papers, discussing my science with me, and truly inspiring me in every way. Thank you for the sacrifices you have made and the continual love you show me. I love you.

I thank God for leading and providing for me throughout this journey and for blessing me with this tremendous group of people who have made me who I am today.



**CURRICULUM VITAE**  
**BROOKE N. JENKINS**

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**RESEARCH OVERVIEW**

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My research program focuses on the intersection of stress, emotion, and health. I investigate how individual differences in emotion and emotion regulation affect health outcomes related to stress. I examine these processes across several different stress contexts in diverse populations, including children, adults with chronic illnesses, and individuals of minority backgrounds. My work focuses on physiological and behavioral health outcomes such as recovery from surgery, pain, autonomic nervous system activity, sleep, and dietary adherence. Working both in the lab and natural environments, I use a multi-method, theoretically-driven approach to solve health problems related to stress by applying rigorous study designs and advanced data analytic techniques, including multilevel modeling, spline growth curve modeling, survival analysis, and nonlinear methods. A unique aspect of my work is the development of methods to accurately measure the emotion and health outcomes that emerge during the stress process.

**EDUCATION**

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- |      |  |
|------|--|
| 2017 | Ph.D., University of California, Irvine (UCI)<br><i>Major:</i> Health Psychology<br><i>Minor:</i> Quantitative Methods |
| 2013 | M.S., Chapman University<br>Graduated <i>Summa Cum Laude</i><br><i>Major:</i> Health and Strategic Communication       |
| 2012 | M.A., California State University, Fullerton<br>Graduated <i>Summa Cum Laude</i><br><i>Major:</i> Psychology           |
| 2010 | B.A., California State University, Fullerton<br>Graduated <i>Summa Cum Laude</i><br><i>Major:</i> Psychology           |

**ACADEMIC POSITIONS**

---

- |            |  |
|------------|--|
| 2012- 2013 | Adjunct Faculty Member<br>Department: Psychology, California State University, Fullerton<br>Taught undergraduate courses in Elementary Statistics. |
|------------|--|

## AWARDS AND RESEARCH GRANTS

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2016	<i>Newkirk Grant Fellowship</i>	<b>[\$8,000]</b>
	Grant awarded to support collaborative community research. Awarded for research that engages the community with scientific knowledge.	
2016	<i>Mount San Antonio College NSF STEM Teacher Preparation Program</i>	<b>[\$4,000]</b>
	Mentor for community college student gaining experience in science and who will become a K-12 teacher. Funds awarded to student (\$3,000) and mentor (\$1,000).	
2016	<i>Paul and Frances Dickman Graduate Student Research Award</i>	<b>[\$1,500]</b>
	Awarded for excellence in research that benefits the local community.	
2016	<i>Graduate Student Travel Grant</i>	<b>[\$400]</b>
2015, 2016,	<i>Graduate Student Mentoring Award</i>	<b>[\$200]</b>
2017	Received each year for excellence in mentoring undergraduate students.	
2015	<i>NIH Ruth L. Kirschstein National Research Service Award (NRSA)</i>	<b>[\$109,296]</b>
	Individual Predoctoral Fellowship; Percentile: 3%; Impact Score: 14 Eunice Kennedy Shriver National Institute of Child Health and Human Development (NICHD) grant number: HD085712	
2015	<i>Multidisciplinary Design Project</i>	<b>[\$3,000]</b>
	Interdisciplinary research grant awarded for collaboration between School of Medicine and School of Social Ecology. Received funding to conduct research in the hospital setting and mentor undergraduate students in the research process.	
2015	<i>Social Ecology Alumni Fellowship Award</i>	<b>[\$1,500]</b>
	Awarded to one graduate student each year for excellence in research.	
2015	<i>Society for Personality and Social Psychology (SPSP) Travel Award</i>	<b>[\$500]</b>
2014	<i>Graduate Student Travel Grant</i>	<b>[\$400]</b>
2012	<i>Scholarly Creative Research Grant</i>	<b>[\$550]</b>
2008	<i>Student of Distinction for Outstanding Academic Achievement</i>	<b>[\$100]</b>
2008	<i>Faculty Association Academic Scholarship</i>	<b>[\$1,000]</b>
2008	<i>Faculty Association Extra Curricular Scholarship</i>	<b>[\$1,000]</b>
2008	<i>Harriet Genung Music Scholarship</i>	<b>[\$500]</b>
2008	<i>Carter Doran Memorial Scholarship for Academic Excellence</i>	<b>[\$500]</b>
2008	<i>Roberta L. Meek Memorial Scholarship for Academic Excellence</i>	<b>[\$1,000]</b>
2008	<i>Covina Women's Club Performing Arts Scholarship</i>	<b>[\$500]</b>

## PEER-REVIEWED PUBLICATIONS

---

- Pressman, S. D., **Jenkins, B. N.**, Kraft, T. L., Rasmussen, H., & Scheier, M. (in press). The whole is not the sum of its parts: Specific types of positive affect influence sleep differentially. *Emotion*.
- Jenkins, B. N.**, Rook, K. S., Borges-Garcia, R., Franks, M. M., & Stephens, M. A. P. (2016). Too much of a good thing? Overexertion of self-control and dietary adherence in patients with diabetes. *British Journal of Health Psychology*, *21*, 648-659.
- Min, C. B., Kain, Z. N., Stevenson, R. S., **Jenkins, B. N.**, & Fortier, M. A. (2016). A randomized trial examining preoperative sedative medication and post-operative sleep in children. *Journal of Clinical Anesthesia*, *30*, 15-20.
- Brown, R., Fortier, M. A., Zolghadr, S., Gulur, P., **Jenkins, B. N.**, & Kain, Z., (2016). Postoperative pain management in children of Hispanic origin: A descriptive cohort study. *Anesthesia & Analgesia*, *122*, 497-502.
- Jenkins, B. N.**, Kain, Z. K., Kaplan, S. H., Stevenson, R. S., Mayes, L. C., Guadarrama, J., & Fortier, M. A. (2015). Revisiting a measure of child postoperative recovery: Development of the Post Hospitalization Behavior Questionnaire for Ambulatory Surgery (PHBQ-AS). *Pediatric Anesthesia*, *25*, 738-745.
- Jenkins, B. N.**, Vincent, N., & Fortier, M. A. (2015). Differences in referral and use of complementary and alternative medicine between pediatric providers and patients. *Complementary Therapies in Medicine*, *23*, 462-468.
- Frederick, D. A., & **Jenkins, B. N.** (2015). Height and body mass on the mating market: Associations with number of sex partners and extra-pair sex among heterosexual men and women aged 18-65. *Evolutionary Psychology*, *13*, 1474704915604563.
- Jenkins, B. N.**, Fortier, M. A., Kaplan, S. H., Mayes, L. C., & Kain, Z. N. (2014). Development of a short version of the Modified Yale Preoperative Anxiety Scale. *Anesthesia & Analgesia*, *119*, 643-650.
- Jenkins, B. N.**, & Fortier, M. A. (2014). Developmental and cultural perspectives on children's postoperative pain management at home. *Pain Management*, *4*, 407-412.
- Jenkins (Gentle), B. N.**, Pillsworth, E. G., & Goetz, A. T. (2014). Changes in sleep time and sleep quality across the ovulatory cycle as a function of fertility and partner attractiveness. *PloS One*, *9*, e92796.
- Jenkins (Gentle), B. N.**, & Goetz, A. T. (2010). Functionality, parsimony, discovery, avoiding hamartia: How evolutionary perspectives are changing psychology. *EvoS Journal: The Journal of the Evolutionary Studies Consortium*, *2*, 24-31.

## BOOK CHAPTERS

---

- Jenkins, B. N.**, Acevedo, A., & Pressman, S. D. (in press). Positive affect in the context of health. *Wiley Encyclopedia of Health Psychology*.
- Jenkins, B. N.**, Fortier, M. A., & Kain, Z. N. (2014). Parent management of children's postoperative pain. In *Advances in Postoperative Pain Management. Current issues in postoperative pain* (pp. 58-67). London: Future Science Group.
- Frederick, D. A., Forbes, M., **Jenkins, B. N.**, Reynolds, T. A., & Walters, T. (2013). Beauty standards from an anthropological perspective. In P. Whelehan & A. Bolin (Eds.), *Encyclopedia of human sexuality* (pp. 1-5). New York: Wiley-Blackwell.

## MANUSCRIPTS UNDER REVIEW

---

*\*Denotes an undergraduate or medical student I directly supervised in research*

- Jenkins, B. N.**, Granger, D. A., Roemer, R., Martinez, A., \*Torres, T., & Fortier, M. A. (under review). Emotion regulation and positive affect in the context of salivary alpha amylase response to pain in children with cancer.
- Jenkins, B. N.**, Roemer, R., Martinez, A., \*Torres, T. & Fortier, M. A. (under review). Cold pressor pain response in children with cancer.
- Jenkins, B. N.**, Hunter, J. F., Cross, M. P., Acevedo, A. M., & Pressman, S. D. (under review). When is affect variability bad for health? The association between affect variability and immune response to the influenza vaccination.
- Jenkins, B. N.**, Fortier, M. A., Stevenson, R., Hayakaway, J., \*Makhlouf, M., \*Lim, P., \*Converse, R., & Kain, Z. N. (under review). The development, formative evaluation, and efficacy testing on an intervention to reduce child postoperative pain: The Nurse-Parent Intervention for the Management of Postoperative Pain (NP-IPP).

## MANUSCRIPTS IN PREPARATION

---

*\*Denotes an undergraduate or medical student I directly supervised in research*

- Jenkins, B. N.**, Hunter, J. F., Richardson, M. J., Tamlin, C., & Pressman, S. D. (in preparation). Affect variability and predictability: Using recurrence quantification analysis to better understand how the dynamics of affect relate to health.
- \*Becerra, M., **Jenkins, B. N.**, \*Wong, E., & Pressman, S. D. (in preparation). The connections between positive emotion and health within a faculty advisor-graduate student relationship: Does a good advisor a day keep the doctor away?
- \*Kaminsky, O., Fortier, M. A., **Jenkins, B. N.**, Golianu, B., Gold, J., Zuk, J., Stevenson, R. S., Kaplan, S. H., & Kain, Z. N. (in preparation). It's all in the eyes of the observer: Parent versus child report of child postoperative pain.

## CONFERENCE PRESENTATIONS

---

*\*Denotes an undergraduate or medical student I directly supervised in research*

\*Calderon Marcos, A. J., \*Makhlouf, M. M., **Jenkins B. N.**, Fortier, M. A., & Kain, Z. (2017, May) *Nurse-Parent Intervention for the Management of Postoperative Pain*. Poster presented at the 29<sup>th</sup> Association for Psychological Science Annual Convention, Boston, Massachusetts.

**Jenkins, B. N.**, Pressman, S. D., Kraft, T. L., Rasmussen, H., & Scheier, F. (2016, June). *Do all types of positive emotion function the same? How vigor, well-being, and calm differentially impact sleep depending on the stress context*. Poster presented at the 28<sup>th</sup> Annual Meeting of the Human Behavior and Evolution Society, Vancouver, CA.

\*Makhlouf, M., \*Zaidi, R., \*Batista, Y., **Jenkins, B. N.**, & Fortier, M. A. (2016, May). *Nurse and Parent Training in Postoperative Stress*. Talk presented at the Multidisciplinary Design Symposium, University of California-Irvine, CA.

\*Becerra, M., **Jenkins, B. N.**, & Pressman, S. D. (2016, May). *The connections between positive emotion and health within a faculty advisor-graduate student relationship: Does a good advisor a day keep the doctor away?* Talk presented at the 23<sup>rd</sup> Annual UCI Undergraduate Research Symposium, University of California-Irvine, CA.

\*Converse, R., \*Acosta, D., \*Zuniga, C., **Jenkins, B. N.**, & Fortier, M. A. (2016, May). *Predictors of adult behaviors that impact child postoperative pain*. Talk presented at the 23<sup>rd</sup> Annual UCI Undergraduate Research Symposium, University of California-Irvine, CA.

\*Rigual, N. L., \*Acosta, E., \*Converse, R., **Jenkins, B. N.**, & Fortier, M. A. (2016, May). *Child postoperative pain and social network size within the home*. Poster presented at the 23<sup>rd</sup> Annual UCI Undergraduate Research Symposium, University of California-Irvine, CA.

\*Lim, P., **Jenkins, B. N.**, & Fortier, M. A. (2016, May). *Nurse and parent intervention for postoperative stress*. Talk presented at the 23<sup>rd</sup> Annual UCI Undergraduate Research Symposium, University of California-Irvine, CA.

\*Lim, P., **Jenkins B. N.**, & Fortier, M. A. (2016, April) *Complementary and alternative medicine use among parents of children with cancer*. Poster presented at the 96<sup>th</sup> Annual Meeting of the Western Psychological Association, Long Beach, CA.

**Jenkins, B. N.**, Roemer, R., & Fortier, M. A. (2016, March). *Emotion regulation and positive affect in the context of pain: Resiliency for pediatric cancer patients*. Talk presented at the 3<sup>rd</sup> Annual Meeting of the Society for Affective Science, Chicago, IL.

**Jenkins, B. N.**, Pressman, S. D., Kraft, T. L., Rasmussen, H., & Scheier, M. F. (March, 2016). *Resiliency to poor sleep: Vigor, well-being, calm, and the stress context*. Poster presented at the 3<sup>rd</sup> Annual Meeting of the Society for Affective Science, Chicago, IL.

- Jenkins, B. N.,** Fortier, M. A., & Kain, Z. N. (2016, January). *Nurse and Parent Training in Postoperative Stress (NP-TIPS): Using adult-child interactions to decrease child postoperative pain*. Poster presented at the 6<sup>th</sup> Annual Meeting for the Society for Personality and Social Psychology Personality Health Network Preconference, San Diego, CA.
- Jenkins, B. N.,** \*King, K., \*Montemayor, P. L. N., Kraft, T. L., Rasmussen, H., Scheier, M. F., & Pressman, S. D. (January, 2016). *The whole is not the sum of its parts: Specific types of positive affect influence sleep differentially*. Poster presented at the 17<sup>th</sup> Annual Meeting for the Society for Personality and Social Psychology, San Diego, CA.
- \*Lim, P., **Jenkins, B. N.,** & Fortier, M. A. (2015, May). *Child and parental factors in complementary and alternative medicine use*. Poster presented at the 22<sup>nd</sup> Annual UCI Undergraduate Research Symposium, University of California-Irvine, CA.
- Mattison, G., \*Olaya, W., Yi, M., **Jenkins, B. N.,** Yakuby, N., Toranto, J., Muhonen, M., & Olaya, J. (2015, May). *Improved outcomes treating deformational plagiocephaly with helmet therapy*. Poster presented at the 65<sup>th</sup> Annual Meeting of the California Society of Plastic Surgeons, Monterey, CA.
- Jenkins, B. N.,** Rook, K. S., Stephens, M. A., Franks, M. (2015, February). *Dietary adherence in patients with Type 2 Diabetes: Applying the strength model of self-control*. Poster presented at the 16<sup>th</sup> Annual Meeting for the Society for Personality and Social Psychology, Long Beach, CA.
- Jenkins, B. N.,** Fortier, M. A., & Kain, Z. K. (2014, April). *The Modified Yale Preoperative Anxiety Scale (mYPAS): Increasing efficiency of administration in clinical settings*. Poster presented at the 35<sup>th</sup> Annual Meeting for the Society of Behavioral Medicine, Philadelphia, PA.
- Jenkins, B. N.,** \*Burkhead, C., \*Tang, S., Omri, G., & Pressman, S. D. (2014, March). *High oxytocin and attachment security prime predict differential parasympathetic responses to stressful events*. Poster presented at the 72<sup>nd</sup> Annual Meeting for the American Psychosomatic Society, San Francisco, CA.
- Jenkins, B. N.,** & Frederick, D. A. (2013, November). *Motivations for infidelity*. In Frederick, D. A. (Chair), *Jealousy, Infidelity, and Fidelity: Who, What, Why, and When?* Symposium conducted at the 54<sup>th</sup> Annual Meeting of the Society for the Scientific Study of Sexuality Conference, San Diego, CA.
- Frederick, D. A., **Jenkins, B. N.,** Oliver, T. (2013, May). *Perceptions of the mating environment as predictors of infidelity*. Poster presented at the 7<sup>th</sup> Annual 3UC+ Conference on Evolutionary Social Science, San Luis Obispo, CA.
- Jenkins, B. N.,** & Frederick, D. A. (2013, January). *Height and body mass as predictors of sexual behavior*. Poster presented at the 14<sup>th</sup> Annual Meeting of the Society for Personality and Social Psychology Conference, New Orleans, LA.

- Jenkins, B. N.,** Goetz, A. T., Beals, K. P., Pillsworth, E. G., & Tessler, J. (2012, June). *The case of the female orgasm function: Indicating good bonds and retaining sperm*. Talk presented at the 24<sup>th</sup> Annual Meeting of the Human Behavior and Evolution Society, Albuquerque, NM.
- Jenkins, B. N.,** Pillsworth, E. G., & Goetz, A. T. (2012, April). *Now I lay me down to sleep: The attractiveness of a woman's partner predicts whether she sleeps more or less at high fertility*. In **Jenkins, B. N. (Chair)**, *Ovulatory research in 2011: New findings in women's behavioral responses to fertility and the hormonal mechanisms that control them*. Symposium conducted at the 92<sup>nd</sup> Annual Meeting of the Western Psychological Association, San Francisco, CA.
- Jenkins, B. N.,** Goetz, A. T., Beals, K. P., Pillsworth, E. G., & Tessler, J. (2012, April). *The case of the female orgasm function: Indicating good bonds and retaining sperm*. Talk presented at the 92<sup>nd</sup> Annual Meeting of the Western Psychological Association, San Francisco, CA.
- Jenkins, B. N.,** & Goetz, A. T. (2012, April). *Accounting for the disparate psychological effects of red*. Poster presented at the 92<sup>nd</sup> Annual Meeting of the Western Psychological Association, San Francisco, CA.
- Jenkins, B. N.,** & Beals, K. P. (2012, April). *Too much media: Eliciting contrast effects in the lab*. Poster presented at the 92<sup>nd</sup> Annual Meeting of the Western Psychological Association, San Francisco, CA.
- Jenkins, B. N.,** Goetz, A. T., Beals, K. P., Pillsworth, E. G., & Tessler, J. (2012, January). *The case of the female orgasm function: Indicating good bonds and retaining sperm*. Poster presented at the 10<sup>th</sup> Annual Meeting of the Society for Personality and Social Psychology Evolutionary Preconference, San Diego, CA.
- Jenkins, B. N.,** Pillsworth, E. G., & Goetz, A. T. (2011, May). *Now I lay me down to sleep: The attractiveness of a woman's partner predicts whether she sleeps more or less at high fertility*. Talk presented at the 5<sup>th</sup> Annual Workshop on Evolutionary Perspectives of Human Behavior, San Luis Obispo, CA.
- Jenkins, B. N.,** Pillsworth, E. G., & Goetz, A. T. (2011, May). *Now I lay me down to sleep: The attractiveness of a woman's partner predicts whether she sleeps more or less at high fertility*. Poster presented at the 91<sup>st</sup> Annual Meeting of the Western Psychological Association Convention, Los Angeles, CA.
- Jenkins, B. N.,** & Beals, K. P. (2011, May). *Contrast effects and viewing attractive models: Do warning labels help?* Poster presented at the 91<sup>st</sup> Annual Meeting of the Western Psychological Association Convention, Los Angeles, CA.
- Jenkins, B. N.,** & Beals, K. P. (2011, March). *Contrast effects and viewing attractive models: Do warning labels help?* Talk presented at the California State University, Fullerton Graduate Forum: Diversity and Graduate Education, Fullerton, CA.

## COURSES TAUGHT

---

2012- 2013 Adjunct Faculty Member  
Department: Psychology, California State University, Fullerton  
Course: **Elementary Statistics**; see table for student evaluations

Date	Class Size	Median Rating	Mean Rating
Spring 2013	25	4.0/4.0	3.7/4.0
Spring 2013	25	4.0/4.0	3.6/4.0
Spring 2013	26	4.0/4.0	3.6/4.0
Spring 2013	26	4.0/4.0	3.5/4.0
Fall 2012	22	4.0/4.0	3.7/4.0
Fall 2012	24	4.0/4.0	3.6/4.0
Spring 2012	21	4.0/4.0	3.6/4.0
Spring 2012	21	4.0/4.0	3.6/4.0

2014-2015 Teaching Assistant (University of California, Irvine):  
*Health Psychology*  
*Environmental Psychology*  
*Psychology Fundamentals*

2011-2012 Teaching Assistant (California State University, Fullerton):  
*Advanced Statistics*  
*Research Methods*  
*Social Psychology*

## PREFERRED TEACHING COURSES

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- Statistics for the Behavioral Sciences
- Research Methods and Laboratory
- Child Development
- Life Span Development
- Physiological Psychology
- Motivation and Emotion
- Health Psychology
- Health and Well-Being
- Children and Trauma
- Structural Equation Modeling (SEM)
- Longitudinal Data Analysis
- Hierarchical Linear Modeling (HLM)
- Survival Analysis
- Bayesian Analysis

## STATISTICS/MATH AND METHODS COURSES COMPLETED

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- Bayesian Analysis (2 courses)
- Structural Equation Modeling (SEM)
- Longitudinal Data Analysis
- Hierarchical Linear Modeling (HLM)
- Multivariate Statistics
- Advanced Statistics
- Computer Applications in Psychology
- Advanced Social Sciences Computer Applications
- Psychophysiology
- Psychobiology of Stress
- Calculus and Analytic Geometry (3 semesters)
- Linear Algebra & Differential Equations
- Computer Programming (C++; VB)



## MENTORSHIP

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I have worked closely with several undergraduate students at the University of California, Irvine. The following are the students I have worked with, the awards they won under my mentorship, and their current position if graduated. (§current mentee; †honors students)

Emily Wong <sup>§</sup>	
Summer Undergraduate Research Opportunity Award (Summer 2016)	<b>[\$1,600]</b>
Robert Twidwell <sup>§</sup>	
Summer Undergraduate Research Opportunity Award (Summer 2016)	<b>[\$1,600]</b>
Anarosa Calderon Marcos <sup>§</sup>	
Undergraduate Research Opportunities Program Award (Fall 2016)	<b>[\$700]</b>
Summer Undergraduate Research Opportunity Award (Summer 2016)	<b>[\$1,500]</b>
Mai Makhoul <sup>§</sup>	
Undergraduate Research Opportunities Program Award (Fall 2016)	<b>[\$700]</b>
Summer Undergraduate Research Opportunity Award (Summer 2016)	<b>[\$1,500]</b>
Undergraduate Research Opportunities Program Award (Spring 2016)	<b>[\$500]</b>
Multidisciplinary Design Project Research Fellow (Winter 2016)	
Remy Converse	
<i>Current position:</i> Master's student	
Undergraduate Research Opportunities Award (Fall 2015)	<b>[\$800]</b>
Monica Becerra <sup>†</sup>	
<i>Current position:</i> Ph.D. student	
Outstanding Contribution to the School of Social Ecology Award (Spring 2016)	
Excellence in Research Award (Spring 2016)	
Summer Undergraduate Research Opportunity Award (Summer 2015)	<b>[\$1,700]</b>
Undergraduate Research Opportunities Program Award (Fall 2015)	<b>[\$900]</b>
Paulina Lim	
<i>Current position:</i> Behavior Therapist	
Summer Undergraduate Research Opportunity Award (Summer 2015)	<b>[\$1,200]</b>
Undergraduate Research Opportunities Program Award (Fall 2014)	<b>[\$300]</b>

## PROFESSIONAL AFFILIATIONS

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American Psychological Association  
American Psychological Association Division 38: Health Psychology  
Human Behavior and Evolution Society  
Society for Personality and Social Psychology  
Psi Chi, International Honor Society in Psychology  
Phi Theta Kappa National Honor Society

## ADDITIONAL TRAINING

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- 2017 *Course Design Certificate Program*  
Four-week workshop through the UCI Center for Engaged Instruction on developing course materials and ways to engage students in active learning in the classroom.
- 2016 *University of California Health Consortium Workshop*  
Two-day workshop covered grant funding, mentorship practices, running a lab, and faculty success training. Workshop funded by a grant used to encourage collaborations between health psychologists in the UC system.
- 2016 *Latent Class and Latent Transition Analysis*  
Weeklong course offered at Dr. Todd Little's Statistics Camp. Covered theory and methods for latent class and latent transition analysis in Mplus.
- 2015 *University of California Health Consortium Workshop*  
Two-day workshop covered missing data, growth curve modeling, meta-analysis, and biological assessment. Workshop funded by a grant used to encourage collaborations between health psychologists in the UC system.
- 2015 *Nonlinear Methods for Psychological Science*  
Weeklong course offered through the APA Advanced Training Institute. Covered recurrence quantification analysis, cross recurrence quantification analysis, fractal time series, and spectral analysis.
- 2015 *Structural Equation Modeling (SEM) for Longitudinal Data Analysis*  
Weeklong course offered through the APA Advanced Training Institute. In an SEM framework, this course covered latent growth curve modeling, latent variable factor analysis, and nonlinear models. Software used was R and Mplus.
- 2015 *Data Mining*  
Weeklong course offered through the APA Advanced Training Institute. Covered confirmatory and exploratory factor and cluster analysis, factor mixture modeling as an exploratory device, classification and regression trees, random forests, and artificial neural networking. Software used was R and Mplus.
- 2014 *Mindware Technologies Heart Rate Variability and Impedance Cardiography*  
Two-day psychophysiology workshop on how to collect and analyze physiology data.

## PROFESSIONAL SERVICE: AD HOC REVIEWER

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*International Journal of Psychology*

*PlosOne*

*Emotion*

*Anesthesia and Analgesia*

*Psychological Assessment*

*Pediatric Anesthesia*

*Evolutionary Studies Consortium*

## PROFESSIONAL SERVICE: POSITIONS

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- 2016-present *Graduate Student Mentor*  
Peer-mentor of a 1<sup>st</sup> year graduate student in the Department of Psychology and Social Behavior. Help guide mentee through first year of graduate school.
- 2014-present *Ratio Christi Club President*  
Organize club meetings and events. Prepare material for discussions.
- 2015-2017 *Graduate Student Grants Information Coordinator*  
Organize grant workshops and grant information for fellow graduate students.
- 2016 *Salivary Bioscience Faculty Search Committee Member*
- 2014-2016 *Statistical Brown Bag Coordinator*  
Organize department statistics workshops each quarter for students.
- 2013 *Statistics book reviewer*
- 2013 *Member of the Society of Pediatric Psychology Pain Special Interest Group Measurement Assessment Project*  
Evaluated pediatric pain assessment measures to determine which measures should be used in research.
- 2009 *Human Behavior and Evolution Society Conference Assistant*  
Assisted with minor organizational tasks of conference.

## **PROFESSIONAL SERVICE: WORKSHOPS AND LECTURES**

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- 2017 *Speaker for the Osher Lifelong Learning Institute*  
Presentation title: Why Emotions are Important for Health  
Presented research to older adults and members of the local community.
- 2017 *Presenter for F31 Workshop for Department of Psychology and Social Behavior*
- 2016 *Tech Trek Summer Science Camp Host*  
Worked with middle school girls to promote entrance to STEM fields by having them participate in research activities in the lab.
- 2016 *Presenter for Statistics Workshop for the Novaco Lab*
- 2016 *Panel Speaker for Graduate Student Panel for Transfer Student Center*
- 2016 *Presenter for F31 Workshop for Research Design Seminar*
- 2015 *Presenter for F31 Workshop for Department of Psychology and Social Behavior*
- 2014, 2015 *Guest Lecturer for Health Psychology*
- 2012 *Panel Speaker for Psychology Day, California State University, Fullerton*  
Topic: What to do with a Psychology Degree
- 2011 *Panel Speaker for Psi Chi Graduate School Preparation Event*  
Topic: What to do after Graduation and How to get into Graduate School

## **MEDIA COVERAGE**

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- The Wall Street Journal*, “Dietary overkill”, by Ann Lukits, 2 May, 2016  
<http://www.wsj.com/articles/can-boys-beat-girls-in-reading-1462202491>
- W Radio*, “Relationships and health behaviors”, 27 May, 2016
- Anesthesia & Analgesia Educational Supplement* “Development of the mYPAS”, 9 September, 2014

## **COMPUTER SKILLS**

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- Statistical Packages: SPSS, R, Stata, Mplus, MatLab  
Online survey creation: Qualtrics, SurveyMonkey

## **ABSTRACT OF THE DISSERTATION**

Affect Variability is Constantly Important: Implications for Health

Brooke N. Jenkins

Doctor of Philosophy in Psychology and Social Behavior

University of California, Irvine, 2017

Sarah D. Pressman, Ph.D., Chair

Positive and negative affect has been associated with numerous health factors. However, what is commonly investigated are the *mean* levels of affect. While means reveal important information, how affect varies over time may provide further information about how the experience of affect relates to important outcomes. This change from moment to moment, day to day, or week to week has been referred to as affect variability and is often operationally defined as the standard deviation of affect over time. While useful, this methodology is incomplete as it loses important information about the temporal aspect of affect variability (i.e., information about patterns of affect responses over time are lost). Nevertheless, greater affect variability has been associated with several psychological health and health-relevant outcomes such as low self-esteem and higher depressive symptoms. What remains unclear is how affect variability patterns and interactions with mean levels relate to such variables. What is also unknown is whether affect variability is associated with markers of physical health. Therefore, the goals of this dissertation are to 1. assess whether affect variability is associated with markers of physical health, 2. examine a new method for assessing affect variability that may overcome the downsides of standard deviation (i.e., loss of information about patterns), and 3. use this new method to better understand how affect variability patterns relate to mental and physical health and health-relevant variables.

## **CHAPTER 1:**

### **Introduction**

## Introduction

Positive and negative affect has been shown to be associated with numerous psychological and physical health outcomes. Positive affect (PA), such as feelings of joy or happiness, has been tied to living longer (Chida & Steptoe, 2008), having better social relationships (Diener & Seligman, 2002), and having fewer health complications (Ostir, Markides, Peek, & Goodwin, 2001) while greater amounts of negative affect (NA), such as feelings of sadness or anger, have been associated with engagement in poor health behaviors (Brummett et al., 2006; Ellis, Orom, Giovino, & Kiviniemi, 2015), greater depressive symptoms (de Carvalho et al., 2014), and worse physical health (Oliveira & Costa, 2013). This research, and by far the majority of affect research in general, examines *mean* levels of affect (i.e., momentary affect; Larsen & Diener, 1985) and does not capture the naturally occurring *changes* in affect over time, possibly missing important components of the affective experience.

These changes in intensity of affect over time are referred to as *affect variability* and are often not considered in affect research. For example, although mean NA is tied to worse physical health (Oliveira & Costa, 2013), little is known about how more or less *variability* in NA (irrespective of or possibly interacting with mean NA) influences health. This variability may be important as an individual who varies between extreme lows and extreme highs on NA is very different from an individual with constant moderate levels of NA but may have the same overall *mean* level (see Figure 1). This difference may matter because larger fluctuations in affect may have health consequences. Furthermore, the pattern of this affect variability can also tell us important information. For example, an individual who “jumps around” on NA more erratically has a very different pattern compared to someone who has consistent or predictable values of NA (see Figure 2). Taken together, affect variability and the patterning of this variability, while also

considering mean levels of affect, may allow researchers to understand the full affective experience over time.

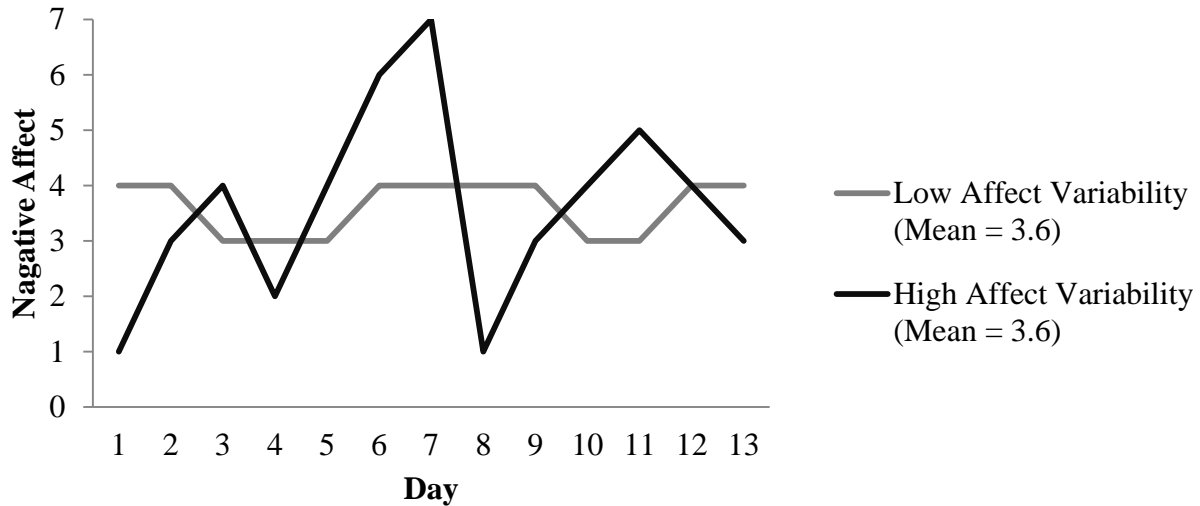


Figure 1. Two individuals with the same mean level of negative affect but different negative affect variability.

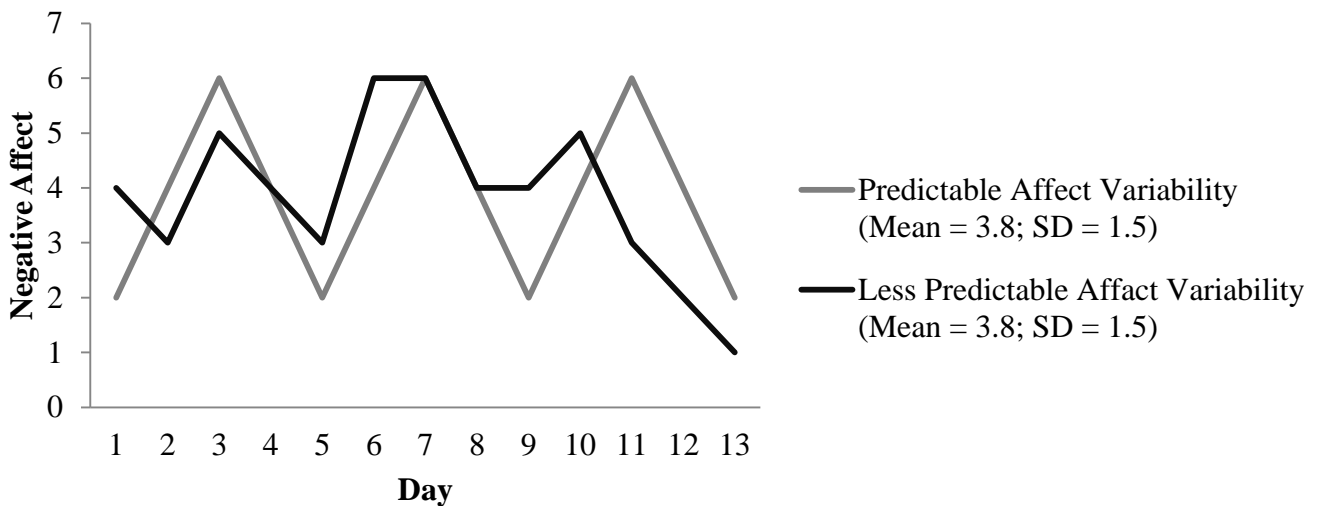


Figure 2. Two individuals with different patterns of affect variability.

Although affect variability is common (e.g., Röcke, Li, & Smith, 2009) and may reflect important information above and beyond mean levels, several important questions remain: What are the best ways to measure affect variability? What are the differences between PA and NA variability? Does affect variability influence physical health? Are there important patterns within



affect variability that have health implications? How does affect variability interact with mean levels to influence health?

The goals of this dissertation are to summarize the current health and affect variability literature (Chapter 2), explore answers to such questions as posed above (Chapters 3 and 4), and describe implications of this dissertation research and future directions in the field of health and affect variability (Chapter 5). In Chapter 2, I first review the affect variability and health literature by detailing measurement issues within affect variability while outlining the strengths and weakness of each approach. Second, I introduce a previously unused method for assessing affect variability that can quantify information about the patterns of affect variability. Third, I briefly review the literature on affect variability as it relates to health and health-relevant outcomes. In this brief review, I identify gaps in the literature that are then addressed in Chapters 3 and 4.

Chapters 3 and 4 present findings from empirical studies that build upon the literature and methods described in Chapter 2 by addressing limitations in the field of health and affect variability. In Chapter 3, affect variability and its interaction with mean affect level is used to predict antibody response to a flu vaccine, a marker of immune system health (Glaser, Kiecolt-Glaser, Speicher, & Holliday, 1985). Chapter 3 adds to the affect variability literature by examining how variability plays a role in a health-relevant physiological function measure and at different levels of mean affect.

In Chapter 4, I present two studies that help to assess patterning of affect variability as it relates to health. Study 1 is a simulation study of a new method, recurrence quantification analysis (RQA), for assessing affect variability patterns. In this simulation, I show that RQA adds important information above and beyond what the typically used method for assessing

affect variability (namely, standard deviation) provides. In Study 2, I incorporate RQA to determine the relationship between patterns of affect variability and mental (depressive symptoms and psychological well-being) and physical (somatic symptoms) health and health-relevant outcomes. Study 2 demonstrates that standard deviation, RQA, and mean affect levels interact in their association with mental and physical health. This study adds to the literature by using a new measure of affect variability (i.e., RQA) that can capture predictability of affect variability.

Throughout Chapters 3 and 4, measures of mean affect level are controlled for to determine whether any found effects occur over and above the effects of mean levels. This is important to consider because it answers the question of whether it is just mean level that determines associations with health or whether it is the actual trajectory of affect over time which adds information.

Affect variability is common and can be assessed in a multitude of ways. Although affect variability has been shown to be associated with several psychological health variables, little is known about the patterning of affect over time and whether these patterns interact with mean levels in their association with both psychological *and* biological (i.e., reported health symptoms, immune function) variables. This dissertation adds to the literature by using new outcome variables, incorporating a new method for assessing patterning of affect variability, and demonstrating that this patterning adds information about the relationship between affect variability and health and health-relevant variables. Affect variability can capture more of the entirety of the affective experience compared to simply assessing mean levels of affect. The implications of this research, as well as future directions, are expanded upon in Chapter 5.

## References

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Hedonic level and emotional intensity. *Personality and Individual Differences*, 6, 631–636.

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Ostir, G. V, Markides, K. S., Peek, M. K., & Goodwin, J. S. (2001). The association between emotional well-being and the incidence of stroke in older adults. *Psychosomatic Medicine*, 63, 210–215.

Röcke, C., Li, S.-C., & Smith, J. (2009). Intraindividual variability in positive and negative affect over 45 days: Do older adults fluctuate less than young adults? *Psychology and Aging*, 24, 863–878. <http://doi.org/10.1037/a0016276>

## **CHAPTER 2:**

### **Methodological Issues of Affect Variability and a Brief Review of the Affect Variability and Health Literature**

## **Methodological Issues of Affect Variability and a Brief Review of the Affect Variability and Health Literature**

Before delving into new research on affect variability and health, it is important to understand issues in this field. First, there are many methods for calculating affect variability and these methods all have pros and cons associated with their use. Other methodological concerns exist as well. For example, variability may be different between positive affect (PA) and negative affect (NA) and these differences may have implications for health. Issues such as these are expanded upon in this chapter. Finally, a brief review of the affect variability and health literature is presented to express the limitations in this field.

### **Measuring Self-Reported State Affect**

Before we understand how to measure affect variability, it is necessary to touch on how state affect itself is measured. Most studies use adjective checklists such as the Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988) and the Profile of Mood States (POMS) Questionnaire (Curran, Andrykowski, & Studts, 1995; McNair, Lorr, & Droppleman, 1971) as self-report methods to assess state PA and NA. The state version of these measures is used to capture state affect (i.e., transient affect) as opposed to trait affect (i.e., stable, dispositional) because theoretically, there should be little movement in trait affect over time. These state measures ask participants to rate the extent to which they are feeling, at that moment, each of several affect adjectives (e.g., anxious, happy, angry) on scale responses usually ranging from “not at all” to “extremely.” PA related words (e.g., happy, cheerful, calm) are combined to form one PA score and the NA related words (e.g., angry, sad, nervous) are similarly combined to form one NA score. Assessing affect is complex and it can be assessed in

a multitude of other ways (e.g., facial expression [Abel & Kruger, 2010], word coding [Pressman & Cohen, 2012]). However, for the purposes of this dissertation, I focus on self-reported affect.

### **Calculating Affect Variability**

To assess affect variability, these state measures are used at multiple time points. Affect variability, therefore, is simply the changing of the levels of intensity of state affect over time. This construct can be calculated in several ways and understanding methods of measuring it is necessary when investigating this construct. Therefore, the goal of this section is to expand upon the number of ways affect variability has been calculated and the strengths and weakness of each approach (see Table 1).

#### **One-Time Assessment Measures**

Although current research on affect variability assesses affect at multiple time points, early research on affect variability simply asked people in a one-time assessment how much their affect changes over time. For example, the Affective Lability Scale (see Table 1) has participants rate items such as “One minute I can be feeling OK, and then the next minute I’m tense, jittery, and nervous” (Harvey, Greenberg, & Serper, 1989). Similarly, the reactivity subscale of the Mood Survey (Underwood & Froming, 1980) asks participants to rate items such as “Compared to my friends, I’m less up and down in my mood states” and “Sometimes my moods swing back and forth very rapidly.” Test-retest reliability as far as 7 weeks apart is strong suggesting that individuals are consistent in their reports and/or their experiences of affect variability.

Table 1

*Methods to Assess Affect Variability.*

<b>Method</b>	<b>Definition</b>	<b>Studies</b>	<b>Benefits</b>	<b>Downsides</b>
One-time assessment	Individuals self-report how variable their affect is	Harvey et al. (1989); Underwood & Froming (1980); Goodman et al. (2003); Eid et al. (1994); Skodol et al. (2002)	Only need to assess an individual at one time point; reflects a person's view of themselves	Sensitive to recall/judgement bias; only assesses affective change in general and does not assess variability within NA versus PA separately; <i>no consideration of temporal patterns</i>
Insufficient variation	Percentage of time a respondent endorses a specific rating on an affect scale item (over several ratings of the same scale) and reflects less affect variability as the percentage value increases	Röcke et al. (2009)		Does not take into consideration the amount of difference between affect ratings over time (only shows that there is a difference); <i>no consideration of temporal patterns</i>
Adjusted squared successive difference scores	Squared change score between consecutive ratings of affect (adjusted by dividing by the amount of time in between the two assessments and correcting for positive autocorrelation)	Trull et al. (2008)	Can look at change from one time point to the next independently	Only considers <i>temporal</i> differences from one time point to the next; no single overall measure of variability
Adjusted acute change/probability of acute change	Adjusted acute change – binary variable representing whether the change is above the 90 <sup>th</sup> percentile mark;	Trull et al. (2008); Jahng et al. (2008)	Identifies large changes	Only considers <i>temporal</i> differences from one time point to the next



	Probability of acute change – dividing total number of adjusted acute changes by total number of changes			
Absolute values of residuals	Residuals calculated by running a regression of the affect values on time and then taking the absolute values of the predicted minus actual affect values	Röcke et al. (2009)		No single overall measure of variability
Standard deviation/coefficient of variation	Standard deviation of an individual's scores on an affect measure over multiple time points (coefficient of variation divides the standard deviation by the mean)	Larsen & Diener (1987); Eid & Diener (1999); Gruber et al. (2013); Hardy & Segerstrom (2016)	Easy to interpret	<i>No consideration of temporal patterns</i>
Multi-level modeling techniques (ICC, testing assumption of equal error variances between groups)	ICC: the ratio of variability between individuals to total variability among time points; assumption of equal error variances: chi square is used to test the differences in log likelihood of multi-level models with equal error variances ( $\sigma_1^2 = \sigma_2^2$ ) and without equal error variances ( $\sigma_1^2 \neq \sigma_2^2$ )	Röcke et al. (2009); Trull et al. (2008)	Covariates can easily be added to the model	Only works for between group differences; <i>no consideration of temporal patterns</i>
Core affect	Pulse: standard deviation	Kuppens et al. (2007)		Data with affect grid is

variability (spin and pulse)	of the length of the lines on the core affect grid; spin: standard deviation of the angles on the core affect grid			more difficult to collect because of the need for a visual grid; <i>no consideration of temporal patterns</i>
Frequency distributions	Frequency distribution of affect is used to determine how dispersed affect ratings are. More dispersion indicates greater affect variability	Watson et al. (1984)		Can only assess between group differences; no test of significance; <i>no consideration of temporal patterns</i>
Repeated measures ANOVA	Time is used as the repeated measure to determine group differences in affect levels at each time point	Steptoe et al. (2011)		Can only look at group differences in patterns
Time series (spectral analysis, harmonic analysis)	Estimates the changes in affect using a set of sine and cosine waves which vary in period, amplitude, and phase	Larsen (1987); Eaton & Funder (2001); Ram & Gerstorff (2009)	<i>Takes into account temporal patterns</i>	Requires large number of time points; assumes stability of affect variability
Recurrence quantification analysis (RQA)	Non-linear method that counts how often affect returns to the same level		<i>Takes into account temporal patterns</i>	

However, measures such as these are extremely sensitive to recall bias (Trull et al., 2008). Although participants may feel as though they may have shifts in affect, their reports may not accurately reflect reality. Given that other methods such as ecological momentary assessment and daily diaries allow researchers to assess affect daily (or even multiple times within days), it is often best to measure affect over multiple time points and then calculate variability using quantitative methods such as those described below.

### **Insufficient Variation**

Insufficient variation is one quantitative method that can be used to examine affect variability when affect is measured at multiple time points. Insufficient variation is the percentage of time a respondent endorses a specific rating on an affect scale item (over several ratings of the same scale) and reflects less affect variability as the percentage value increases (Röcke et al., 2009; see Table 1). For example, Röcke and colleagues (2009) found that NA items had greater insufficient variation compared to PA items and that this was especially true among older, as compared to younger adults. This finding meant that older adults were more likely to use the same rating value on NA scales (e.g., always picking 8 on a 1 to 10 scale) over time relative to younger individuals. Although, this method allows for an assessment of changes in self-reported affect over time, it does not consider the magnitude of the change. For example, on a 1 to 10 scale, someone who rates their level of NA a 5 one day and then a 6 the next shows the same amount of variation as someone who rates NA as a 5 one day and a 10 the next. Further, this method loses information about the pattern of affect over time. For example, someone who rates their affect as 4, 5, and 6 over the course of three days receives the same insufficient variation value as someone who rates their affect as 4, 10, and 7 over three consecutive days even though these patterns of affect are very different. Not only are patterns not

considered but the temporal nature of the data is disregarded as well. For example, someone who rates their affect as 7, 8, and 9 over the course of three days would have the same insufficient variation value as someone who rates their affect as 9, 8, and 7.

### **Adjusted Squared Successive Differences**

One method that may overcome the lack of information about magnitude is to examine the difference between affect at successive time points (i.e., successive difference scores). To calculate successive differences, the difference between consecutive ratings of affect is calculated (i.e., change score; Trull et al., 2008). For example, if a participant rates their affect as a 5 one day and then a 7 the next, this participant's successive difference is 2. This change score is often squared resulting in a measure of only distance and not direction. Squared successive differences are useful when the time interval between ratings is the same across all measurement points because it results in a similar interpretation for each difference score. However, if the spacing between measurements is unequal, then it is central to adjust for time resulting in a final calculation for an *adjusted* squared successive difference score (see Table 1). This adjustment essentially divides by the amount of time in between the two assessments but then corrects for positive autocorrelation (see Trull et al., 2008 for the formula with this correction). Adjusted squared successive differences represent how much affect changes from one time point to the next (while giving more weight to larger differences) and is considered a measure of "instability."

One limitation of the calculation of adjusted squared successive differences is that it only examines the change from one time point to the next. When there are three or more time points, several adjusted squared successive differences must be calculated. This is limiting because it results in many measures of affect variability as opposed to one overall value. However, if

researchers are interested in examining these detailed changes (from one time point to the next), adjusted squared successive differences are quite useful. One extension of this metric is mean square successive differences (MSSD) which is the mean of the squared differences. This value gives an overall average of the change from one time point to the next (Jahng, Wood, & Trull, 2008)<sup>1</sup>.

### **Adjusted Acute Change/Probability of Acute Change**

Another extension of squared successive difference scores is adjusted acute change (Jahng et al., 2008; Trull et al., 2008). This measure is a binary variable that identifies whether a change from one time point to the next is above the 90<sup>th</sup> percentile mark of the entire sample's distribution of changes. This measure is calculated by first examining the frequency distribution of change scores for the entire sample and then identifying the 90<sup>th</sup> percentile mark. Then, any changes that are above this mark are identified with a 1 and all others are identified with a 0. As was the case with adjusted squared successive differences, an adjustment for time is used to account for the positive autocorrelation (see Trull et al., 2008 for the formula with this correction). Adjusted acute change can also be used to calculate probability of acute change. This calculation for probability of acute change is the total number of adjusted acute changes divided by the total number of changes to give a percentage score that represents the probability of an individual experiencing an acute change. However, the downside of this overall value is that it does not take into account the temporal patterning of affective changes.

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<sup>1</sup>Jahng and colleagues (2008) differentiate between affect variability and instability with variability represented by dispersion of affect scores and instability as low temporal dependency paired with high affect variability. For the purpose of this review, affect variability is a catch all term to refer to any changes in affect whether temporal considerations are made or not.

## **Absolute Values of Residuals**

Absolute values of residuals have been calculated by running a regression of the affect values on time (i.e., the independent variable is time point [1, 2, 3, etc.] and the dependent variable is the score on the affect measure). Then the residuals can be calculated based on the predicted values minus the expected values and taking the absolute value (Röcke et al., 2009). One limitation of this calculation is that it results in many measures of affect variability as opposed to one overall value (i.e., a residual is calculated at each time point).

## **Standard Deviation**

Standard deviation has been utilized to capture affect variability as a single value by calculating the standard deviation of an individual's scores on an affect measure over multiple time points (Eid & Diener, 1999; Gruber, Kogan, Quoidbach, & Mauss, 2013; Hardy & Segerstrom, 2016; Ram & Gerstorf, 2009; Röcke & Brose, 2013; see Table 1). The standard deviation approach estimates affect variability for each person allowing the standard deviation to be used as a predictor or outcome variable in subsequent models. An advantage of this approach is that standard deviation is widely used and understood (Röcke et al., 2009). Nevertheless, this single value assumes an independence of assessment such that an assessment at time,  $t$ , is not necessarily related to time,  $t + 1$  (Ram & Gerstorf, 2009). Essentially, standard deviation collapses temporal data across time leading to a loss of information about patterns. This results in information about the magnitude (average height) of the change in affect but provides no information about the frequency. The coefficient of variation in which standard deviation is divided by the mean has also been utilized as a relative standard deviation (e.g., Steptoe, Leigh, & Kumari, 2011) that takes into account mean level. However, the coefficient of variation still

suffers the same problem of losing temporal information and information about the patterning of data.

### **Multi-Level Modeling Techniques**

The previously described methods for assessing affect variability require a two-step approach in which first observations are used to calculate a metric of affect variability and then second this metric is used in models (e.g., regression, analysis of variance) to test its relationship with other variables. This two-step process ignores the fact that error created in the first step is unaccounted for in the second step. In other words, when the affect variability metric is used in further analyses, it is assumed to be an observation (i.e., treated as real data) as opposed to a calculation with error. This error is therefore unaccounted for leading to a deflation of the standard error which in turn increases the probability of a Type 1 error (Jahng et al., 2008). Furthermore, using these techniques does not account for different numbers of observations due to missing data. Multi-level modeling techniques may overcome these issues (see Table 1). One multi-level modeling technique that has been used is the intraclass correlation coefficient (ICC; Röcke et al., 2009). Multilevel models in which the level 2 variable is the individual and the level 1 variable is the time point (i.e., time points grouped within individuals), the ICC represents the ratio of variability between individuals to total variability among time points (i.e., total variability equals between person variability plus within person variability). The ICC coefficient represents the amount of *heterogeneity* between people which can also be thought of as the correlation among time points within individuals. In other words, this gives us a measure of how much of the variance is due to the individuals. For example, an ICC of 30% would indicate that 30% of the variance in affect is accounted for by the person level effect. This method is advantageous because it allows for other variables to be put into the same model to see whether

there is a significant amount of variance explained by the person level variable once other variables have been accounted for. However, the downsides of this approach are that it only estimates the ICC of the entire sample which does not allow for the examination of individual differences in this variance metric. Another disadvantage is that patterning of affect is lost because the ICC does not take into account which affect assessment comes first vs. second and so on.

One way researchers have been able to differentiate the variance estimated by the ICC has been to divide the estimates of the variance among groups. To do this, chi square is used to test the differences in log likelihood of multi-level models with the assumption of equal (homogenous) error variances ( $\sigma_1^2 = \sigma_2^2$ ) and without equal (heterogeneous) error variances ( $\sigma_1^2 \neq \sigma_2^2$ ) to see if there are group differences in variability of affect where  $e_{ti} \sim N(0, \sigma_k^2)$  (Trull et al., 2008). For example, Trull et al. (2008) performed a multi-level modeling analysis predicting daily affect with psychiatric group (borderline personality disorder vs. major depressive disorder) as a level 2 predictor and found that the heterogeneous error variance model was a better fit for the data compared to the homogenous error variance model. This implied that the variability in daily affect was significantly different between the two groups. Benefits of this method are that mean levels of affect (between the groups) and other covariates can be added to the model. However, this method is best for between group differences and not person to person differences.

### **Core Affect Variability (Spin and Pulse)**

Kuppens and colleagues (2007) measured core affect intensity and quality using the Affect Grid (Russell, Weiss, & Mendelsohn, 1989) in which participants rate their activation (rows) and valence (columns) on a 9 X 9 grid. Using these reports, they then calculated variability in core affect intensity and quality by first calculating line length and angles between



successive affect assessments. Line lengths represent how far on the affect grid an individual's affect report was from the center. Angles were the degree distance from one line to the next. Affect intensity was conceptualized as the standard deviation of the length of the lines (i.e., pulse) and affect quality was conceptualized as the standard deviation of the angles (i.e., spin; Kuppens et al., 2007) using a method developed by Moskowitz and Zuroff (2004, 2005; this method was originally used to measure variability in behavior). In other words, pulse was how much the line lengths varied over time and represented how the intensity of affect changed between time points. On the other hand, spin calculated as the standard deviation of the angles represented the variability among the qualitatively different affective states. They also looked at variability of valence (standard deviation of fluctuations between rows) and activation (standard deviation of fluctuations between columns) as well. Although these different variability measures (mean valence, mean activation, valence variability, activation variability, pulse, and spin) were somewhat correlated, spin, when entered into the model with the other measures, was the strongest predictor of higher neuroticism, pessimism, and depression and lower extraversion, optimism, and self-esteem. This indicated that it was the fluctuation between qualitatively different states (as opposed to only fluctuations on valence, activation, or intensity) that was the most prominent predictor of outcomes associated with poor adjustment.

Using core affect variability allows for multiple ways of assessing different types of affect variability and measures variability on a multi-dimension affect scale. Additionally, this method allows for the measurement of fluctuation between NA and PA (on the valence dimension) and arousal level as opposed to measuring variability of NA and PA separately. However, all of the calculations use standard deviation and so succumb to the same problem of losing information about the patterning of affect variability. For example, when looking at pulse,

the line lengths may be long, short, long, short respectively for four consecutive days for one person but then long, long, short, short for another and both individuals will have the same standard deviation for line length (i.e., the same pulse). Similarly, if someone's angles are 90°, 90°, 60°, and 60° over four consecutive days they will have the same standard deviation of angle size as someone with angles of 90°, 60°, 90°, and 60° over four consecutive days. What is interesting with spin is that it is not variability among qualitatively different states but variability of the *changes* between qualitatively different states. For example, if someone has angles of 180°, 180°, and 180° over three days, they will actually have low spin (i.e., a low standard deviation of angles). What might better represent the changes between qualitatively different states would be the mean of the angles. Individuals with high means would therefore be those with greater changes in qualitatively different states between consecutive time points.

### **Frequency Distributions**

Frequency distributions in which the affect ratings are simply graphed has been used as a method for assessing group differences in affect variability (Watson, Clark, & Tellegen, 1984). Groups with larger dispersion of affect ratings (i.e., frequency distributions that are more platykurtic) are considered to have more affect variability. This method for assessing variability is not ideal because it does not take into account patterning of affect over time and presents no significance tests of between group differences in variability.

### **Repeated Measures Analysis of Variance**

Repeated measures analysis of variance (ANOVA) has been used to examine how affect fluctuates over time (Steptoe et al., 2011). This method assesses whether there are group differences in affect values at each time point. Only being able to assess group differences is one

downside of this approach. Another downside is that no overall metric of affect variability is produced.

### **Time Series Analysis (Spectral Analysis/Harmonic Analysis/Autocorrelation)**

As we can see, many of the methods that are used to assess affect variability do not take into consideration the patterning of affect variability. One method that has taken patterns into consideration is time series analysis (Ram & Gerstorf, 2009; see Table 1) using, for example, spectral analysis (Larsen, 1987) or harmonic analysis (Eaton & Funder, 2001). These analyses estimate the changes in affect using a series of sine and cosine waves which vary in period, amplitude, and phase (see Eaton & Funder, 2001 p. 416 for an example equation). The sine and cosine waves represent changes in affect ranging from very slow (affect rarely changing) to very fast (affect changing often). Once a set of sine-cosine waves are fit to each individual's data, an  $R^2$  is calculated for each individual to represent how much of the variance in each person's data the set of periodic components can account for. If a model with fast sine and cosine waves is used, individuals with affect that changes often will have higher  $R^2$  values than will individuals with affect that changes less often. These  $R^2$  values can then be used in further analyses as either predictor or outcome variables. This method is beneficial because it gets at frequency of change with slow periodic waves representing fewer frequencies of changes in the data and fast periodic waves representing changes that occur more often. This is in contrast to the standard deviation approach which measures magnitude of change and provides no information on the rate of change. In essence, standard deviation provides magnitude (average extent) while time series can account for rate of change (i.e., period or how quickly something changes), amplitude, and phase. Downsides of the time series approach is that it requires a large number of time points and

that the variability is stable (Eid & Diener, 1999). For variability to be stable, this means that the same up and down pattern must repeat throughout the time points.

### **Recurrence Quantification Analysis**

Many of the commonly used methods for assessing affect variability (e.g., standard deviation, multilevel modeling) do not consider the temporal patterns of affect variability. It is possible that these patterns may provide additional insight into the affect process and help us uncover why affect variability is associated with health outcomes. Therefore, it is important to examine previously unused methods that may account for this patterning. Recurrence quantification analysis (RQA; Anderson, Bischof, Laidlaw, Risko, & Kingstone, 2013; Webber Jr & Marwan, 2015) is one method that has not been applied to assessing affect variability but may add relevant information to this field. This method can account for patterning of affect and may add additional information beyond previously used methods.

RQA is a non-linear method that assesses temporal sequences of change over time. Although, RQA has not been used in the context of affect variability, it has been successfully used to investigate other dynamic systems (e.g., eye gaze [Anderson et al., 2013], posture [Riley & Clark, 2003]). Because of the temporal nature of affect, RQA lends itself nicely to studying the processes of affect variability (Richardson, Dale, & Marsh, 2014). RQA provides measures such as regularity (i.e., how *often* does a person experience the same level of affect as before?) and average regularity (i.e., on average how long is the same level of affect experienced?). These RQA measures provide more information than just average distance from the mean (i.e., standard deviation) and may help to more precisely capture temporal systems. The benefit of RQA is that it quantifies the predictability of affect over time (see Table 1). Although less predictable but still

variable affect may be detrimental, stable (or more predictable) variability may not be as unfavorable.

### **Other Methodological Issues Associated with Affect Variability**

#### **Positive Versus Negative Affect in Affect Variability**

In addition to the consideration of how to measure affect variability, it is also useful to distinguish between positive versus negative (valence) affect variability. Specifically, individuals may not have the same amount of PA variability as they do NA variability. There is substantial evidence that PA and NA variability differ. For example, Eid and Diener (1999) and Steptoe and colleagues (2011) found that PA variability was larger than NA variability. Röcke and colleagues (2009) found a similar pattern that NA items had less variation compared to PA items and that this was especially true among older, as compared to younger, adults. Similarly, Watson, Clark, and Tellegen (1984) and Zevon and Tellegen (1982) found that when assessing PA and NA over 90 consecutive days that there was more variability in PA compared to NA. When examining the frequency distribution of each affect type, PA was flatter while NA was more peaked and positively skewed. This distribution reflects the relative variability of PA while also showing that high ratings of NA were rare. This research as a whole demonstrates consistency in the finding that PA is more variable than NA.

Although PA and NA variability are not the same, they may be correlated. The estimates of the correlation between variability of PA and NA in one study was 0.37 (time 1) and 0.39 (time 2; Hardy & Segerstrom, 2016). In contrast, the same study reported the correlation between mean PA and NA as -0.50 (time 1) and -0.66 (time 2) which suggests that mean levels may be more strongly correlated than are levels of variability. Additionally, the sign on these correlations demonstrates that while mean NA and PA are negatively correlated, variability of

these affects is positively correlated such that greater variability in NA is associated with greater variability in PA. Other studies report the same finding that PA and NA variability are positively correlated (Eid & Diener, 1999; Penner, Shiffman, Paty, & Fritzsche, 1994). Given that NA and PA variability are not perfectly correlated, it is important that we assess both to determine whether they have differential associations with other variables. Furthermore, it should be kept in mind that the relationship between NA and PA variability is not the same as the relationship between NA and PA mean levels.

### **Affect Variability as a Distinct and Stable Construct**

The affect variability literature suggests that affect variability is a distinct and stable construct from other measures of affect such as mean level of affect. For example, Trull et al. (2008) found that variances of PA, but not mean levels of PA, between individuals with borderline personality disorder and individuals with major depressive disorder were significantly different. This demonstrates that affect variability may be a distinct concept from mean level of affect. This is important because if affect variability provides no additional information beyond mean levels, then it is not beneficial to study changes in affect over time (i.e., affect variability). However, it is not surprising that affect variability could provide substantial information because the same mean level of affect could be associated with differing levels of variability.

Affect variability must also be considered a distinct construct from psychological flexibility, which is the *ability* to change affect in response to situational demands. This contrasts with affect variability, which is the actual *experience* of variability in affect over time and not the *capability* to vary in affect. Psychological flexibility has been measured in multiple ways. In laboratory settings, individuals who self-report affect and demonstrate facial expressions that more closely match the experimentally manipulated environmental context are deemed to have

greater psychological flexibility (Waugh, Thompson, & Gotlib, 2011). In studies assessing affect in naturalistic contexts through daily diary studies, psychological flexibility is defined as the extent to which self-reported affect matches the appropriate response to daily events (Hardy & Segerstrom, 2016). Psychological flexibility may be a predictor of health as individuals higher in psychological flexibility demonstrate greater trait resilience (Waugh et al., 2011) and have better physical and psychological health (Hardy & Segerstrom, 2016). Furthermore, psychological flexibility has been shown to be negatively correlated with affect variability (Hardy & Segerstrom, 2016) indicating that psychological flexibility is not the same as affect variability. Psychological flexibility (and not affect variability) allows for an adaptive response to changing contexts in the environment (Kashdan & Rottenberg, 2010; Waugh et al., 2011).

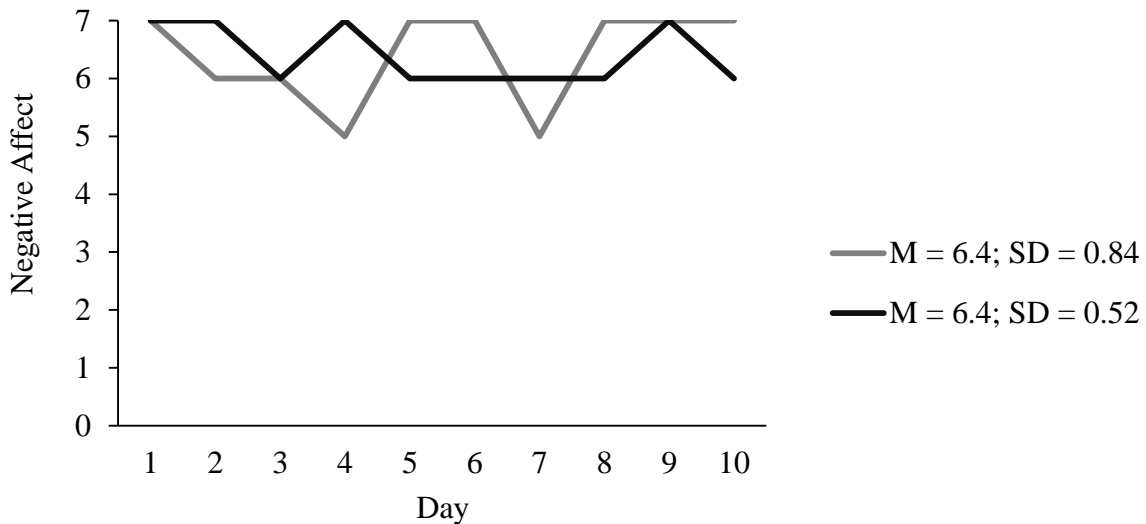
Delving more into methodology issues, some studies have demonstrated affect variability may be a somewhat stable construct across time (Eaton & Funder, 2001; Larsen, 1987; Penner et al., 1994). One study examined variability in the same sample across two time points with a seven to 13 year gap in between and found that the test-retest reliability for affect variability of NA and PA was  $r = 0.37$  and  $r = 0.19$ , respectively (Hardy & Segerstrom, 2016). Although more research is needed, it may be the case that NA variability is more stable than PA variability. Additionally, the test-retest reliability for PA is quite low suggesting PA variability is less stable.

### **Interactions Between Mean Level and Types of Variability**

None of the measures of variability discussed (apart from the coefficient of variation) consider mean levels of affect. This is important because variability may interact with mean level. Individuals may have high mean levels of PA or NA and have more or less fluctuation (i.e., variability) around these mean levels. For example, someone who is extremely high on NA may benefit from having a larger standard deviation because this will necessarily indicate that

they have more occurrences of low NA (Figure 1). On the other hand, they will also have more occurrences of high NA which may be detrimental. Because affect variability research often does not examine this interaction between affect variability and mean level, it is an open question as to if and when greater variability could be beneficial.

In conclusion, affect variability usually requires the assessment of affect over time and can be calculated in several ways. However, the majority of approaches do not take into consideration the patterning of affect over time and instead only focus on the magnitude. Affect variability is a distinct construct and may be stable across time. Furthermore, it is important to consider the valence (PA versus NA) as they are only moderately correlated. Mean levels of affect should also be taken into account because they may interact with affect variability in their association with other variables.



*Figure 1.* Two individuals with the same mean negative affect but with different standard deviations. M = mean; SD = standard deviation.



## **Affect Variability and Health and Health-Relevant Outcomes**

Given a better understanding of the methods used to assess affect variability, the next step is to review how affect variability is associated with health and health-relevant outcomes. In this section, Table 2 is presented to summarize some of the prominent findings. Table 2 states which methods were used to measure affect variability, whether each study considered mean levels of affect (to determine whether associations are beyond just mean levels of affect), and if the interaction between affect variability and mean level was tested. In this review, health is used as a catch-all term to refer to both psychological/mental (e.g., depressive symptoms) and physical (e.g., chronic conditions) health. Furthermore, variables such as anxiety are referred to as health-relevant outcomes as they may have consequences for future psychological and physical health.

Evidence suggests that affect variability may be associated with worse mental health (see Table 2). For example, Gruber and colleagues (2013) measured affect once a day for 14 consecutive days in an adult sample and found that greater variability (measured with standard deviation) in PA, but not NA, was associated with worse psychological functioning as well as greater depression and anxiety. These findings held even when controlling for mean levels of affect which suggests that affect variability may predict mental health over and above mean levels of affect. However, no interaction between affect variability and mean affect was tested.

Table 2

*Summary of Health and Health-Relevant Factors Associated with Affect Variability.*

<b>Study</b>	<b>Participants</b>	<b>Timing of Affect Assessment</b>	<b>Measurement of Affect Variability</b>	<b>Relevant Findings</b>	<b>Did Findings Hold Controlling for Mean Levels?</b>	<b>Interaction with Mean Level Tested?</b>
Gruber et al. (2013)	244 participants from the Denver, Colorado community ( $M_{age} = 40.69$ )	Once a day for 2 weeks	Standard deviation	Greater affect variability in PA was associated with worse psychological health (e.g., functioning, depression, and anxiety)	Yes	No
Hardy & Segerstrom (2016)	MIDUS	Reported once per night for 8 consecutive nights	Standard deviation	Greater affect variability in NA and PA was associated with worse psychological and physical health; only variability in NA at time 1 was associated with these outcomes (years later) at time 2	Yes	No
Underwood & Froming (1980)	Total across six studies: 1,435	One day	One-time assessment (Reactivity subscale of Mood Survey)	Reactivity was positively associated with social anxiety (Study 5) and depression scores (Study 6)	Did not control for mean level in anxiety analyses; Did control for mean level in depression analysis and findings held	No
Peeters et al. (2006)	47 individuals diagnosed with depression who were not taking antidepressants and 39 healthy individuals	Reported 10 times per day for 6 consecutive days	Multi-level modeling (test of homogeneous error variances)	Individuals with depression had a different pattern of PA such that the maximum report of PA occurred later in the day compared to the healthy controls; affect variability of PA was similar between the two groups; longer time since depression	Yes	No

				diagnosis was associated with reduced PA variability; Individuals with depression had a different NA pattern as well as more variable NA		
Trull et al. (2008)	34 individuals with borderline personality disorder and affective instability and 26 individuals with major depressive disorder/dysthymic disorder and no report of affective instability	Reported 6 times a day for 28 days	Multi-level modeling (test of homogeneous error variances between groups); adjusted squared successive differences; adjusted acute change	Variances of PA were greater for borderline personality disorder compared to major depressive disorder/dysthymic disorder; Successive difference scores were larger for borderline personality disorder compared to major depressive disorder/dysthymic disorder	Yes	No
Jahng et al. (2008)	46 individuals with borderline personality disorder and affective instability and 38 individuals with major depressive disorder/dysthymic disorder and no report of affective instability	Reported 6 times a day for 28 days (only NA analyzed)	Mean square successive differences; probability of acute change; standard deviation	Individuals with borderline personality disorder had significantly greater within-day and between-day NA variability compared to individuals with major depressive disorder/dysthymic disorder	Yes	No

Similarly, Hardy and Segerstrom (2016) using the National Study of Daily Experiences (NSDE) Waves 1 and 2 from the Midlife Development in the United States (MIDUS) dataset, found that participants with greater variability in both PA and NA experienced greater psychological distress (a composite of general mental health, depression, anxiety, and well-being) and worse physical health (a composite of general physical health, chronic conditions, activities of daily living, and medications) even when controlling for each respective mean level of affect (i.e., mean NA controlled in the modeling examining variability in NA). However, variability of NA had a larger effect on health when looking at the standardized coefficients. Additionally, it was only greater variability in NA at Wave 1 that predicted psychological distress and worse physical health at Wave 2 (7 to 13 years later). When both types of affect variability (PA and NA) and mean levels of both PA and NA were placed into one model, greater affect variability of NA remained significant while affect variability of PA became non-significant.

This same pattern of findings occurs with one-time self-reported affect variability. For example, Underwood and Froming (1980) compared their reactivity subscale of the Mood Survey with depression scores as measured with the Beck Depression Inventory (BDI; Beck, 1967) and found that higher reactivity was significantly associated with higher BDI scores even when controlling for the average mood level subscale. Additionally, reactivity was associated with greater anxiety.

In addition to studies of non-clinical participants, investigations with clinical populations suggest that pathologies such as major depressive disorder (Peeters, Berkhof, Delespaul, Rottenberg, & Nicolson, 2006), anxiety disorders, and borderline personality disorder (Trull et al., 2008) are also linked to high affect variability. Peeters and colleagues (2006) compared

ratings of affect of individuals with major depressive disorder to healthy controls and found that the pattern of PA, assessed using multilevel modeling, was different for individuals with major depressive disorder such that they experienced their highest level of PA later in the day relative to healthy controls. Additionally, individuals with major depressive disorder had a different pattern of NA such that NA tended to rise and then fall throughout the day while for healthy controls, NA remained flat. Further, NA was more variable throughout the day, but not between days, for individuals with major depressive disorder compared to the healthy controls. There were no differences in PA variability between the groups (even though patterns of PA were different between the groups).

Individuals with borderline personality disorder have been shown to have especially high rates of affect variability. For example, in one dataset with affect rated six times a day for 28 consecutive days, individuals with borderline personality disorder had greater affect variability (tested by comparing multi-level models with and without the assumption of equal variances between groups, and using adjusted squared successive differences and adjusted acute change) compared to those with major depressive disorder or dysthymic disorder (Trull et al., 2008). Interestingly, there were no differences in mean levels of PA or NA between the groups suggesting that affect variability is a distinct construct from mean levels of affect. Using this same dataset, Jahng and colleagues (2008) used mean square successive differences and probability of acute change and found that NA was more variable in the borderline personality disorder group compared to the group with major depressive disorder/dysthymic disorder. Furthermore, the pattern of findings was the same when these researchers used standard deviation as the measure of affect variability.

What is most interesting is that although these disorders such as depression, anxiety disorders, and borderline personality disorder are associated with *high affect variability* (i.e., experienced affect variability) they are also linked to *low psychological flexibility* (i.e., the *inability* to change affect; see previous discussion on psychological flexibility). For example, a major symptom of depression is the experience of a low mood and flat affect in response to many circumstances. A meta-analysis demonstrated that individuals with depression experience less positive and less negative affect in response to positive and negative stimuli, respectively, suggesting insensitivity to the affective context (Bylsma, Morris, & Rottenberg, 2008). This finding along with the pathology findings, may suggest that psychological flexibility (*ability*) does not translate into *experienced* affect variability and that possibly individuals with lower psychological flexibility may be unable to regulate their affective experiences leading to much more variable states. It may be that these individuals have poor affect regulation skills or use what skills they do have ineffectively (Linehan, 1993). So, although it is likely that psychological flexibility is adaptive and that engaging appropriately with the environment is beneficial, we see that maladaptive psychological functioning is associated with more experienced affect variability.

### **Conclusion**

A better understanding of how state affect and affect variability are calculated allows researchers to assess findings associated with affect variability and health. Building upon this knowledge, we see that affect variability has been tied to several health and health-relevant outcomes showing that increases in affect variability are usually associated with worse outcomes (e.g., depression, anxiety). However, several points seem to be missing from the literature. First, studies often only assess psychological health and not markers of physical health or

physiological parameters that are tied to health. Second, although studies have controlled for mean levels of affect, they typically do not look at the interaction between mean levels and affect variability (see Table 2, column 7). As previously described, this interaction may provide distinct predictions as to when affect variability may be beneficial or harmful. Third, the current methods for assessing affect variability do not take into account temporal patterns. Therefore, this dissertation adds to the literature by addressing these limitations in the field of affect variability and health.

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## **CHAPTER 3:**

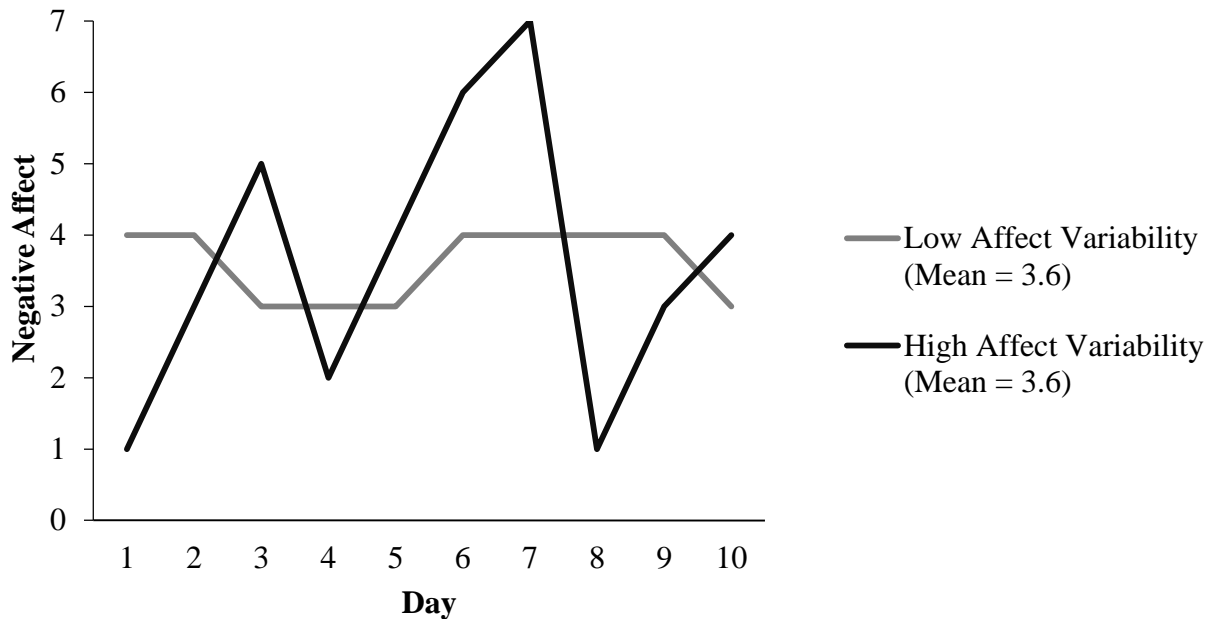
### **When is Affect Variability Bad for Health? The Association between Affect Variability and Immune Response to the Influenza Vaccination**

### 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 influence an individual's immune response. Those high in mean positive affect (PA) who had low PA variability were more likely to have higher Ab levels in contrast to those who had high mean PA and high PA variability. Although it did not interact with mean negative affect (NA), NA variability did influence Ab levels, whereby those with less NA variability mounted a more robust immune response. **Conclusion:** Affect variability influences immune response to an influenza vaccination and in some cases interacts with mean levels of affect. Low NA variability and low PA variability paired with high mean PA is the ideal affective experience for mounting a strong immune response. These oscillations in affective experiences are critical to consider in order to unpack the intricacies of how affect influences health. These findings should implore future researchers to consider the important role of affect variability on health-relevant outcomes.

Positive affect (PA), such as feelings of joy or happiness, has been repeatedly tied to better health and physiological function (Chida & Steptoe, 2008; Prather, Marsland, Muldoon, & Manuck, 2007; Pressman & Cohen, 2005), while the converse is true of negative affect (NA; e.g., feelings of sadness or anger; (Suls & Bunde, 2005)). 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 (Larsen & Diener, 1985).

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; Pace et al., 2009; Steptoe, Wardle, & Marmot, 2005), it seems plausible that these variability differences may have health-relevant consequences.



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

A substantial body of evidence suggests that affect variability may be associated with worse mental health (see meta analytic review Houben, Van Den Noortgate, & Kuppens, 2015). For example, Gruber and colleagues (Gruber, Kogan, Quoidbach, & Mauss, 2013) 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 (2016) 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 health, there are a few limitations to this work. First, previous research has 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. 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 (i.e., valleys in variability). For NA, similar instances could occur. Individuals with high mean NA may benefit from high variability because this provides “breaks” in NA (during the valleys in variability), 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. A second limitation in the literature is the near absence 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 (Human et al., 2015). If we are to better understand the toll affect variability takes on health, we must continue to study objective markers of health.

Another 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 (Burns, Carroll, Drayson, Whitham, & Ring, 2003; Cohen, Miller, & Rabin,

2001; Vedhara, Fox, & Wang, 1999). This is a useful paradigm because it measures how a person responds to a viral challenge in the body (in contrast to *in vitro* Ab levels which reflect the Ab levels naturally circulating in the body). Given the importance of a quick and large rise in Ab to ensure protection against virus exposure (Cohen et al., 2001), vaccination response provides us with a health-relevant indicator of immune functioning. For the influenza vaccine, Ab response one month post-vaccination represents maximum Ab production in response to the vaccination, while Ab response after that time typically declines (e.g., Phillips et al., 2006). Critically, affect variability experienced immediately following vaccination might have physiological or behavioral implications that may be associated with these Ab levels.

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, examining previously unexplored interaction effects, and measuring a novel health-relevant biomarker that provides rich information about immunocompetence. 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 (Eid & Diener, 1999; Ram & Gerstorf, 2009; Röcke & Brose, 2013). We interacted mean affect with affect variability to uncover whether affect variability has different implications for 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 (Sheldon Cohen, Doyle, Turner, Alper, & Skoner, 2003; Usala & Hertzog, 1989). Participants reported how much each adjective represented their current affect at each of the diary entries. PA was assessed with the items active, intense, enthusiastic, lively, happy, cheerful, relaxed, and calm. NA was assessed with the items jittery, nervous, unhappy, and sad. PA and NA items were rated on a scale from 0 (Not at all) to 4 (Extremely). Cronbach's alphas for PA ranged from .68 to .85 and Cronbach's alphas for NA ranged from .56 to .84 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 values<sup>2</sup>. Therefore, adjective items were averaged over each of the 44 time points (11 days\*4 assessments) to create a PA and NA mean value. Then, standard deviations over the 44 time points were calculated for PA and NA. These calculations resulted in the values 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 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 (Burns et al., 2003) and past work with this data set has found associations with only the A/New Caledonia virus (Pressman et al., 2005), 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

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<sup>2</sup> We used only these days because past research has shown that psychological variables are more influential after the vaccine (Miller et al., 2004).

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  $\mu$ 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. Due to problems of substantial negative skewness of the outcome variable that could not be alleviated using transformations, Ab level at both the one month and four month follow-ups was dichotomized into high (coded as 1; Ab levels greater than or equal to 1024 titers) and low (coded as 0; Ab levels ranging from 4 to 256 titers). 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.

The independent variables of interest were  $NA_{SD}$  and  $PA_{SD}$  and their interaction 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.

Probit models (StataCorp, 2015) were used to investigate the effect of the independent variables on Ab level. Probit models assess the probability of being in the group coded 1 (when a dichotomous dependent variable is used) given a set of predictor variables. Probit models assume that the errors are normally distributed. This is in contrast to logistic regression in which the

errors are assumed to follow a Bernoulli distribution (however, the pattern of results remains the same when logistic models are used).

Consistent with previous research (e.g., Pressman et al., 2005), 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).

## Results

### Descriptive Statistics

$NA_{MEAN}$  and  $PA_{MEAN}$  were significantly and largely different in value ( $t(82) = 8.40, p < .001$ ), while  $NA_{SD}$  and  $PA_{SD}$  were closer in value but still significantly different ( $t(82) = 3.14, p = .002$ ; 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  $PA_{SD}$  ( $r = 0.16, p = .140$ ).

Table 1

*Descriptive Statistics of Affect Mean and Standard Deviation Measures.*

Measure	Mean	SD	2	3	4
1. NA <sub>MEAN</sub>	0.81	0.43	-0.31**	0.63**	0.11
2. PA <sub>MEAN</sub>	1.52	0.51		-0.31**	0.16
3. NA <sub>SD</sub>	0.46	.17			0.50**
4. PA <sub>SD</sub>	0.50	.16			

*Note.* NA = Negative Affect; PA = Positive Affect; SD = Standard Deviation.

\*\* $p < 0.01$ ; \*  $p < 0.05$

### **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:  $B = -2.12$ ,  $z = -1.96$ ,  $p = .050$ ; four months:  $B = -2.49$ ,  $z = -2.15$ ,  $p = .031$ ) 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 (see Tables 2 and 3; one month:  $B = -1.95$ ,  $z = -1.48$ ,  $p = .139$ ; four months:  $B = -2.37$ ,  $z = -1.71$ ,  $p = .087$ ). Additionally, there was no significant interaction between  $NA_{SD}$  and  $NA_{MEAN}$  (see Tables 2 and 3; one month:  $B = -0.34$ ,  $z = -0.14$ ,  $p = .888$ ; four months:  $B = 0.40$ ,  $z = 0.16$ ,  $p = .874$ ).



Table 2

*Standard Deviation and Mean of Negative Affect and Positive Affect Predicting Antibody Level at One 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 <sub>SD</sub>	<b>-2.12<sup>†</sup></b>		-1.95	-1.90					<b>-5.55*</b>
NA <sub>MEAN</sub>		-0.51	-0.11	-0.13					0.27
NA <sub>SD</sub> * NA <sub>MEAN</sub>				-0.34					1.52
PA <sub>SD</sub>					-1.76		-1.57	-2.73	0.44
PA <sub>MEAN</sub>						-0.36	-0.28	<b>-1.01<sup>†</sup></b>	<b>-1.98**</b>
PA <sub>SD</sub> * PA <sub>MEAN</sub>								<b>-15.39**</b>	<b>-17.94**</b>
Baseline Ab Level	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.01	0.01
Female	0.16	0.09	0.15	0.16	0.11	-0.01	0.05	0.31	0.37
(1 = female)									
Caucasian	<b>0.66<sup>†</sup></b>	<b>0.59<sup>†</sup></b>	<b>0.66<sup>†</sup></b>	<b>0.65<sup>†</sup></b>	<b>0.63<sup>†</sup></b>	0.56	<b>0.64<sup>†</sup></b>	0.50	0.77
(1 = Caucasian)									
Cohort	<b>-1.19**</b>	<b>-1.21**</b>	<b>-1.20**</b>	<b>-1.20**</b>	<b>-1.14**</b>	<b>-1.17**</b>	<b>-1.12**</b>	<b>-1.43**</b>	<b>-1.72**</b>
Constant	<b>1.76**</b>	<b>1.95**</b>	<b>1.78**</b>	<b>1.80**</b>	<b>1.72**</b>	<b>1.86**</b>	<b>1.70**</b>	<b>2.21**</b>	<b>2.39**</b>

*Note.* NA = Negative Affect; PA = Positive Affect; SD = Standard Deviation; Ab = Antibody. \*\* $p < 0.01$ ; \* $p < 0.05$ ; <sup>†</sup> $p < 0.10$ .

Table 3

*Standard Deviation and Mean of Negative Affect and Positive Affect Predicting Antibody Level at Four 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 <sub>SD</sub>	<b>-2.49*</b>		<b>-2.37<sup>†</sup></b>	<b>-2.44<sup>†</sup></b>					<b>-5.16*</b>
NA <sub>MEAN</sub>		-0.57	-0.08	-0.05					0.16
NA <sub>SD</sub> * NA <sub>MEAN</sub>				0.40					1.62
PA <sub>SD</sub>					-1.98		-1.89	-2.72	-0.15
PA <sub>MEAN</sub>						-0.26	-0.16	-0.58	<b>-1.53*</b>
PA <sub>SD</sub> * PA <sub>MEAN</sub>								<b>-10.24*</b>	<b>-12.63*</b>
Baseline Ab Level	<b>0.01*</b>	<b>0.01*</b>	<b>0.01*</b>	<b>0.01<sup>†</sup></b>	<b>0.02*</b>	<b>0.02*</b>	<b>0.02*</b>	<b>0.02*</b>	<b>0.01*</b>
Female	0.07	-0.01	0.07	0.07	0.04	-0.09	0.01	0.10	0.07
(1 = female)									
Caucasian	0.31	0.28	0.31	0.32	0.31	0.23	0.32	0.23	0.39
(1 = Caucasian)									
Cohort	<b>-1.08**</b>	<b>-1.10**</b>	<b>-1.09**</b>	<b>-1.09**</b>	<b>-0.97**</b>	<b>-1.03**</b>	<b>-0.96**</b>	<b>-1.13**</b>	<b>-1.37**</b>
Constant	<b>1.59*</b>	<b>1.77**</b>	<b>1.61*</b>	<b>1.60*</b>	<b>1.46*</b>	<b>1.65*</b>	<b>1.45*</b>	<b>1.80*</b>	<b>2.04*</b>

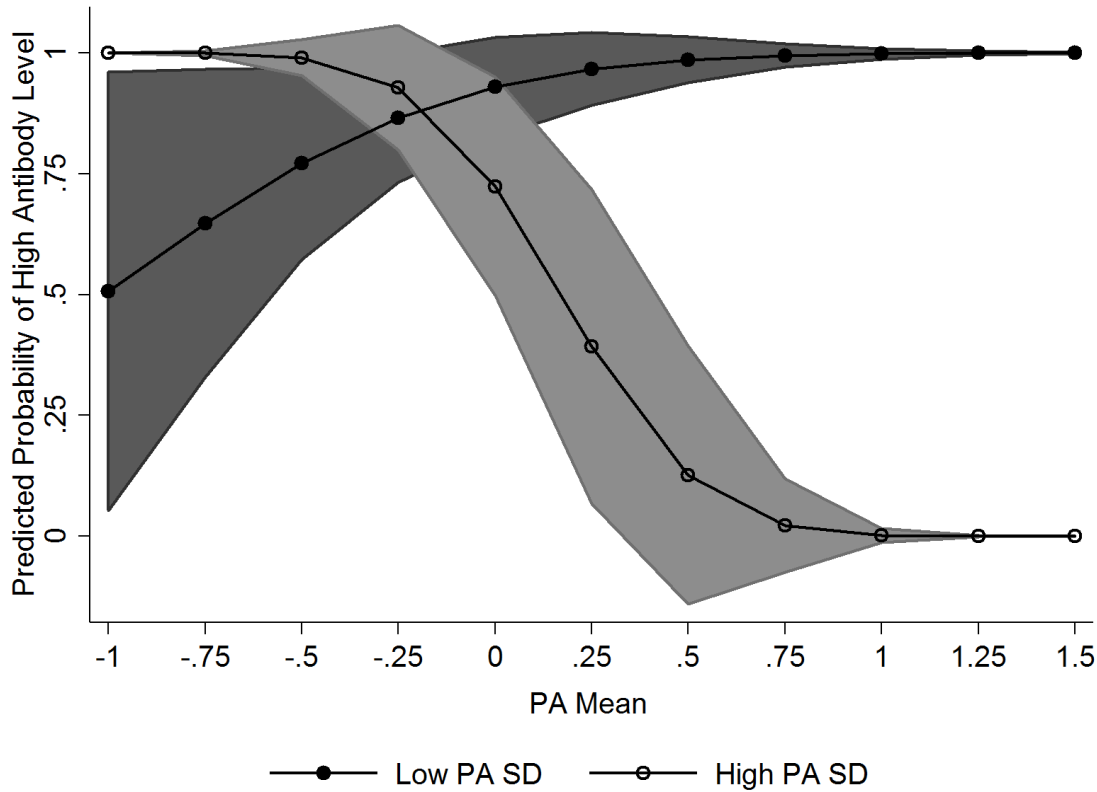
*Note.* NA = Negative Affect; PA = Positive Affect; SD = Standard Deviation; Ab = Antibody. \*\* $p < 0.01$ ; \* $p < 0.05$ ; <sup>†</sup> $p < 0.10$ .

PA<sub>SD</sub> was not significantly associated with Ab levels (see Tables 2 and 3; one month:  $B = -1.76$ ,  $z = -1.45$ ,  $p = .147$ ; four months:  $B = -1.98$ ,  $z = -1.52$ ,  $p = .128$ ). This lack of association remained non-significant when controlling for PA<sub>MEAN</sub> (see Tables 2 and 3; one month:  $B = -1.57$ ,  $z = -1.26$ ,  $p = .420$ ; four months:  $B = -1.89$ ,  $z = -1.43$ ,  $p = .152$ ). However, PA<sub>SD</sub> did significantly interact with PA<sub>MEAN</sub> to predict Ab levels at both time points (see Tables 2 and 3; one month:  $B = -15.39$ ,  $z = -2.95$ ,  $p = .003$ ; four months:  $B = -10.24$ ,  $z = -2.02$ ,  $p = .043$ ). Specifically, at high values of PA<sub>MEAN</sub>, individuals with lower levels of PA<sub>SD</sub> (i.e., less variability) were more likely to have higher Ab levels at follow-up (see Figures 2 and 3). In contrast, at high PA<sub>MEAN</sub>, individuals with higher PA<sub>SD</sub> were less likely to have higher Ab levels, while at low PA<sub>MEAN</sub>, higher PA<sub>SD</sub> was tied to a higher Ab response level (at least at one month post-vaccination).

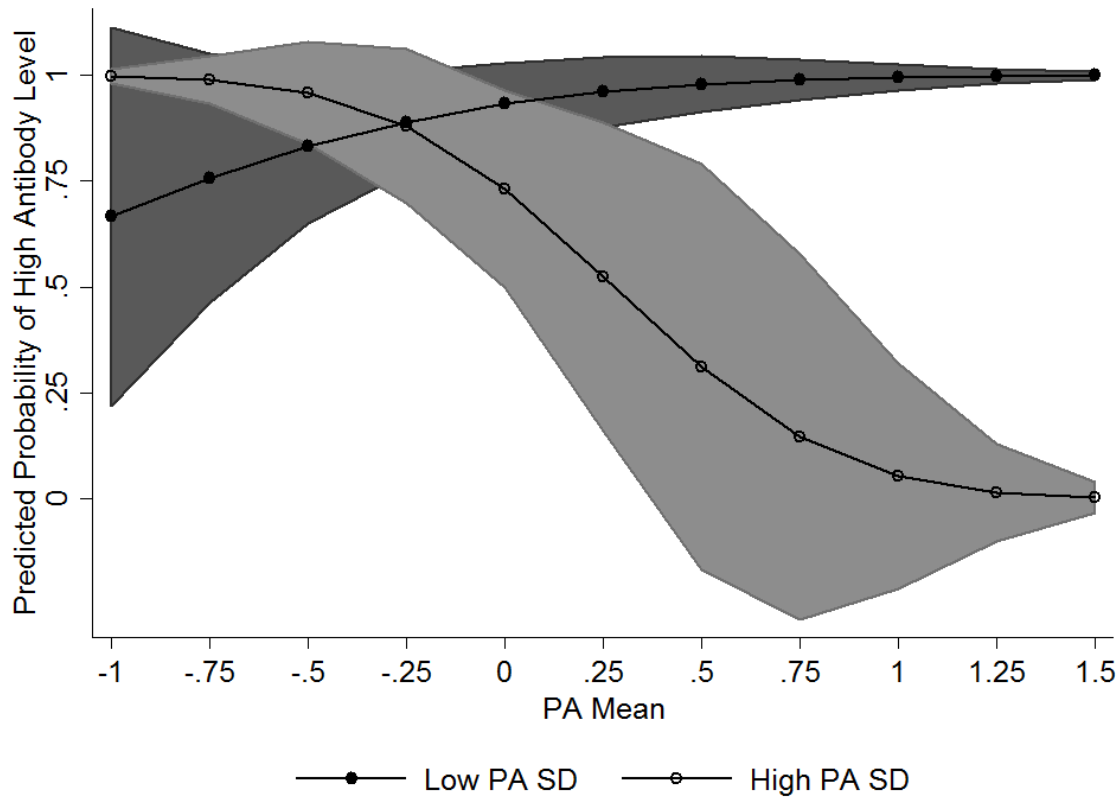
Interestingly, when the NA and PA variables were placed in the same model, the findings for the interaction between PA<sub>SD</sub> and PA<sub>MEAN</sub> (see Tables 2 and 3; one month:  $B = -17.94$ ,  $z = -3.26$ ,  $p = .001$ ; four months:  $B = -12.63$ ,  $z = -2.48$ ,  $p = .013$ ) as well as the main effect of NA<sub>SD</sub> (see Tables 2 and 3; one month:  $B = -5.55$ ,  $z = -2.41$ ,  $p = .016$ ; four months:  $B = -5.16$ ,  $z = -2.25$ ,  $p = .024$ ) held<sup>3</sup>.

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<sup>3</sup> Past 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<sub>SD</sub> became marginally significant as a main effect and NA<sub>SD</sub> became non-significant in some models. This non-significance may reflect the substantial overlap in NA<sub>SD</sub> with loneliness ( $r = 0.47$ ,  $p < .001$ ) and stress ( $r = 0.52$ ,  $p < .001$ ).



*Figure 2.*  $PA_{MEAN}$  and  $PA_{SD}$  interaction on antibody (Ab) levels at one month post-vaccination. Low  $PA_{SD}$  is one SD below the mean on  $PA_{SD}$  while high  $PA_{SD}$  is one SD above the mean on  $PA_{SD}$ . Lines represent adjusted predictions. Shaded regions are the 95% confidence intervals around the predictions. Regions of  $PA_{SD}$  (low vs. high) that do not overlap are significantly different from one another in terms of the predicted probability of having a high Ab level. Possible predicted probability values range between 0 and 1. However, confidence intervals exceed this range.



*Figure 3.*  $PA_{MEAN}$  and  $PA_{SD}$  interaction on antibody (Ab) levels at four months post-vaccination. Low  $PA_{SD}$  is one SD below the mean on  $PA_{SD}$  while high  $PA_{SD}$  is one SD above the mean on  $PA_{SD}$ . Lines represent adjusted predictions. Shaded regions are the 95% confidence intervals around the predictions. Regions of  $PA_{SD}$  (low vs. high) that do not overlap are significantly different from one another in terms of the predicted probability of having a high Ab level. Possible predicted probability values range between 0 and 1. However, confidence intervals exceed this range.

## 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). Specifically, high NA variability is associated with lower Ab levels. However, this effect becomes marginal when controlling for  $NA_{MEAN}$  levels. Interestingly,  $NA_{MEAN}$  on its own did not predict Ab levels. While this finding runs counter to past studies demonstrating the negative association between NA and immune response (e.g., [Evans, Bristow, Hucklebridge, Clow, & Walters, 1993]), this may be due to the low mean of NA in our sample ( $NA_{MEAN} = 0.81$  on a 0 to 4 scale) creating a floor effect.

The results in regard to PA are more nuanced and depend on the interaction between PA mean and variability. Individuals high in  $PA_{MEAN}$  who have low variability (consistently stay at their high PA level as opposed to drastically bouncing up and down around it) have the most robust immune response. However, if an individual has high  $PA_{MEAN}$ , then high PA variability is detrimental for mounting a large Ab response. It is possible that the negative ramifications of dropping into a valley of variability (e.g., lower PA) is not offset by the peaks of variability (e.g., higher PA) that an individual with high PA variability may experience. In contrast, if an individual has low levels of  $PA_{MEAN}$ , then variability in PA has slight beneficial effects on Ab levels (see Figure 2). These results suggest that high variability in PA may compensate for low  $PA_{MEAN}$ . Potentially, the occasional peaks in PA that these individuals experience are, to some extent, strong enough to positively impact Ab levels (at least at one month post-vaccination).

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; Eaton & Funder, 2001; Jahng, Wood, & Trull, 2008; Kuppens, Van Mechelen, Nezlek, Dossche, & Timmermans, 2007; R ocke, Li, & Smith, 2009; Trull et al., 2008) that were not considered. However, we chose the standard deviation approach since it is the most common and understood, in addition to the fact that different variability measures often lead to similar results (e.g., Gruber et al., 2013; R ocke et al., 2009). A second limitation was that we did not consider the effect of affect variability before the vaccination. Because past research has shown that psychological variables are more influential after the vaccine (Miller et al., 2004), we used only the days on or after the flu vaccination to calculate variability<sup>4</sup>. Third, we only considered one virus. This decision was based on previous findings that psychosocial variables influence responses to only some virus strains (Phillips et al., 2006; Phillips, Carroll, Burns, & Drayson, 2005). Although it is impossible to say whether certain viruses are more sensitive to psychosocial factors, it has been suggested that there may be differences in strain novelty and the participant's previous exposure to the strain that could explain the differing findings in the literature (Vedhara et al., 1999).

Although the purpose of this study was not to elucidate the mechanisms that allow this association to operate, it is important to consider why affect variability determines whether or not mean affect levels exhibit their expected effects on Ab levels. One explanation may be

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<sup>4</sup> Furthermore, 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. Follow up analyses were conducted using the variability just the two days before the vaccination. However, this variability did not influence Ab levels. This may suggest that it is the variability that occurs after the vaccination that is most important for immunocompetence, but future research using more pre-vaccination days is needed.

connected to the influence of emotional well-being on social relationships. High affect variability may be indicative of poor emotion regulation which has implications for health (Sheldon Cohen & Wills, 1985; Gross & John, 2003). Additionally, affect variability is a key factor in neuroticism (e.g., Kuppens et al., 2007), and high levels of neuroticism have been shown to negatively influence immune response (Phillips et al., 2005). Future researchers should consider potential mechanisms to determine *how* affect variability influences immune response.

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 health-relevant parameters. Assessing vacillations in affect will help paint a more vivid picture of how our affective experiences influence health.



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## **CHAPTER 4:**

**Affect Variability and Predictability: Using Recurrence Quantification Analysis to Better  
Understand How the Dynamics of Affect Relate to Health**

## Abstract

Affect variability has been associated with health outcomes. However, previously utilized measurement methods for assessing affect *variability* (such as standard deviation) often do not account for the temporal patterns of affect over time which may be an important feature in understanding how the dynamics of affect relate to health. Recurrence quantification analysis (RQA) may help alleviate this problem by assessing temporal characteristics unassessed by past methods. RQA metrics, such as recurrence and determinism, measure the *predictability* of affect, and demonstrate how often patterns within affective experiences repeat. In this manuscript, we first contrast RQA metrics with standard deviation and mean approaches to demonstrate that RQA can further differentiate among patterns of affect (Study 1). In Study 2, we analyze the associations between these new metrics and health (namely, depressive symptoms, well-being, and somatic symptoms). We find that RQA metrics predict above and beyond mean levels and standard deviation of affect over time. In general, it was found that the most desirable outcomes stemmed from those who had high mean positive affect, low mean negative affect, low affect variability, and high affect predictability. Furthermore, metrics of affect predictability (i.e., RQA) and variability (i.e., standard deviation) interacted with mean levels of affect. These studies are the first to demonstrate that RQA techniques can add important information about how temporal patterns influence the association between affective experiences and health outcomes.



Affective experiences are dynamic in nature. Feelings fluctuate from moment to moment and are intricately interconnected in a complex temporal system. Many studies have demonstrated that average affect is linked to a variety of psychological and physical health outcomes (Chida & Steptoe, 2008; Pressman & Cohen, 2005), but these findings have often overlooked the importance of how affect variability, and possibly patterns in naturally occurring variation in affect over time may further predict health. Both the positive and negative affect (PA and NA, respectively) levels a person has are a product of both internal (e.g., affect regulation) and external (e.g., stressful situations) factors and as these factors change, so do levels of affect. These changes in intensity of affect over time are referred to as *affect variability* and are shown to have important consequences for health. For example, greater levels of affect variability have been associated with higher levels of depressive symptoms, worse psychological well-being, greater anxiety (Gruber, Kogan, Quoidbach, & Mauss, 2013; Peeters, Berkhof, Delespaul, Rottenberg, & Nicolson, 2006), more favorable daily cortisol trajectories (Human et al., 2015), and better immunocompetence (Jenkins, Hunter, Cross, Acevedo, & Pressman, under review). Thus, it is critical that researchers are equipped with the proper tools to accurately assess the intricacies of affect variability. Unfortunately, current measurements of affect variability may fail to capture important information about changes over time because of their limited ability to assess patterns of affective change.

The most common metric used to assess affect variability is the standard deviation approach (Röcke, Li, & Smith, 2009). This technique captures affect variability as a single value by calculating the standard deviation of an individual's scores on an affect measure over multiple time points (Eid & Diener, 1999; Gruber, Kogan, Quoidbach, & Mauss, 2013; Hardy & Segerstrom, 2016; Ram & Gerstorf, 2009; Röcke & Brose, 2013). An advantage of this approach

is that standard deviation is easily understood, and the value can be used as a predictor or outcome variable in subsequent models (Röcke et al., 2009). Nevertheless, this single value assumes an independence of assessment such that an assessment at time,  $t$ , is not necessarily related to time,  $t + 1$  (Ram & Gerstorf, 2009). For example, the same standard deviation value will result from a person scoring 2, 3, and 4 on an affect measure over three consecutive days and a person scoring 3, 4, and 2 on three consecutive days even though both individuals will have different patterns of affect over time. However, in most real-world environments, this assumption does not hold true. Previous affect is related to current affect, and current affect can predict future affect (Bai & Repetti, 2017). Assessing only standard deviation offers information about the magnitude of the change in affect, but provides no information about the sequential dependence or *predictability* of affective experiences. Essentially, standard deviation collapses temporal data across time leading to a loss of information about the temporal patterns or dynamics (change over time) of affective experiences.

Information about the dynamics of affect over time can provide more detailed insights over and above that of standard deviation. For example, an individual who has more structured or “predictable” NA values that denote a more recurrent pattern of affective experiences over time (i.e., exhibits a less stochastic and more stationary or periodic structure of affective change over time) may have much different outcomes compared to an individual who has “less predictable” values of NA that create a more inconsistent (i.e., stochastic) or less recurrent pattern of affect (see Figure 1). The predictability (i.e., regularity and consistency) of fluctuations in affective experiences over time may matter more than or in addition to just assessing the magnitude of those fluctuations. Taken together, affect *variability* and the patterning or dynamics of this variability may allow researchers to better understand the full

affective experience over time. Therefore, metrics that assess the dynamic structure and predictability of affect experiences over time are needed.

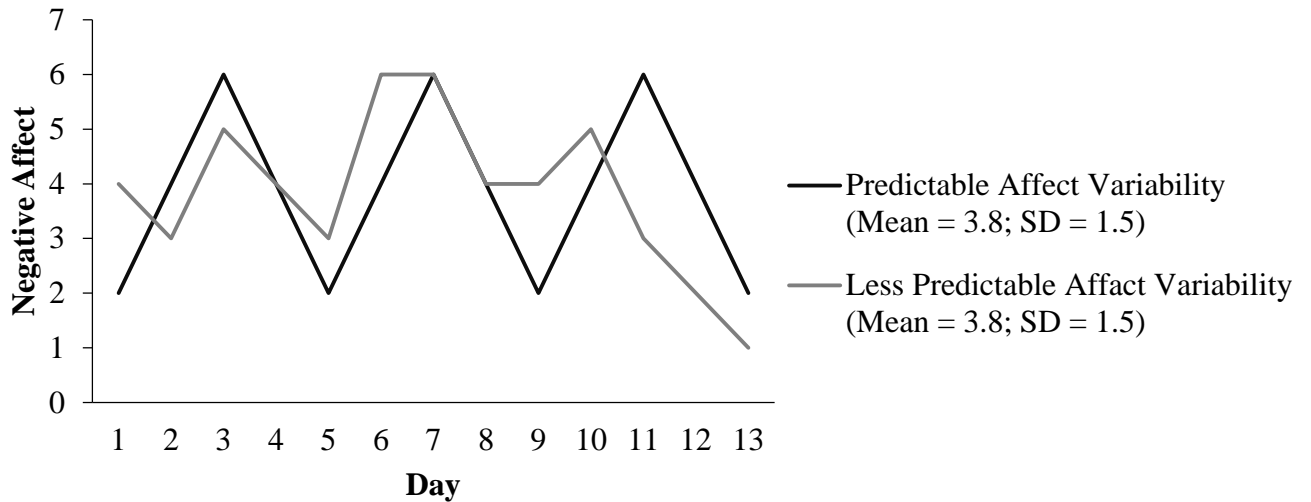


Figure 1. Two individuals with different predictability of affect variability. SD = standard deviation.

Recurrence quantification analysis (RQA) maybe one such method for assessing patterns of affect predictability. RQA is a non-linear event- or time-series analysis method that assesses the dynamics of temporal sequences of change over time, without researchers having to make any a-priori assumptions about the nature of the dynamics that define a given behavioral event or time-series recording. Although RQA has not been previously used to examine changes in affect over time, it has been successfully employed to investigate a wide range of other dynamic human behaviors (e.g., eye gaze [Anderson et al., 2013], posture changes [Riley & Clark, 2003]). Because of the temporal nature of affect, RQA lends itself nicely to studying dynamics of affect and, in particular, the degree of affect predictability (Richardson, Dale, & Marsh, 2014). RQA provides multiple metrics of predictability, two of the most common being recurrence rate or percent recurrence (%REC) and the percentage or degree of deterministic structure (%DET) within a measure series. %REC is a measure of state regularity which, in terms of affect, reflects

how *often* a person experiences the same (or similar) level of affect over time (i.e., the degree to which the same state of affect reoccurs over time). %DET measures the degree to which recurrent states exhibit regular or structured patterns of change over time. With regard to how affect changes over time, %DET captures the degree to which the same (or similar) sequences of affective change occur over time, such that more structured or “predictable” patterns of affective change over time should result in high levels of %DET. Although %REC and %DET may provide different information for longer time-series data (e.g., over 50 time points), they are often correlated for shorter time series (e.g., 15 time points).

Of particular relevance to the current study, is that the RQA metrics of %REC and %DET should provide more information than just average distance from the mean (i.e., standard deviation) and may help to more precisely capture temporal systems. In other words, the benefit of these RQA metrics with regard to understanding the dynamics of affect is that they can quantify the *predictability* of affect over time. Although previous research has demonstrated that greater affect variability (i.e., higher standard deviation) has negative implications for health (e.g., Gruber et al., 2013), higher affect predictability may be beneficial for health. Specifically, the regularity or predictability of how affect changes over time may allow individuals to better prepare and then cope with affective experiences. For example, an individual who knows that NA is always high Monday mornings may be better prepared to cope with such NA. Thus, it is important to see how mean levels of affect, affect variability (as captured through standard deviation), and affect predictability (as measured by RQA) interact, as these factors may operate together with well-being implications.

The purpose of the current research was to demonstrate the validity of the RQA method for quantifying the structure or predictability of affective time-series using simulated data (Study

1) and then apply RQA to a large, real data set (Study 2). Given the common use of standard deviation as a measure of affect variability, in Study 1 the RQA metrics of %REC and %DET were compared to standard deviation using simulated data. This simulation allowed these variability metrics to be compared to see if each could add independent information about the patterns of affect across time (i.e., days). Specifically, the predictions were that the same values of standard deviation (a measure of variability that does not take into account temporal structure) can be associated with, but not differentiate between, patterns that are more or less predictable (i.e., stochastic and/or periodic), but that the latter could be quantifiably differentiated using the RQA metrics of %REC and %DET. This predictability paired with variability produces different “cells” in which some simulated strands of data (i.e., data from one “person”) have high variability but low predictability (dynamics structure) while other stands have high variability and high predictability. Alternative combinations are low variability with high or low predictability. In Study 2, RQA metrics were then used, along with mean levels and standard deviation of affect, to predict health outcomes. We hypothesized that, as in previous literature, more variability (as measured with standard deviation) would be associated with worse psychological and physical health outcomes (i.e., more depressive symptoms, lower psychological well-being, and more somatic symptoms). In contrast, predictability (as measured by the RQA metrics %REC and %DET) would be associated with better psychological and physical health outcomes (i.e., fewer depressive symptoms, higher well-being, and fewer somatic symptoms).

## Study 1

### Methods

**Data simulation.** Affect data were simulated by creating 16 instances (i.e., days) for 900 cases (i.e., people). Data was generated in a way that would alter the standard deviation and predictability (i.e., stochasticity, periodicity) of the data over a 16 instance integer-value time-series. Nine groups of 100 cases each were created (see Table 1). Groups 1 through 3 had integer values generated between 1 and 7 (see Table 1 column 3). Groups 4 through 6 had integer values generated between 2 and 6. Groups 7 through 9 had integer values generated between 3 and 5. Generating the integer valued time-series in this way ensured that the first 3 groups would have large standard deviations (i.e., high variability), the second 3 groups would have medium standard deviations (i.e., medium variability), and the last 3 groups would have small standard deviations (i.e., low variability). In addition to altering the range of integer values, some cases had value sequences repeated (see Table 1 column 4), such that the data series contained levels of periodic structure. Repeating the values ensured that there would be greater predictability within these groups of data series, with greater levels of repeated (periodic) structure corresponding to higher predictability. One third of the groups had no values repeated (i.e., low predictability). One third of the groups had instances 1 through 8 repeated over instances 9 through 16 (i.e., medium predictability). One third of the groups had instances 1 through 4 repeated 3 times over instances 5 through 8, 9 through 12, and 13 through 16 (i.e., high predictability). It is important to note that this method of generating the data was not specific to a particular affect measure per se, but was employed to simply represent different amounts of variability and predictability that might be associated with changes in affect over time (e.g., days) when rated on either discrete or continuous scales.

Table 1

*Data Simulation Parameters and Predicted Results.*

Group	Group Name	Range of Numbers Generated	# of Days that repeat (to get predictability)	Predictions	
				SD (Variability)	RQA (Predictability)
1	High Variability – Low Predictability	1 – 7	none	Large	Small
2	High Variability – Medium Predictability	1 – 7	8 (first 8 are repeated a second time)	Large	Medium
3	High Variability – High Predictability	1 – 7	12 (first 4 are repeated 3 more times)	Large	Large
4	Medium Variability – Low Predictability	2 – 6	none	Medium	Small
5	Medium Variability – Medium Predictability	2 – 6	8 (first 8 are repeated a second time)	Medium	Medium
6	Medium Variability – High Predictability	2 – 6	12 (first 4 are repeated 3 more times)	Medium	Large
7	Low Variability – Low Predictability	3 – 5	none	Small	Small
8	Low Variability – Medium Predictability	3 – 5	8 (first 8 are repeated a second time)	Small	Medium
9	Low Variability – High Predictability	3 – 5	12 (first 4 are repeated 3 more times)	Small	Large

## **Measures.**

**Mean.** Means were calculated within individuals by summing the values for each of the 16 days and then dividing by 16. Each participant's mean therefore represents their average score over the entire daily diary period.

**Standard deviation.** Standard deviations were calculated within individual by summing the squared distances for each day from the overall mean and then averaging those squared distances.

**Recurrence quantification analysis (RQA).** RQA measures were calculated using the RQA software developed by Richardson, Riley, Shockley, and Dale (APA ATI, 2015; <http://xkiwilabs.com/software-toolboxes/>). Given that integer value time-series were investigated here and in Study 2, a form of RQA known as Categorical-RQA was employed. As illustrated in Figure 2, this method of RQA first involves identifying reoccurring (recurrent) values within a discrete time-series by plotting them on a 2-dimensional recurrence plot (Figure 2). Essentially, a data time-series is represented on both the  $x$  and  $y$  axis of a 2-dimensional grid, with recurrent points indicating when the same value with the data series reoccurs. For example, the first row of simulated data (i.e., data for one individual across 16 days) is presented in Figure 2. Recurrent points (i.e., "dots") within the recurrence plot correspond to when the same value reoccurs. Given that each value with the data series is current with itself, the main diagonal (line of identify) always includes recurrent points.



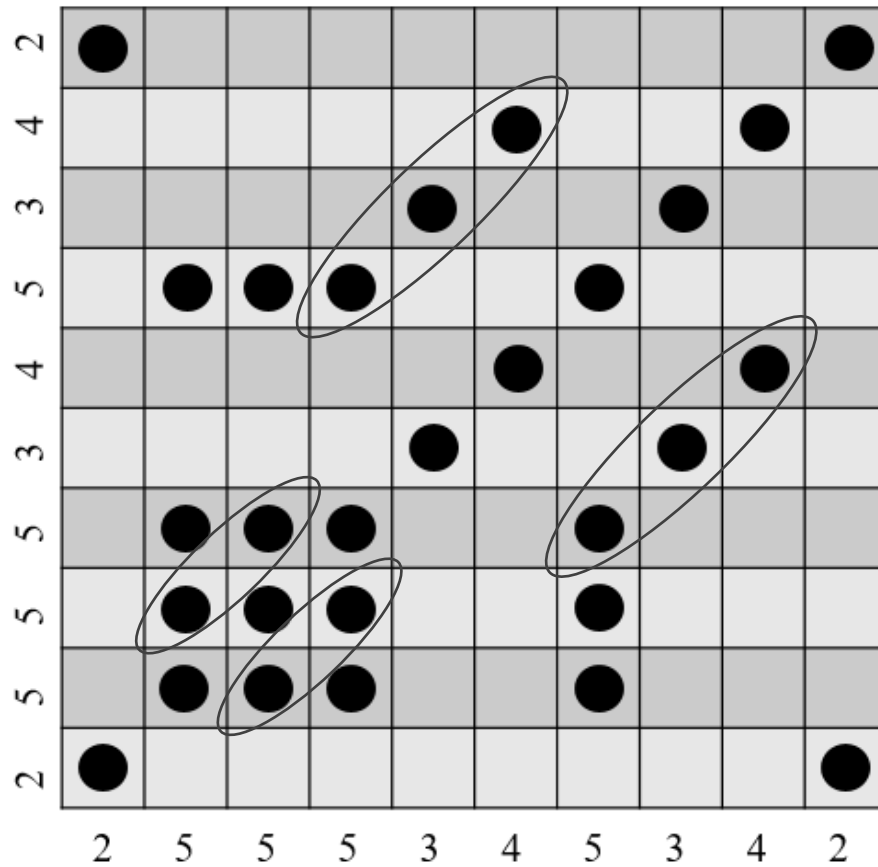


Figure 2. Data analysis for recurrence quantification analysis. One event- or time-series of data is placed on the x-axis. Then, the exact same event- or time-series is placed on the y-axis. “Dots” are placed where same values intersect. For example, in the lower left corner a “dot” is placed because there is a 2 on the x-axis and a 2 on the y-axis. Dots that are circled are those that lie on a diagonal line. Only dots that form diagonal lines (excluding the line of identity) that contain two or more recurrent points are used in the determinism calculation.

With regard to quantifying the recurrent structure within a recurrence plot, recurrent points along the line of identity are excluded given that these points reflect trivial recurrences. %REC is calculated by dividing the number recurrent points that do not fall along the main diagonal (in this case 18) by the number of spaces (in this case 90). So, in Figure 2,  $\%REC = 18/90 = .20 \rightarrow 20\%$  and therefore there is 20% REC in this strand of data. %REC represents the percentage of time an individual experiences the same level of affect as before. Therefore, 20% of the time, this individual is experiencing the same level of affect as before. Again, excluding the main diagonal, %DET equals the percentage of recurrent points that form diagonal lines within a recurrence plot, where a diagonal line corresponds to two or more consecutive recurrent points. For instance, in Figure 2,  $\%DET = 10/18 = .56 \rightarrow 56\%$ . %DET represented the percentage of time an individual experiences the same pattern of change in affect over time and, therefore, the degree of predictability or determinism within an affect time series.

**Statistical analysis.** Analysis of variance and post hoc pairwise comparisons with a Bonferroni correction for familywise error were used to assess differences in the metrics (i.e., mean, standard deviation, %REC, and %DET) among the 9 groups.

## Results

Figure 3 presents a visual depiction of the mean, standard deviation, and RQA measures for each of the 9 groups. Analysis of variance results indicated that there were overall differences among the 9 groups for each of the measures ( $p = .016$  for mean;  $ps < .001$  for standard deviation, %REC, and %DET). Post hoc pairwise comparisons with a Bonferroni correction for familywise error showed that the only difference between affect means was between groups 3 and 6,  $p = .002$  (all other group differences for the mean,  $ps > .05$ ; see Table 2). As can be seen in Figure 3a, the mean was relatively stable over the 9 groups. Even the two groups that were

significantly different from one another (i.e., groups 3 and 6) still had relatively similar means (4.14 and 3.89, respectively).

As for standard deviation, groups 1 and 2 were the largest, followed by group 3, followed by groups 4 through 6, and then followed by groups 7 through 9 (see Table 2 and Figure 3a). With the exception of group 3 being significantly smaller than groups 1 and 2, the simulated data were exactly in line with the predictions in Table 1 (namely, that groups 1 through 3 were the same as each other, groups 4 through 6 were the same as each other, and groups 7 through 9 were the same as each other). When data was simulated in such a way as to allow for more extreme values (i.e., increasing the range of possible values), standard deviations were larger.

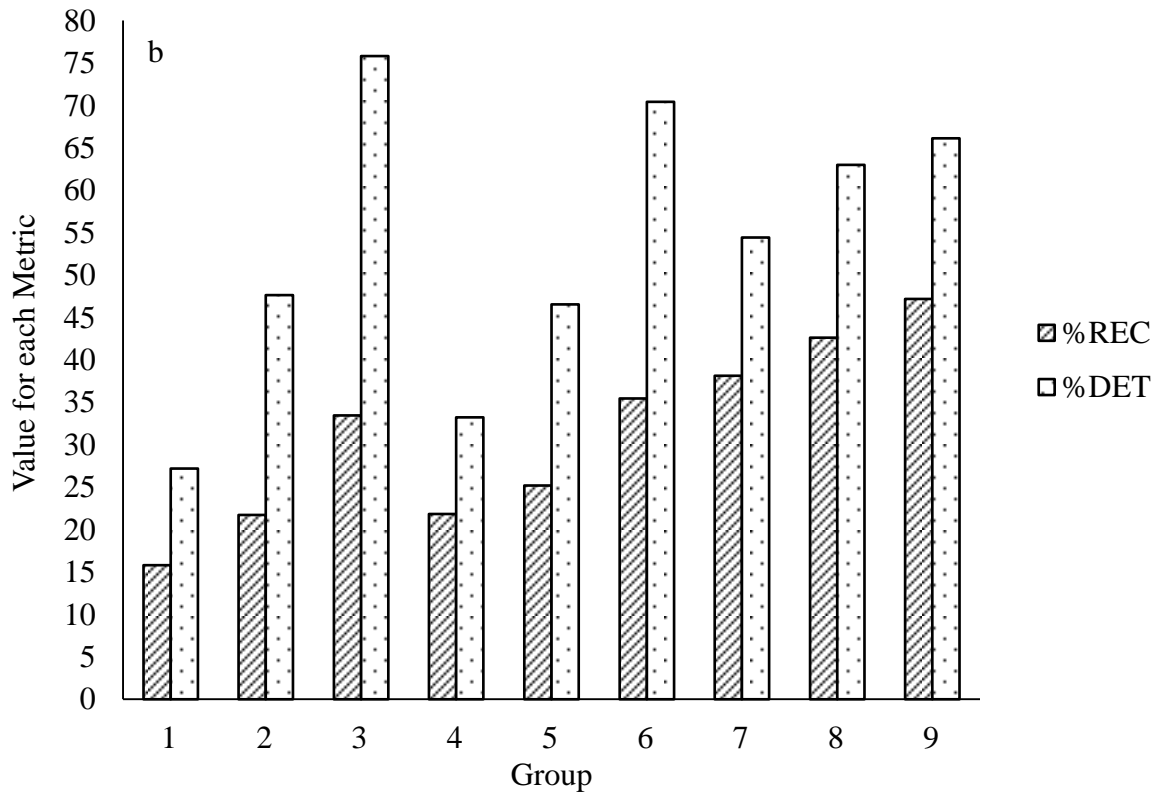
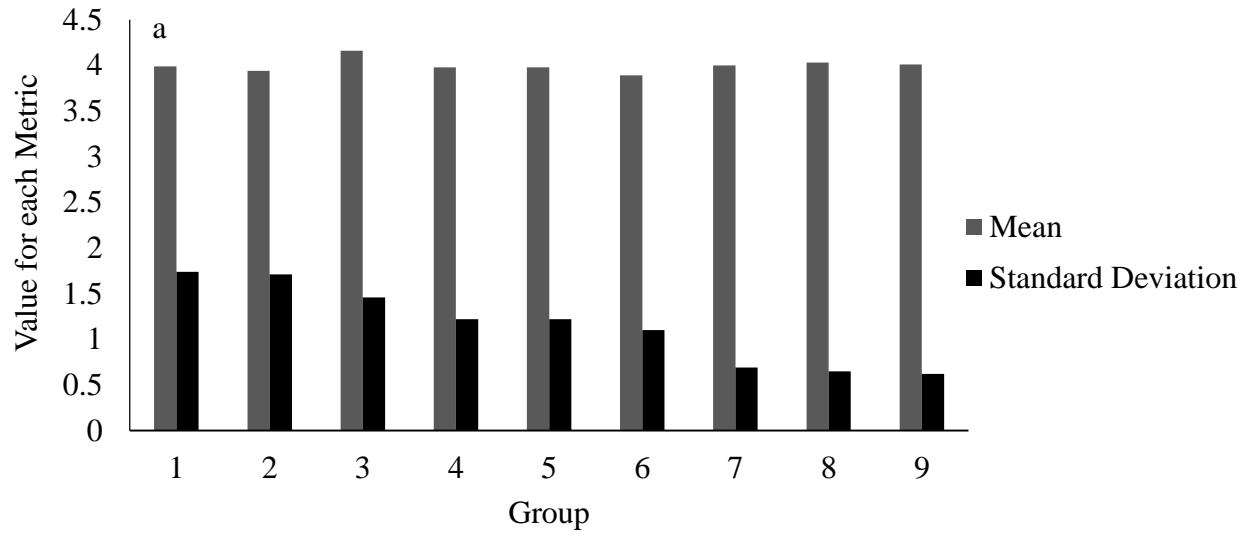


Figure 3. Mean, standard deviation, percent recurrence (%REC), and percent determinism (%DET) values by group. Note that the mean is about the same across all groups while the standard deviation is similar for groups 1 through 3, then 4 through 6, and then 7 through 9. %REC and %DET distinguish within these sets of groups.

Table 2

*Mean, Standard Deviation, and Recurrence Quantification Measures by Group.*

Group	Mean	Standard Deviation	%REC	%DET
1	3.99 <sup>ab</sup>	1.74 <sup>a</sup>	15.81 <sup>a</sup>	27.19 <sup>a</sup>
2	3.94 <sup>ab</sup>	1.71 <sup>a</sup>	21.73 <sup>b</sup>	47.65 <sup>bd</sup>
3	4.16 <sup>a</sup>	1.46 <sup>b</sup>	33.47 <sup>c</sup>	75.81 <sup>c</sup>
4	3.98 <sup>ab</sup>	1.22 <sup>c</sup>	21.86 <sup>b</sup>	33.24 <sup>a</sup>
5	3.98 <sup>ab</sup>	1.22 <sup>c</sup>	25.21 <sup>b</sup>	46.55 <sup>b</sup>
6	3.89 <sup>b</sup>	1.10 <sup>c</sup>	35.47 <sup>c</sup>	70.44 <sup>ce</sup>
7	4.00 <sup>ab</sup>	0.69 <sup>d</sup>	38.12 <sup>cd</sup>	54.42 <sup>d</sup>
8	4.03 <sup>ab</sup>	0.65 <sup>d</sup>	42.63 <sup>de</sup>	62.99 <sup>f</sup>
9	4.01 <sup>ab</sup>	0.62 <sup>d</sup>	47.20 <sup>e</sup>	66.13 <sup>ef</sup>

*Note.* Column values with similar letters indicate no significant difference ( $p > .05$ ). %REC = percent recurrence; %DET = percent determinism.

The %REC and %DET measures followed similar patterns as the hypothesized results (see Figure 3b). Looking within large (groups 1 through 3), medium (groups 4 through 6), and small (groups 7 through 9) standard deviation groups, %REC and %DET became larger as data was repeated more often (i.e., more predictable; in line with the hypotheses). So, for example, %REC grew from 15.81 to 21.73 to 33.47 across groups 1 through 3, respectively (see Figure 3b and Table 2) because, although the range of values was held constant for these groups, more instances of repeated data (i.e., predictability) occurred for group 3 compared to group 2 (and more repeated data occurred for group 2 compared to group 1). However, when the standard deviation was smaller (i.e., groups 7 through 9), there was less discrepancy between %REC and %DET values among the groups. For example, the pairwise comparisons between groups 7 and 8 and groups 8 and 9 were no longer significantly different,  $ps > .05$  (see Table 2). Nevertheless, the same pattern of results occurred throughout the data whereby as predictability increased, so did %REC and %DET values.

## **Discussion**

The results demonstrate that the RQA metrics of %REC and %DET can be employed to the dynamics of variables like affect and provide different and new pieces of information about how affect changes over time compared to traditional measures of standard deviation or mean. RQA further differentiates the simulated cases based on metrics of predictability that consider the role of time in assessing patterns of affective experiences. These findings show that using the mean as a measure of affect loses information about variability and predictability when looking at affect across time. Over the 9 groups, mean levels were relatively stable and did not distinguish between the groups. However, as noted by the other measures (standard deviation, %REC, %DET), the same mean level of affect can be associated with different levels of

variability and predictability. This demonstrates that studies relying only on mean level of affect across time may be overlooking important information.

In addition, these results demonstrate that relying solely on the standard deviation approach for assessing variability is insufficient for capturing the finer details about the patterns of variation across time. For groups 1 through 3 (see Figure 3a), the standard deviations (along with the means) were kept constant. Based on these values, previous researchers may have assumed that each of these groups were equal. Importantly, once the RQA measures are taken into consideration, a substantial difference in variation emerges and it becomes apparent that these groups are in fact not equal. When the same patterns of affect are repeated more often, there is an increase in %REC and %DET (see Figure 3b). For example, group 1 and group 3 have nearly identical means and standard deviations as one another. However, group 3 is different from group 1 because the same pattern of affect was repeated 3 times (i.e., more predictability), as noted by %REC and %DET. Repeated patterns, and the predictability that follows, vary from person to person and are indicative of individual differences in affective experiences. This is critical, because those differences in patterns of variation were not uncovered by simply using the standard deviation approach, and those differences may have distinct implications for various outcomes. The addition of RQA metrics adds more information about the dynamic nature of affect and demonstrates how the relation over time between affective experiences may influence how affective profiles are categorized and understood.

There are limitations to Study 1 that should be noted. First, the range and repeated nature of the data were selected arbitrarily and may or may not match ranges or repeated patterns in the natural environment. Yet, 5 point scales (such as the one that we simulated) are commonly used in affect measures (Curran, Andrykowski, & Studts, 1995) and so the medium standard deviation

groups are likely ecologically plausible. Further, in certain situations individuals may not use the full range of these scales, which would result in values mirroring the small standard deviation groups (in which the range was 3 points). Similarly, the repeated nature of the data may or may not be ecologically valid. It is likely that repeated affect could occur from week to week with, for example, affect on Mondays looking similar to affect on other Mondays and affect on Fridays looking similar to affect on other Fridays. Therefore, the medium repeated data (in which data from 8 days are repeated across the next 8 days) may closely reflect this week by week repeated structure. Additionally, a no repeated condition (small repeat) was included which would mirror the natural environment if affect did not follow a specific repeated structure. It is important to note that even when data were not purposefully repeated, random repeated days could have occurred.

Regardless of these limitations, this study demonstrates that RQA measures may add more detailed information above and beyond the simple standard deviation measure of variability. Thus, it is worth exploring if this new methodology offers additional explanatory power in terms of its association with real world outcomes. Being able to predict a future affective experience based on measures of predictability may allow an individual to be better prepared to cope with upcoming experiences and could lead to improvements in overall health and well-being.

Building on the findings of Study 1, in Study 2 we link affect mean, variability, and predictability to depressive symptoms, psychological well-being, and somatic symptom report (e.g., cold symptoms) as most previous studies assessing affect variability have concentrated on similar outcomes (Gruber et al., 2013; Houben, Van Den Noortgate, & Kuppens, 2015; Human et al., 2015; Peeters et al., 2006). Capturing averages, variability, *and* predictability of affective



experiences may provide researchers with a better understanding of how the intricacies of the affective experience influence mental and physical health. Furthermore, since these factors are not perfectly correlated, it may be advantageous to understand how they interact to predict certain health outcomes. This initial simulation study provided a foundational rationale for why these factors are important, and the following study applied the RQA methodology to real health outcomes.

## Study 2

### Method

**Participants.** Study 2 used data from the “Daily Life Study” conducted at the University of Otago. Participants included 1,482 college students ( $M_{age} = 19.76$ ,  $SD_{age} = 2.43$ ). Sixty-seven percent of the participants were female. Participants were 78% Caucasian, 10% Asian, 5% Pacific Islander, 3% Indian, and 4% were another ethnicity or mixed ethnicity.

**Procedure.** Participants completed an initial survey asking about demographics and depressive symptoms. Participants then completed daily diaries for 13 consecutive days. The diaries consisted of several questionnaires including affect and stress measures. After the 13 consecutive days, participants then completed a follow up survey asking about health and well-being.

### Daily measures.

**Affect mean, variability, and predictability.** State emotion adjectives were assessed each day for 13 days on a scale from 1 (Not at all) to 5 (Extremely) describing how much each of the words reflected how the participant felt that day. Nine PA words (happy, excited, cheerful, pleasant, calm, energetic, enthusiastic, content, and relaxed) were averaged to create a daily PA value (Cronbach’s alpha range for each of the 13 days = .88 to .92) and nine NA words (nervous,

dejected, irritable, hostile, sad, angry, unhappy, anxious, and tense) were averaged to create a daily NA value (Cronbach's alpha range for each of the 13 days = .87 to .91). Day averages were then averaged over the 13 days to create an overall PA and NA mean value. Then, standard deviations over the 13 time points were calculated for PA and NA. Finally, the RQA metrics, %REC and %DET, were calculated using the methods and software described in Study 1. All PA and NA daily mean values were rounded to the nearest integer value to allow for Categorical-RQA. These calculations resulted in the mean, variability, and predictability values used in the analyses: NA mean ( $NA_{MEAN}$ ), PA mean ( $PA_{MEAN}$ ), NA standard deviation ( $NA_{SD}$ ), PA standard deviation ( $PA_{SD}$ ), NA %REC ( $NA_{\%REC}$ ), PA %REC ( $PA_{\%REC}$ ), NA %DET ( $NA_{\%DET}$ ), and PA %DET ( $PA_{\%DET}$ ).

**Distress.** Distress was assessed each day with the question “Overall, how much stress (e.g., because of hassles, demands, or other stressors) have you been under today?” rated on a scale of 0 (no stress) to 4 (a great deal of stress). The response for each day was averaged over the 13 days and used as a control variable in all analyses (as has been done in previous research e.g., Gruber et al, 2013).

#### **Baseline measures.**

**Depressive symptoms.** The Center for Epidemiologic Studies Depression Scale (CES-D; Radloff, 1977), a 20 item measure, was used to assess depressive symptoms. Items included statements such as “I did not feel like eating; my appetite was poor,” “I thought my life had been a failure,” and “My sleep was restless.” Items were scored using the following scale: rarely (0), some (1), occasionally (2), or most (3). Scores were summed with higher values indicating higher levels of depressive symptoms.

### **Follow up measures.**

***Psychological well-being.*** Psychological well-being was assessed during the follow up survey with the Psychological Well-Being scale (Ryff & Keyes, 1995). The 18 items included statements such as “In general, I feel I am in charge of the situation in which I live” and “I like most aspects of my personality.” Items were rated on a scale from strongly disagree (1) to strongly agree (5). Items were reverse scored (when necessary) and summed together so that higher values reflected *worse* well-being. Well-being was scored in this way so that all dependent variables could be interpreted such that higher values represented worse outcomes (i.e., more depressive symptoms, worse well-being, and more somatic symptoms [see below]) and would fit the distribution assumption of poisson regression (see Statistical analysis section below).

***Self-reported somatic symptoms.*** During the follow up survey, participants were asked whether they felt like they had a cold or flu in the past two weeks (rated on a scale from 0 = not at all to 4 = very). Additionally, they were asked whether they “felt physically ‘run down’,” “felt tired,” and “felt refreshed when [they] woke up in the mornings.” These items were summed with higher values reflecting a greater number of self-reported somatic symptoms.

**Statistical analysis.** Depressive symptoms, well-being, and somatic symptoms were used as the dependent variables in all analyses. Due to the skewed distributions of all dependent variables, poisson regression was used.  $NA_{MEAN}$ ,  $PA_{MEAN}$ ,  $NA_{SD}$ , and  $PA_{SD}$ ,  $NA_{\%REC}$ ,  $PA_{\%REC}$ ,  $NA_{\%DET}$ , and  $PA_{\%DET}$  were used as predictor variables in the analyses.  $\%REC$  and  $\%DET$  were divided by 100 to be similar in value to the NA and PA means and standard deviations (as poisson does not produce standardized beta values). Poisson regression was used to predict each of the three dependent variables (depressive symptoms, well-being, and somatic symptoms) with

the predictor variables  $NA_{MEAN}$ ,  $PA_{MEAN}$ ,  $NA_{SD}$ , and  $PA_{SD}$ ,  $NA_{\%REC}$ ,  $PA_{\%REC}$ ,  $NA_{\%DET}$ , and  $PA_{\%DET}$ ) while also controlling for distress in Stata 14 (StataCorp, 2015). All predictor variables were mean centered to allow for easy interpretations in interaction terms. Because many models were used, AIC and BIC values are presented in the tables as a way to assess the best model fit. In the table notes, the AIC and BIC values are presented for a model with only mean levels and distress as a control. This enables readers to assess whether models with variability and predictability better account for the data over and above mean levels.

## Results

**Descriptive statistics.** Table 3 presents the means and standard deviations of the affect metrics as well as their associations.  $PA_{MEAN}$  was higher than  $NA_{MEAN}$  ( $t(1,298) = 59.90, p < .001$ , 95% CI of the difference [1.29, 1.37]).  $PA_{SD}$  was greater than  $NA_{SD}$  ( $t(1,298) = 15.44, p < .001$ , 95% CI of the difference [0.08, 0.10]). NA was more predictable compared to PA as evidenced by the RQA measures ( $NA_{\%REC}$  vs.  $PA_{\%REC}$ ,  $t(1,298) = 12.41, p < .001$ , 95% CI of the difference [6.73, 9.25];  $NA_{\%DET}$  vs.  $PA_{\%DET}$ ,  $t(1,298) = 11.52, p < .001$ , 95% CI of the difference [6.18, 8.72]). Interestingly,  $NA_{MEAN}$  and affect variability (i.e.,  $NA_{SD}$  and  $PA_{SD}$ ) were all positively associated while  $NA_{MEAN}$  was negatively associated with affect predictability (i.e.,  $NA_{\%REC}$ ,  $PA_{\%REC}$ ,  $NA_{\%DET}$ , and  $PA_{\%DET}$ ). In other words, individuals higher in NA were more likely to have variable affect but less likely to have predictable affect. On the other hand,  $PA_{MEAN}$  was positively associated with the affect predictability (i.e.,  $NA_{\%REC}$ ,  $PA_{\%REC}$ ,  $NA_{\%DET}$ , and  $PA_{\%DET}$ ) and negatively associated with affect variability (i.e.,  $NA_{SD}$  and  $PA_{SD}$ ). These PA findings indicate that individuals higher in PA have more predictable affect but less variable affect.

All associations between mean levels of affect and the outcome variables were consistent with previous literature. Specifically, greater  $NA_{MEAN}$  was associated with more depressive symptoms, worse well-being, and greater somatic symptoms (see first row in tables 4, 6, and 8).  $PA_{MEAN}$  was associated with fewer depressive symptoms, better well-being, and fewer somatic symptoms (see first row in tables 5, 7, and 9).

Table 3

*Mean, Standard Deviation, and Pearson's Correlation of Affect Metrics.*

	Mean	SD	NA <sub>SD</sub>	NA <sub>%REC</sub>	NA <sub>%DET</sub>	PA <sub>MEAN</sub>	PA <sub>SD</sub>	PA <sub>%REC</sub>	PA <sub>%DET</sub>
NA <sub>MEAN</sub>	1.67	0.47	0.64***	-0.59***	-0.52***	-0.36***	0.17***	-0.12***	-0.12***
NA <sub>SD</sub>	0.41	0.22		-0.76***	-0.66***	-0.24***	0.44***	-0.32***	-0.30***
NA <sub>%REC</sub>	58.36	21.11			0.84***	0.28***	-0.34***	0.28***	0.25***
NA <sub>%DET</sub>	71.67	19.03				0.26***	-0.29***	0.23***	0.22***
PA <sub>MEAN</sub>	3.00	0.50					-0.12***	0.09***	0.06*
PA <sub>SD</sub>	0.50	0.18						-0.75***	-0.63***
PA <sub>%REC</sub>	50.38	17.25							0.80***
PA <sub>%DET</sub>	64.22	18.32							

*Note.* SD = Standard Deviation; %REC = percent recurrence; %DET = percent determinism. \* $p < 0.05$ , \*\* $p < 0.01$ , \*\*\* $p < 0.001$

**Main effects of affect variability and predictability.** For affect variability, greater amounts of both  $NA_{SD}$  and  $PA_{SD}$  were associated with more depressive symptoms ( $NA_{SD}$ :  $b = 0.19$ ,  $z = 4.59$ ,  $p < .001$ , 95% CI [0.11, 0.27];  $PA_{SD}$ :  $b = 0.31$ ,  $z = 7.64$ ,  $p < .001$ , 95% CI [0.23, 0.39]), worse well-being ( $NA_{SD}$   $b = 0.07$ ,  $z = 2.01$ ,  $p = .045$ , 95% CI [0.00, 0.14]), and more somatic symptoms ( $NA_{SD}$ :  $b = 0.22$ ,  $z = 3.53$ ,  $p < .001$ , 95% CI [0.10, 0.34];  $PA_{SD}$ :  $b = 0.48$ ,  $z = 8.17$ ,  $p < .001$ , 95% CI [0.36, 0.59]; see Tables 4 through 9 model 1). The only exception to this was that higher  $PA_{SD}$  was associated with better well-being ( $b = -0.07$ ,  $z = -2.09$ ,  $p = .037$ , 95% CI [-0.14, -0.00]). However, it is important to note that both  $NA_{SD}$  and  $PA_{SD}$  were only weakly associated with well-being (while their associations with depressive and somatic symptoms were much stronger [see coefficient values]).

For recurrence metrics (%REC), higher  $NA_{\%REC}$  was associated with fewer depressive symptoms ( $b = -0.29$ ,  $z = -4.85$ ,  $p < .001$ , 95% CI [-0.41, -0.17]), higher well-being ( $b = -0.13$ ,  $z = -2.54$ ,  $p = .011$ , 95% CI [-0.22, -0.03]), and fewer somatic symptoms ( $b = -0.28$ ,  $z = -3.31$ ,  $p = .001$ , 95% CI [-0.45, -0.11]; see Tables 4, 6, and 8 model 3).  $PA_{\%REC}$ , on the other hand, was only associated with depressive symptoms ( $b = -0.19$ ,  $z = -2.81$ ,  $p = .005$ , 95% CI [-0.32, -0.06]) whereby greater PA predictability was associated with fewer depressive symptoms (see Table 5 model 3).  $PA_{\%REC}$  was not associated with well-being ( $b = 0.07$ ,  $z = 1.36$ ,  $p = .175$ , 95% CI [-0.03, 0.18]) or somatic symptoms ( $b = -0.10$ ,  $z = -1.00$ ,  $p = .319$ , 95% CI [-0.29, 0.09]; see Tables 7 and 9 model 3). Although all models were built on the model with the interaction between mean level and standard deviation of affect (i.e., model 2), all recurrence associations with the dependent variables remained the same when removing the interaction term and only controlling for mean and standard deviation. However, when only controlling for mean affect (and not for affect variability),  $PA_{\%REC}$  became associated with well-being ( $b = 0.09$ ,  $z = 2.44$ ,  $p$

= .015, 95% CI [0.02, 0.16]) and somatic symptoms ( $b = -0.43$ ,  $z = -6.61$ ,  $p < .001$ , 95% CI [-0.56, -0.30]) while all other associations remained the same. It is likely these associations became significant when removing  $PA_{SD}$  from the model because of the correlation between  $PA_{REC}$  and  $PA_{SD}$ .

For determinism metrics (%DET), higher  $NA_{DET}$  was associated with fewer depressive symptoms ( $b = -0.16$ ,  $z = -3.25$ ,  $p = .001$ , 95% CI [-0.26, -0.06]) and fewer somatic symptoms ( $b = -0.25$ ,  $z = -3.31$ ,  $p = .001$ , 95% CI [-0.39, -0.10]) but not well-being ( $b = -0.01$ ,  $z = -0.12$ ,  $p = .903$ , 95% CI [-0.09, 0.08]; see Tables 4, 6, and 8 model 6). Higher  $PA_{DET}$ , on the other hand, was associated with worse well-being ( $b = 0.09$ ,  $z = 2.11$ ,  $p = .035$ , 95% CI [0.01, 0.18]) but was not associated with depressive ( $b = 0.00$ ,  $z = 0.08$ ,  $p = .938$ , 95% CI [-0.10, 0.11]) and somatic symptoms ( $b = 0.10$ ,  $z = 1.40$ ,  $p = .162$ , 95% CI [-0.04, 0.25]). Although all models were built on the model with the interaction between mean level and standard deviation of affect (i.e., model 2), all determinism associations with the dependent variables remained the same when removing the interaction term and only controlling for mean and standard deviation. However, when only controlling for mean affect,  $PA_{DET}$  became associated with depressive symptoms ( $b = -0.19$ ,  $z = -4.63$ ,  $p < .001$ , 95% CI [-0.27, -0.11]) and somatic symptoms ( $b = -0.23$ ,  $z = -3.90$ ,  $p < .001$ , 95% CI [-0.34, -0.11]) while all other associations remained the same. It is likely these associations became significant when removing  $PA_{SD}$  from the model because of the correlation between  $PA_{DET}$  and  $PA_{SD}$ .



Table 4

*Variability Metrics of Negative Affect Predicting Depressive Symptoms.*

VARIABLES	1	2	3	4	5	6	7	8	9
NA <sub>MEAN</sub>	0.56***	0.61***	0.59***	0.59***	0.59***	0.60***	0.60***	0.58***	0.59***
NA <sub>SD</sub>	0.19***	0.32***	0.13*	0.11	0.12	0.24***	0.21***	0.23***	0.11
NA <sub>MEAN</sub> *NA <sub>SD</sub>		-0.69***	-0.61***	-0.37***	-0.36**	-0.66***	-0.55***	-0.51***	-0.36**
NA <sub>%REC</sub>			-0.29***	-0.29***	-0.29***				-0.23*
NA <sub>MEAN</sub> *NA <sub>%REC</sub>				0.34**	0.33*				0.69***
NA <sub>SD</sub> *NA <sub>%REC</sub>					0.06				-0.39
NA <sub>MEAN</sub> *NA <sub>SD</sub> *NA <sub>%REC</sub>					0.01				-0.92
NA <sub>%DET</sub>						-0.16**	-0.21***	-0.22***	-0.08
NA <sub>MEAN</sub> *NA <sub>%DET</sub>							0.20	0.08	-0.44**
NA <sub>SD</sub> *NA <sub>%DET</sub>								0.67**	0.50
NA <sub>MEAN</sub> *NA <sub>SD</sub> *NA <sub>%DET</sub>								-0.16	1.30
Distress	0.00	-0.01	-0.02	-0.02	-0.02	-0.01	-0.02	-0.02	-0.02
Constant	2.60***	2.66***	2.67***	2.67***	2.67***	2.66***	2.67***	2.68***	2.67***
Observations	1,301	1,301	1,301	1,301	1,301	1,301	1,301	1,301	1,301
AIC	10,761	10,640	10,619	10,613	10,617	10,632	10,630	10,625	10,614
BIC	10,781	10,666	10,650	10,649	10,664	10,663	10,666	10,672	10,681

Note. \* $p < 0.05$ , \*\* $p < 0.01$ , \*\*\* $p < 0.001$ . For model (not shown) with only NA<sub>MEAN</sub> and Distress, AIC = 10,780 and BIC = 10,795.

Table 5

*Variability Metrics of Positive Affect Predicting Depressive Symptoms.*

VARIABLES	1	2	3	4	5	6	7	8	9
PA <sub>MEAN</sub>	-0.48***	-0.48***	-0.48***	-0.48***	-0.48***	-0.48***	-0.48***	-0.47***	-0.48***
PA <sub>SD</sub>	0.31***	0.35***	0.22***	0.21***	0.32***	0.35***	0.34***	0.37***	0.32***
PA <sub>MEAN</sub> *PA <sub>SD</sub>		0.33***	0.34***	0.25*	0.23	0.33***	0.29**	0.30**	0.25
PA <sub>%REC</sub>			-0.19**	-0.20**	-0.08				-0.24*
PA <sub>MEAN</sub> *PA <sub>%REC</sub>				-0.14	-0.15				-0.15
PA <sub>SD</sub> *PA <sub>%REC</sub>					0.72**				1.25***
PA <sub>MEAN</sub> *PA <sub>SD</sub> *PA <sub>%REC</sub>					-0.06				-0.60
PA <sub>%DET</sub>						0.00	-0.00	-0.01	0.19*
PA <sub>MEAN</sub> *PA <sub>%DET</sub>							-0.07	-0.06	0.01
PA <sub>SD</sub> *PA <sub>%DET</sub>								0.48*	-0.74*
PA <sub>MEAN</sub> *PA <sub>SD</sub> *PA <sub>%DET</sub>								0.39	0.76
Distress	0.18***	0.18***	0.18***	0.18***	0.18***	0.18***	0.18***	0.18***	0.18***
Constant	2.37***	2.37***	2.37***	2.37***	2.39***	2.37***	2.37***	2.38***	2.38***
Observations	1,300	1,300	1,300	1,300	1,300	1,300	1,300	1,300	1,300
AIC	10,928	10,913	10,907	10,908	10,902	10,915	10,917	10,915	10,901
BIC	10,949	10,939	10,938	10,944	10,949	10,946	10,953	10,961	10,968

Note. \* $p < 0.05$ , \*\* $p < 0.01$ , \*\*\* $p < 0.001$ . For model (not shown) with only PA<sub>MEAN</sub> and Distress, AIC = 10,984 and BIC = 11,000.

Table 6

*Variability Metrics of Negative Affect Predicting Negative Well-Being.*

VARIABLES	1	2	3	4	5	6	7	8	9
NA <sub>MEAN</sub>	0.32***	0.34***	0.34***	0.34***	0.32***	0.34***	0.34***	0.33***	0.32***
NA <sub>SD</sub>	0.07*	0.11**	0.03	0.03	0.08	0.11*	0.11*	0.12**	0.06
NA <sub>MEAN</sub> *NA <sub>SD</sub>		-0.30***	-0.26***	-0.36***	-0.38**	-0.30***	-0.29***	-0.25**	-0.40**
NA <sub>%REC</sub>			-0.13*	-0.13**	-0.08				-0.18*
NA <sub>MEAN</sub> *NA <sub>%REC</sub>				-0.14	-0.27*				-0.39*
NA <sub>SD</sub> *NA <sub>%REC</sub>					0.42*				0.04
NA <sub>MEAN</sub> *NA <sub>SD</sub> *NA <sub>%REC</sub>					-0.28				0.30
NA <sub>%DET</sub>						-0.01	-0.01	-0.01	0.10
NA <sub>MEAN</sub> *NA <sub>%DET</sub>							0.03	-0.08	0.14
NA <sub>SD</sub> *NA <sub>%DET</sub>								0.59**	0.47
NA <sub>MEAN</sub> *NA <sub>SD</sub> *NA <sub>%DET</sub>								-0.34	-0.85
Distress	-0.06***	-0.07***	-0.07***	-0.07***	-0.07***	-0.07***	-0.07***	-0.07***	-0.08***
Constant	3.10***	3.13***	3.13***	3.13***	3.14***	3.13***	3.13***	3.14***	3.14***
Observations	1,283	1,283	1,283	1,283	1,283	1,283	1,283	1,283	1,283
AIC	9,698	9,670	9,665	9,666	9,664	9,672	9,674	9,668	9,665
BIC	9,719	9,695	9,696	9,702	9,711	9,703	9,710	9,714	9,732

Note. \* $p < 0.05$ , \*\* $p < 0.01$ , \*\*\* $p < 0.001$ . For model (not shown) with only NA<sub>MEAN</sub> and Distress, AIC = 9,700 and BIC = 9,716.

Table 7

*Variability Metrics of Positive Affect Predicting Negative Well-Being.*

VARIABLES	1	2	3	4	5	6	7	8	9
PA <sub>MEAN</sub>	-0.40***	-0.40***	-0.40***	-0.40***	-0.38***	-0.40***	-0.40***	-0.39***	-0.38***
PA <sub>SD</sub>	-0.07*	-0.08*	-0.03	-0.03	0.00	-0.02	-0.02	-0.03	-0.00
PA <sub>MEAN</sub> *PA <sub>SD</sub>		-0.11	-0.11	-0.01	0.08	-0.11	-0.07	-0.07	0.10
PA <sub>%REC</sub>			0.07	0.07	0.12				0.01
PA <sub>MEAN</sub> *PA <sub>%REC</sub>				0.13	0.29*				0.41*
PA <sub>SD</sub> *PA <sub>%REC</sub>					0.25				0.88**
PA <sub>MEAN</sub> *PA <sub>SD</sub> *PA <sub>%REC</sub>					0.76				-0.05
PA <sub>%DET</sub>						0.09*	0.10*	0.11*	0.14*
PA <sub>MEAN</sub> *PA <sub>%DET</sub>							0.06	0.07	-0.14
PA <sub>SD</sub> *PA <sub>%DET</sub>								-0.14	-0.86**
PA <sub>MEAN</sub> *PA <sub>SD</sub> *PA <sub>%DET</sub>								0.66	1.21*
Distress	0.02	0.02	0.02*	0.02	0.02*	0.02*	0.02*	0.02*	0.02*
Constant	2.98***	2.97***	2.97***	2.97***	2.98***	2.97***	2.97***	2.97***	2.97***
Observations	1,282	1,282	1,282	1,282	1,282	1,282	1,282	1,282	1,282
AIC	9,191	9,190	9,190	9,191	9,190	9,188	9,189	9,188	9,181
BIC	9,211	9,216	9,221	9,227	9,236	9,219	9,225	9,235	9,248

Note. \* $p < 0.05$ , \*\* $p < 0.01$ , \*\*\* $p < 0.001$ . For model (not shown) with only PA<sub>MEAN</sub> and Distress, AIC = 9,193 and BIC = 9,208.

Table 8

*Variability Metrics of Negative Affect Predicting Somatic Symptoms.*

VARIABLES	1	2	3	4	5	6	7	8	9
NA <sub>MEAN</sub>	0.13***	0.13***	0.11***	0.11***	0.11**	0.12***	0.11***	0.10**	0.10**
NA <sub>SD</sub>	0.22***	0.24***	0.05	0.04	0.02	0.12	0.07	0.06	0.04
NA <sub>MEAN</sub> *NA <sub>SD</sub>		-0.15	-0.05	0.32*	0.20	-0.10	0.11	0.06	0.21
NA <sub>%REC</sub>			-0.28***	-0.25**	-0.23*				-0.02
NA <sub>MEAN</sub> *NA <sub>%REC</sub>				0.55**	0.50*				0.52
NA <sub>SD</sub> *NA <sub>%REC</sub>					-0.10				0.53
NA <sub>MEAN</sub> *NA <sub>SD</sub> *NA <sub>%REC</sub>					-0.42				-1.63
NA <sub>%DET</sub>						-0.25***	-0.31***	-0.27***	-0.24
NA <sub>MEAN</sub> *NA <sub>%DET</sub>							0.40*	0.42*	-0.02
NA <sub>SD</sub> *NA <sub>%DET</sub>								0.03	-0.77
NA <sub>MEAN</sub> *NA <sub>SD</sub> *NA <sub>%DET</sub>								-0.68	1.65
Distress	0.16***	0.16***	0.15***	0.15***	0.15***	0.15***	0.15***	0.15***	0.15***
Constant	1.69***	1.70***	1.71***	1.72***	1.72***	1.70***	1.71***	1.72***	1.72***
Observations	1,290	1,290	1,290	1,290	1,290	1,290	1,290	1,290	1,290
AIC	6,291	6,291	6,282	6,276	6,279	6,282	6,277	6,280	6,280
BIC	6,312	6,317	6,313	6,312	6,325	6,313	6,314	6,326	6,347

Note. \* $p < 0.05$ , \*\* $p < 0.01$ , \*\*\* $p < 0.001$ . For model (not shown) with only NA<sub>MEAN</sub> and Distress, AIC = 6,302 and BIC = 6,317.

Table 9

*Variability Metrics of Positive Affect Predicting Somatic Symptoms.*

VARIABLES	1	2	3	4	5	6	7	8	9
PA <sub>MEAN</sub>	-0.19***	-0.19***	-0.19***	-0.19***	-0.20***	-0.19***	-0.19***	-0.17***	-0.19***
PA <sub>SD</sub>	0.48***	0.48***	0.42***	0.42***	0.46***	0.55***	0.55***	0.57***	0.46***
PA <sub>MEAN</sub> *PA <sub>SD</sub>		0.16	0.16	0.11	0.07	0.15	0.13	0.14	0.07
PA <sub>%REC</sub>			-0.10	-0.10	-0.05				-0.24
PA <sub>MEAN</sub> *PA <sub>%REC</sub>				-0.07	-0.11				-0.06
PA <sub>SD</sub> *PA <sub>%REC</sub>					0.28				-0.33
PA <sub>MEAN</sub> *PA <sub>SD</sub> *PA <sub>%REC</sub>					-0.25				-1.77
PA <sub>%DET</sub>						0.10	0.10	0.10	0.20
PA <sub>MEAN</sub> *PA <sub>%DET</sub>							-0.03	-0.02	-0.08
PA <sub>SD</sub> *PA <sub>%DET</sub>								0.65*	0.64
PA <sub>MEAN</sub> *PA <sub>SD</sub> *PA <sub>%DET</sub>								0.83	2.17*
Distress	0.19***	0.19***	0.19***	0.19***	0.19***	0.19***	0.19***	0.19***	0.19***
Constant	1.64***	1.65***	1.65***	1.65***	1.65***	1.64***	1.64***	1.66***	1.65***
Observations	1,289	1,289	1,289	1,289	1,289	1,289	1,289	1,289	1,289
AIC	6,197	6,197	6,198	6,200	6,203	6,197	6,199	6,197	6,198
BIC	6,218	6,223	6,229	6,236	6,250	6,228	6,236	6,243	6,266

Note. \* $p < 0.05$ , \*\* $p < 0.01$ , \*\*\* $p < 0.001$ . For model (not shown) with only PA<sub>MEAN</sub> and Distress, AIC = 6,261 and BIC = 6,276.

### Interactions between variability/predictability metrics and mean levels. $NA_{SD}$

interacted with  $NA_{MEAN}$  to predict depressive symptoms ( $b = -0.69$ ,  $z = -10.63$ ,  $p < .001$ , 95% CI [-0.81, -0.56]) and well-being ( $b = -0.30$ ,  $z = -5.43$ ,  $p < .001$ , 95% CI [-0.41, -0.19]) but not somatic symptoms ( $b = -0.15$ ,  $z = -1.58$ ,  $p = .114$ , 95% CI [-0.34, 0.04]; see Tables 4, 6, and 8 model 2). Specifically, at higher levels of  $NA_{MEAN}$ , more NA variability (i.e., higher  $NA_{SD}$ ) was associated with fewer depressive symptoms (see Figure 4a) and higher well-being (see Figure 4b).  $PA_{SD}$  interacted with  $PA_{MEAN}$  to predict depressive symptoms ( $b = 0.33$ ,  $z = 4.17$ ,  $p < .001$ , 95% CI [0.18, 0.49]) but not well-being ( $b = -0.11$ ,  $z = -1.60$ ,  $p = .110$ , 95% CI [-0.24, 0.02]) or somatic symptoms ( $b = 0.16$ ,  $z = 1.36$ ,  $p = .175$ , 95% CI [-0.07, 0.38]; see Tables 5, 7, and 9 model 2). At higher levels of  $PA_{MEAN}$ , less PA variability (i.e., higher  $PA_{SD}$ ) was associated with fewer depressive symptoms (see Figure 5).

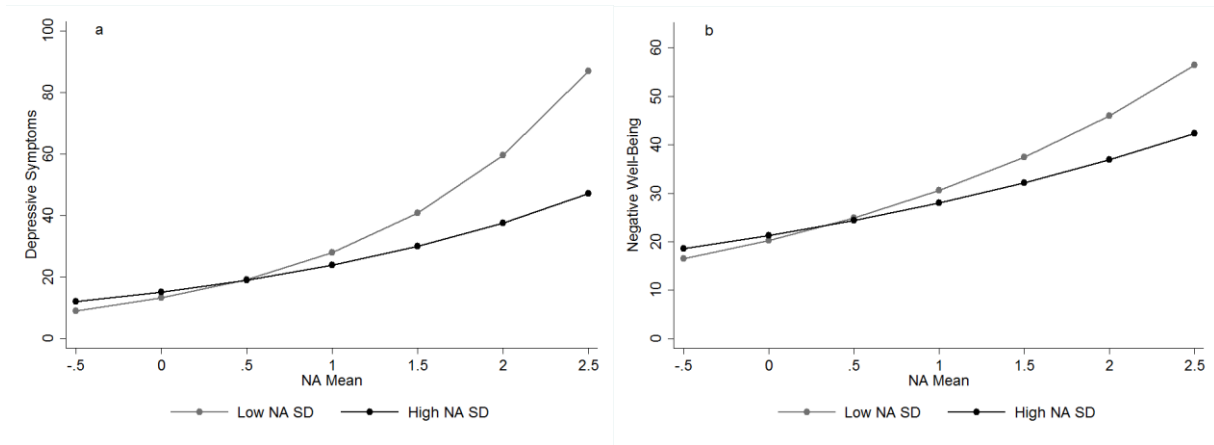


Figure 4. Interaction between  $NA_{MEAN}$  and  $NA_{SD}$  predicting depressive symptoms and negative well-being.

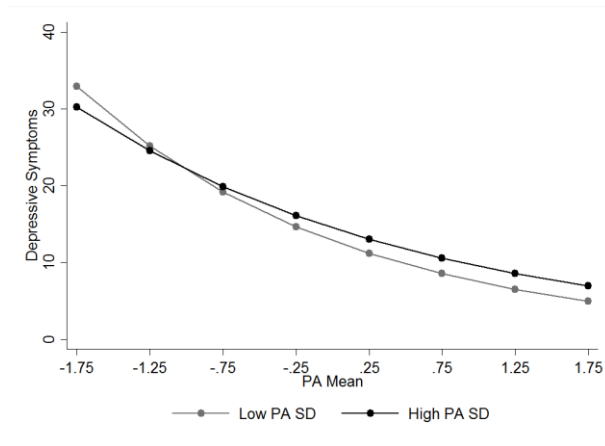


Figure 5. Interaction between  $PA_{MEAN}$  and  $PA_{SD}$  predicting depressive symptoms.

$NA_{\%REC}$  interacted with  $NA_{MEAN}$  to predict depressive symptoms ( $b = 0.34$ ,  $z = 2.76$ ,  $p = .006$ , 95% CI [0.10, 0.59]) and somatic symptoms ( $b = 0.55$ ,  $z = 2.89$ ,  $p = .004$ , 95% CI [0.18, 0.92]) but not well-being ( $b = -0.14$ ,  $z = -1.30$ ,  $p = .194$ , 95% CI [-0.36, 0.07]); see Tables 4, 6, and 8 model 4). Specifically, at higher levels of  $NA_{MEAN}$ , less NA predictability (i.e., lower  $NA_{\%REC}$ ) was associated with fewer depressive symptoms (see Figure 6a) and fewer somatic symptoms (see Figure 6b).  $PA_{\%REC}$  did not interact with  $PA_{MEAN}$  to predict depressive symptoms ( $b = -0.14$ ,  $z = -1.04$ ,  $p = .297$ , 95% CI [-0.39, 0.12]), well-being ( $b = 0.13$ ,  $z = 1.28$ ,  $p = .201$ , 95% CI [-0.07, 0.34]), or somatic symptoms ( $b = -0.07$ ,  $z = -0.39$ ,  $p = .697$ , 95% CI [-0.45, 0.30]); see Tables 5, 7, and 9 model 4). All models were built on the model with the interaction between mean level and standard deviation of affect and the main effect of %REC (i.e., model 3). When removing the mean level by standard deviation interaction from the analyses<sup>5</sup>, the interaction between  $PA_{\%REC}$  and  $PA_{MEAN}$  became significant for depressive symptoms ( $b = -0.33$ ,  $z = -3.93$ ,  $p < .001$ , 95% CI [-0.50, -0.17]) and well-being ( $b = 0.14$ ,  $z = 2.07$ ,  $p = .038$ , 95% CI [0.01, 0.28]), but not somatic symptoms ( $b = -0.16$ ,  $z = -1.31$ ,  $p = .191$ , 95% CI [-0.41, 0.08]).

<sup>5</sup> The pattern of results reported in these analyses is the same when the interaction term is removed (but the main effect of standard deviation is kept in) and when standard deviation is removed from the model completely.



Additionally, the interaction between  $NA_{\%REC}$  and  $NA_{MEAN}$  became significant for well-being ( $b = 0.18, z = 2.76, p = .006, 95\% \text{ CI } [0.05, 0.31]$ ). All other findings remained significant.

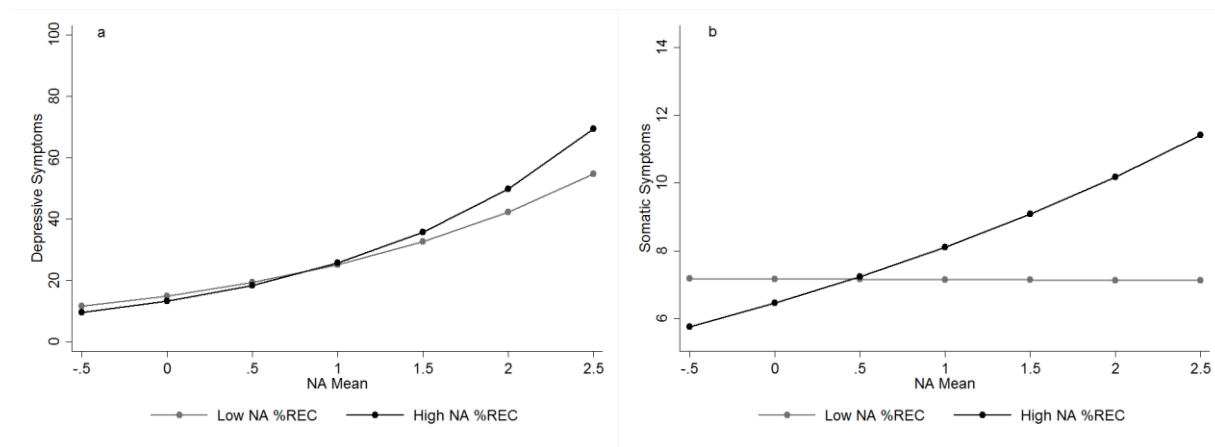


Figure 6. Interaction between  $NA_{MEAN}$  and  $NA_{\%REC}$  predicting depressive symptoms and somatic symptoms.

$NA_{\%DET}$  interacted with  $NA_{MEAN}$  to predict somatic symptoms ( $b = 0.40, z = 2.55, p = .011, 95\% \text{ CI } [0.09, 0.70]$ ) and, marginally, depressive symptoms ( $b = 0.20, z = 1.96, p = .051, 95\% \text{ CI } [-0.00, 0.39]$ ) but not well-being ( $b = 0.03, z = 0.31, p = .755, 95\% \text{ CI } [-0.15, 0.21]$ ); see Tables 4, 6, and 8 model 7). Specifically, at higher levels of  $NA_{MEAN}$ , less NA predictability (i.e., lower  $NA_{\%DET}$ ) was associated with marginally fewer depressive symptoms (see Figure 7a) and fewer somatic symptoms (see Figure 7b).  $PA_{\%DET}$  did not interact with  $PA_{MEAN}$  to predict depressive symptoms ( $b = -0.07, z = -0.70, p = .483, 95\% \text{ CI } [-0.27, 0.13]$ ), well-being ( $b = 0.06, z = 0.74, p = .462, 95\% \text{ CI } [-0.10, 0.23]$ ), or somatic symptoms ( $b = -0.03, z = -0.21, p = .835, 95\% \text{ CI } [-0.32, 0.26]$ ); see Tables 5, 7, and 9 model 7). All models were built on the model with the interaction between mean level and standard deviation of affect and the main effect of %DET (i.e., model 6). When removing the mean level by standard deviation interaction from the

analyses<sup>6</sup>, the interaction between PA<sub>%DET</sub> and PA<sub>MEAN</sub> became significant for depressive symptoms (PA<sub>%REC</sub>:  $b = -0.25$ ,  $z = -3.05$ ,  $p = .002$ , 95% CI [-0.41, -0.09]) but not well-being ( $b = 0.11$ ,  $z = 1.57$ ,  $p = .117$ , 95% CI [-0.03, 0.24]) or somatic symptoms ( $b = -0.11$ ,  $z = -0.94$ ,  $p = .350$ , 95% CI [-0.34, 0.12]). Additionally, the interaction between NA<sub>%DET</sub> and NA<sub>MEAN</sub> became significant for depressive symptoms (NA<sub>%REC</sub>:  $b = 0.63$ ,  $z = 8.29$ ,  $p < .001$ , 95% CI [0.48, 0.78]) and well-being ( $b = 0.26$ ,  $z = 3.86$ ,  $p < .001$ , 95% CI [0.13, 0.39]). All other findings remained significant.

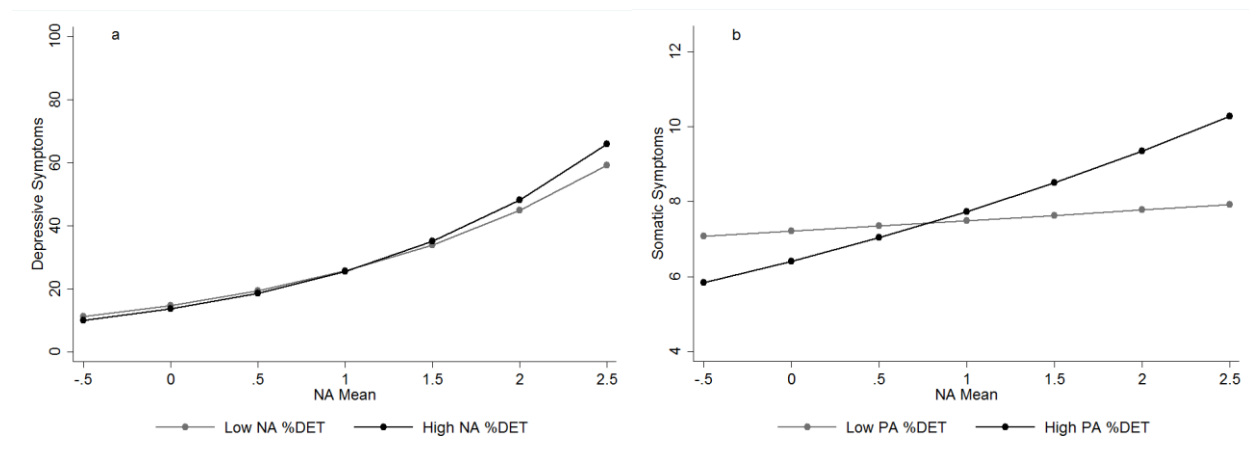


Figure 7. Interaction between NA<sub>MEAN</sub> and NA<sub>%DET</sub> predicting depressive and somatic symptoms.

**Interactions between variability and predictability.** Variability did interact with predictability on some occasions. NA<sub>%REC</sub> interacted with NA<sub>SD</sub> to predict well-being ( $b = 0.42$ ,  $z = 2.00$ ,  $p = .046$ , 95% CI [0.01, 0.83]) but not depressive symptoms ( $b = 0.06$ ,  $z = 0.24$ ,  $p = .808$ , 95% CI [-0.41, 0.52]) or somatic symptoms ( $b = -0.10$ ,  $z = -0.27$ ,  $p = .787$ , 95% CI [-0.79, 0.60]; see Tables 4, 6, and 8 model 5). At higher levels of NA<sub>SD</sub>, more NA predictability (i.e., higher NA<sub>%REC</sub>) was associated with worse well-being (see Figure 8). PA<sub>%REC</sub> interacted with

<sup>6</sup> The pattern of results reported in these analyses is the same when the interaction term is removed (but the main effect of standard deviation is kept in) and when standard deviation is removed from the model completely.

PA<sub>SD</sub> to predict depressive symptoms ( $b = 0.72, z = 3.01, p = .003, 95\% \text{ CI } [0.25, 1.19]$ ) but not well-being ( $b = 0.25, z = 1.30, p = .195, 95\% \text{ CI } [-0.13, 0.63]$ ) or somatic symptoms ( $b = 0.28, z = 0.84, p = .403, 95\% \text{ CI } [-0.38, 0.94]$ ; see Tables 5, 7, and 9 model 5). Specifically, at higher levels of PA<sub>SD</sub>, more PA predictability (i.e., higher PA<sub>%REC</sub>) was associated with more depressive symptoms (see Figure 9).

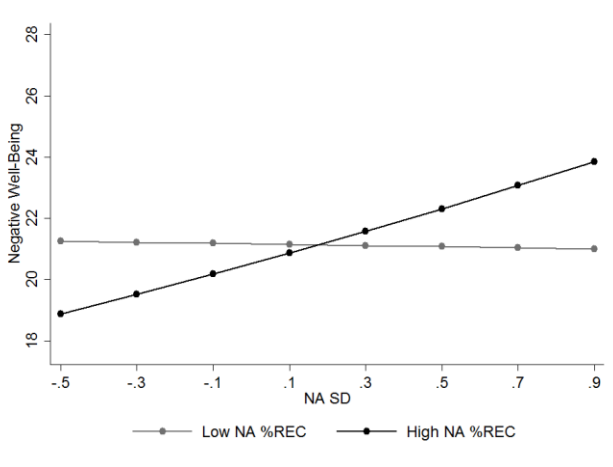


Figure 8. Interaction between NA<sub>SD</sub> and NA<sub>%REC</sub> predicting negative well-being.

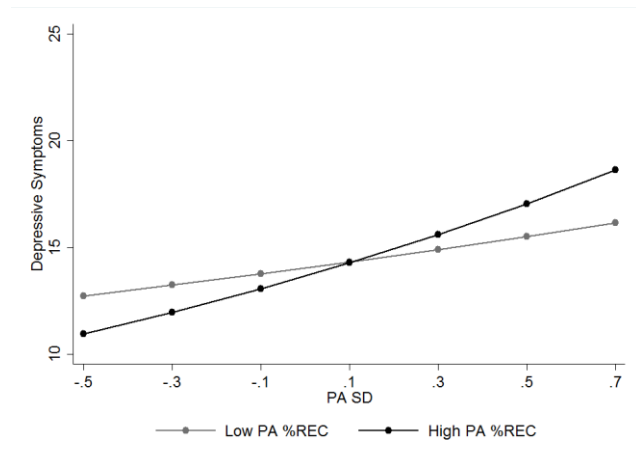


Figure 9. Interaction between PA<sub>SD</sub> and PA<sub>%REC</sub> predicting depressive symptoms.

NA<sub>%DET</sub> interacted with NA<sub>SD</sub> to predict depressive symptoms ( $b = 0.67, z = 2.96, p = .003, 95\% \text{ CI } [0.23, 1.11]$ ) and well-being ( $b = 0.59, z = 3.08, p = .002, 95\% \text{ CI } [0.22, 0.97]$ ) but not somatic symptoms ( $b = 0.03, z = 0.10, p = .918, 95\% \text{ CI } [-0.63, 0.70]$ ; see Tables 4, 6, and 8

model 8). At higher levels of  $NA_{SD}$ , more NA predictability (i.e., higher  $NA_{\%DET}$ ) was associated more depressive symptoms (see Figure 10a) and lower well-being (see Figure 10b).  $PA_{\%DET}$  interacted with  $PA_{SD}$  to predict depressive symptoms ( $b = 0.48, z = 2.37, p = .018, 95\% CI [0.08, 0.87]$ ) and somatic symptoms ( $b = 0.65, z = 2.24, p = .025, 95\% CI [0.08, 1.22]$ ) but not well-being ( $b = -0.14, z = -0.85, p = .396, 95\% CI [-0.47, 0.19]$ ; see Tables 5, 7, and 9 model 8). At higher levels of  $PA_{SD}$ , more PA predictability (i.e., higher  $PA_{\%DET}$ ) was associated with more depressive symptoms (see Figure 11a) and more somatic symptoms (see Figure 11b).

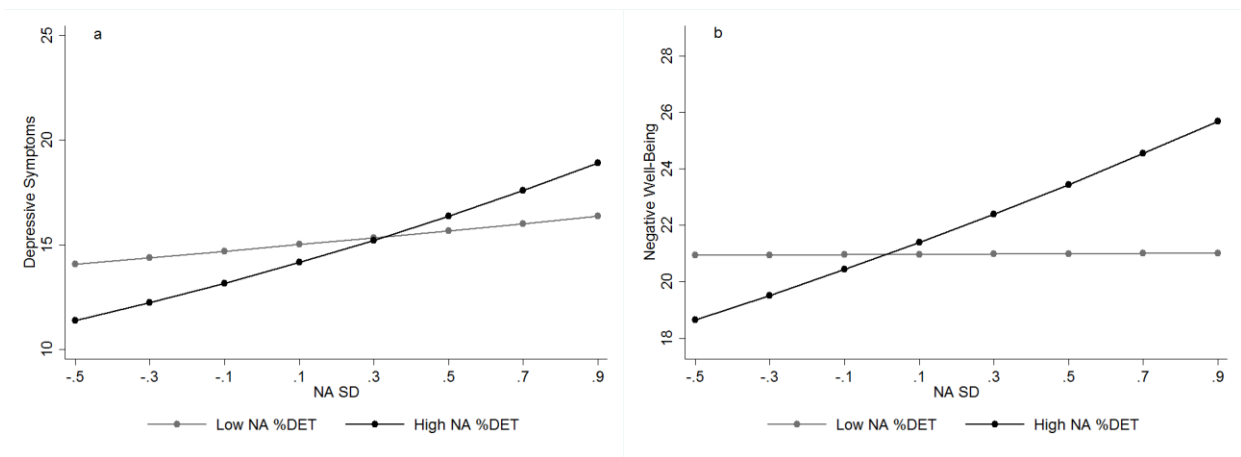


Figure 10. Interaction between  $NA_{\%DET}$  and  $NA_{SD}$  predicting depressive symptoms and negative well-being.

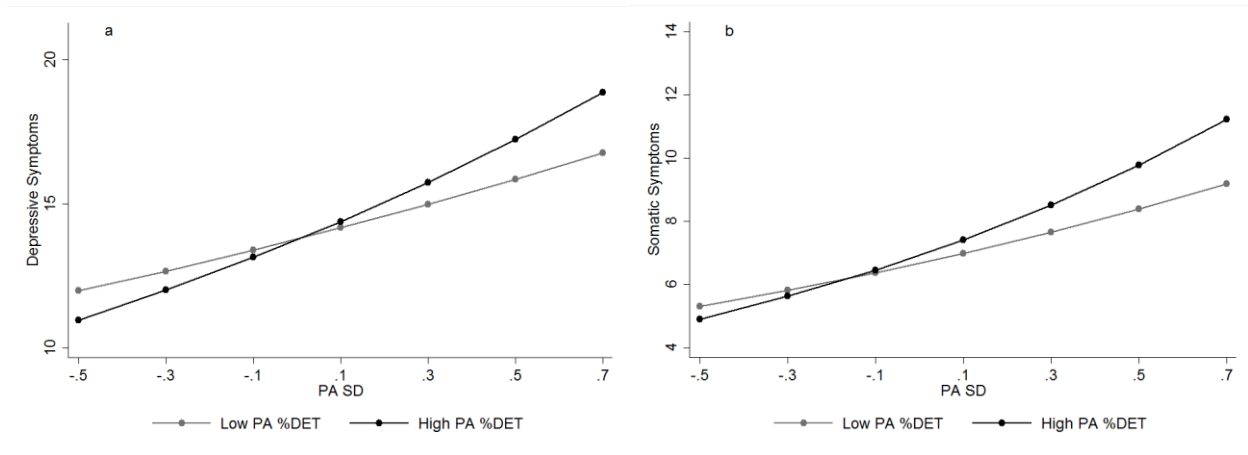


Figure 11. Interaction between PA<sub>%DET</sub> and PA<sub>SD</sub> predicting depressive symptoms and somatic symptoms.

**Three-way interactions between variability, predictability, and mean.** In models 5 and 8, there were no three-way interactions between SD, RQA, and mean levels (all  $ps > .05$ ; see Tables 4 through 9). However, in Model 9 when all predictors were placed in the same model, 2 three-way interactions became significant (see Tables 7 and 9). At low levels of PA<sub>MEAN</sub>, more predictability (i.e., more PA<sub>%DET</sub>) was associated with lower psychological well-being but only when variability (i.e., PA<sub>SD</sub>) was lower ( $b = 1.21, z = 1.99, p = .047, 95\% \text{ CI } [0.02, 2.41]$ ; see Figure 12a). At low levels of PA<sub>MEAN</sub>, less predictability (i.e., less PA<sub>%DET</sub>) was associated with fewer somatic symptoms but only when variability was low (i.e., low PA<sub>SD</sub>;  $b = 2.17, z = 2.21, p = .027, 95\% \text{ CI } [0.24, 4.10]$ ; see Figure 12b).

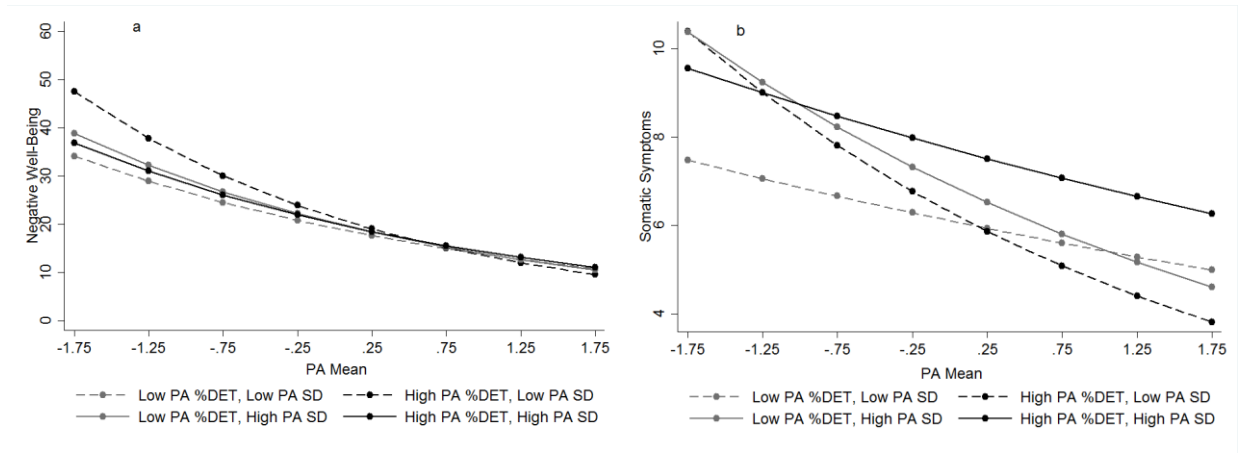


Figure 12. Three-way interaction between  $PA_{MEAN}$ ,  $PA_{SD}$ , and  $PA_{\%DET}$  predicting negative well-being and somatic symptoms.

### Discussion

This study shows for the first time that measures of affect predictability add important new information in regard to well-being, and depressive and somatic symptoms. Critically, measures of predictability differ from a measure of variability (i.e., standard deviation) in their association with affect valence. For example, this study revealed that individuals with higher average NA had more variable but less predictable affect. Conversely, those with greater mean PA had less variable but more predictable affect. This implies that individuals high in NA generally have greater and more erratic fluctuations in affect compared to those who have low NA. Additionally, individuals high in PA generally have less intense and more stable fluctuations in affect compared to those who have low PA.

These factors of variability and predictability are not only associated with PA and NA differentially, but they also have different associations with health outcomes. In general, more variable affect was associated with worse outcomes (i.e., more depressive symptoms, lower well-being, more somatic symptoms) consistent with past studies on similar topics (Gruber et al., 2013; Hardy & Segerstrom, 2016). On the other hand, our newly studied predictability metrics

revealed that affect patterns that are more expected are associated with better outcomes (i.e., fewer depressive symptoms, higher well-being, and fewer somatic symptoms). When we consider the nature of repeated patterns of affect (i.e., RQA metrics) in addition to measures of variability (i.e., standard deviation), the conclusions drawn about how affect changes wellness outcomes are altered. Assessing variability on its own is important, but additionally assessing predictability provides a clearer and more interesting picture about how fluctuations in affect influence mental and physical health.

Drawing on the discrepancies found between predictability and variability, it is also informative to examine how these factors interact with mean levels of affect to predict those same health outcomes. Although variability generally resulted in less favorable outcomes, at higher levels of mean NA, higher NA variability actually became associated with better outcomes. For example, individuals with higher mean levels of NA had fewer depressive symptoms when they had more variable NA. It is possible that more variation for those high in mean NA allows individuals to have some “breaks” from their typical high levels of negativity. For PA, variability made less of a difference but had the reverse effect whereby at higher levels of mean PA, more variability was associated with worse outcomes. For these individuals, greater variability means that they are frequently dropping below their normally high positive state of being, so those “breaks” from normality are undesirable. In these ways, variability has different effects depending on the valence and mean level of affect.

Regarding the RQA measures of %REC and %DET, as hypothesized, more predictability was generally associated with better outcomes. However, higher levels of predictability were associated with worse outcomes for those with high levels of NA. High predictability of NA for those already high in NA could signify that a person is “stuck” in a negative situation that they

are continually experiencing. Someone who feels poorly now and expects to continue feeling poorly in the future will likely exhibit the least desirable outcomes. Interestingly, PA predictability did not interact with mean PA levels when controlling for standard deviation. However, if standard deviation is not controlled for, then the interaction between PA mean and predictability becomes significant. This implies that regarding PA, the RQA metrics do not provide much more information beyond what is captured by standard deviation. It appears then, that individuals high in PA tend to have positive outcomes and those low in PA tend to have negative outcomes, regardless of the predictability of their affect variation. Nonetheless, these results taken together indicate that predictability is generally beneficial except in the case of high mean NA. It also highlights the importance in the affect and health literature of considering both PA and NA separately and the possibility that they operate on well-being in different manners.

How are those same outcomes altered when we examine the combined influence of variability, predictability, and mean levels? At higher levels of variability, more predictability was associated with worse outcomes regardless of affect type (i.e., NA vs. PA) or mean level. For individuals low in variability with low  $PA_{MEAN}$ , more predictability (i.e., more  $PA_{\%DET}$ ) was associated with lower psychological well-being. These individuals have continually low levels of PA and will likely not increase those levels, so their psychological well-being stays low as well. On the other hand, if those same individuals with low variability and low  $PA_{MEAN}$  have less predictability (i.e., less  $PA_{\%DET}$ ), then they exhibit fewer somatic symptoms (possibly because they expect to feel better soon). Overall, when  $PA_{MEAN}$  is low, the combination of high predictability and low variability is detrimental; and when  $PA_{MEAN}$  is high, then high predictability and high variability are least desirable.



There are a number of limitations in this work that need to be addressed. First, we are unable to make causal conclusions about how affect is related to health, which leaves open the possibility of reverse causation. More variability could conceivably lead to higher levels of depressive symptoms, but it is also possible that having high levels of depressive symptoms could lead to more variable affect. Similarly, high somatic symptoms or low well-being could have led to changes in affect variability and predictability. As in other observational studies on affect, our study design does not allow us to answer these types of directional questions. Nevertheless, this study adds substantially to the affect literature by demonstrating that predictability plays at least some role in the affect-health association. Additionally, it must be acknowledged that variability and predictability are highly correlated. This high correlation partially accounts for why there were sometimes no significant effects of PA predictability. However, these metrics are not perfectly correlated, which allows the RQA metrics to further differentiate certain cases and provide additional information. Finally, the results of our study are not generalizable to the population at large because of our limited study sample. The participants were primarily Caucasian undergraduates, so our conclusions only apply to these types of individuals. However, these methods may be extended to other populations and so future research may address this gap.

Furthermore, it is worth mentioning why the results were less consistent amongst the well-being models compared to depressive and somatic symptom models. For example, in the case of the NA results, there was no main effect of  $NA_{\%DET}$  or interaction between mean affect and predictability when well-being was the dependent variable. However, there were main effects of  $NA_{SD}$  and  $NA_{\%REC}$  as well as a significant interaction between  $NA_{MEAN}$  and  $NA_{SD}$  when well-being was the dependent variable. In contrast, the PA results were consistently non-

significant or in the opposite direction as other findings when well-being was the dependent variable. Examining goodness of fit measures, the BIC for the model with only mean PA was lower than any of the 9 models suggesting that mean PA alone (and not variability or predictability) may account for well-being the most. Levels of affective well-being (as noted by PA and NA) are important components of the operationalization of psychological well-being. So, it would be expected that individuals high in PA would also be high in psychological well-being because of the overlapping constructs in both measurements.

It should be noted that the main goal of this study was not to necessarily present a clear and concise portrayal of how different combinations of levels of mean affect, variability, and predictability influence the outcome variables. This goal of this initial foray into RQA methodology was to demonstrate its efficacy with a practical example and encourage other researchers to consider this unique new set of metrics. The authors were not expecting to draw sweeping theoretical conclusions or solidify demonstrable associations between affective experiences and health outcomes given the need for replication in differing and similar data sets. Nonetheless, there are a few important conclusions about the divergent impacts of variability and predictability that deserve consideration. In general, it was found that the most desirable outcomes stemmed from those who had high PA, low NA, low variability, or high predictability. PA did not interact with variability or predictability (high PA was good in all scenarios), but there were some interesting findings in regard to NA. The results indicate that for individuals high in  $NA_{MEAN}$ , it is good to have high variability (possibly because one gets “breaks” from the negativity) and it is bad to have high predictability (possibly because the negativity is unchangeable). As demonstrated by these findings, the additional RQA measures add explanatory depth to how the dynamics of affect are associated with health.

## General Discussion

These studies are the first to demonstrate how RQA metrics can add interesting new information about the association between affective experiences and health outcomes, on top of the effects of average affect or its standard deviation. When assessing psychosomatic connections, the vast majority of studies rely on indicators of mean affect (Pressman & Cohen, 2005). Our findings should implore future researchers to also consider the role of predictability as well as encourage the growing interest in affect variability and health. Affect unfolds over time, so the consideration of temporal patterns is critical in order to capture the dynamic nature of affective experiences. RQA metrics such as %REC and %DET provide information about temporal patterns that are often overlooked when relying only on standard deviation or mean levels. Furthermore, this study not only considers the independent effect of each metric, but also examines the combined effects of how they interact together to predict health outcomes.

The primary aim of these studies was to demonstrate how RQA can provide more in-depth analyses of affective experience (Study 1) and explore how this technique can be applied to various outcomes (Study 2). The wide range of results derived from different combinations of levels of mean affect, and variability and predictability demonstrate that affective experiences influence health in a highly complex manner. In certain situations, higher or lower variability and predictability may be beneficial, but in other situations the reverse may hold true. While we did uncover some interesting associations between affect and psychology well-being, depressive and somatic symptoms; these findings are not the focal point of our investigation. Rather, we hope that these studies will encourage future researchers to use RQA measures of predictability and temporal structure in a wide range of potential applications. Before recommendations can be made about how affect predictability impacts health, more work is needed to uncover the

mechanisms by which these processes operate and to disentangle how the interaction of these factors influence a variety of health outcomes.

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Affect variability and predictability: Using recurrence quantification analysis to better understand how the dynamics of affect relate to health.

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## **CHAPTER 5:**

### **Epilogue**

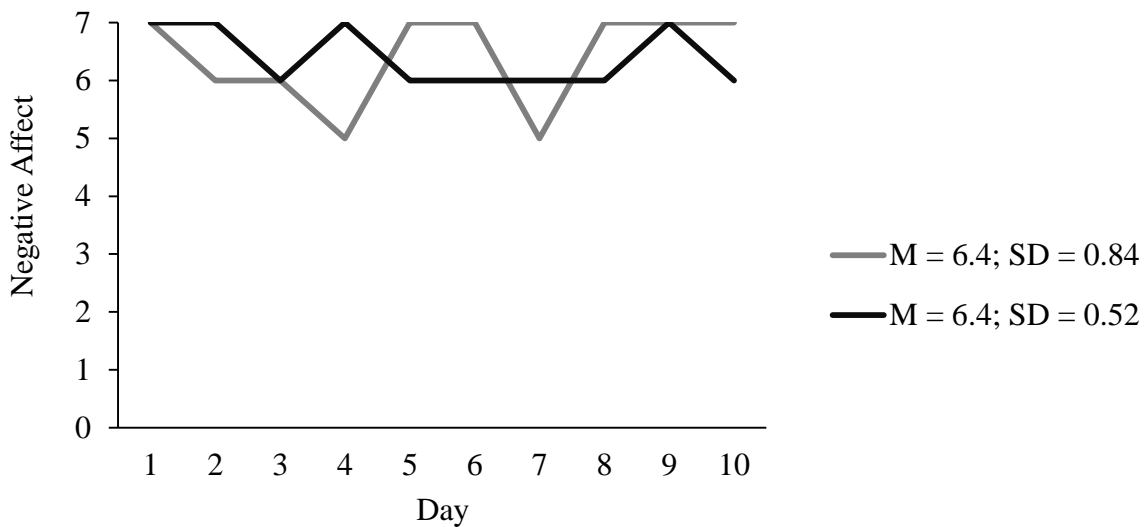
## Epilogue

This dissertation presents further evidence that affect variability has important implications for health and health-relevant outcomes. Furthermore, the studies presented here address significant gaps in the health and affective variability literature. First, Chapter 2 revealed what we know and do not know in the current field of affect variability and health, as well as some of the central limitations such as the limited use of health outcomes in affect variability research, a lack of examination of the interaction between mean affect and affect variability, and measurement issues surrounding the assessment of affect variability. Next, Chapter 3 demonstrated that affect variability is associated with a physical health-relevant biomarker, namely antibody (Ab) response to the influenza vaccination. This study showed that affect variability and its interaction with mean levels of affect influence an individual's immune response. Those high in mean positive affect (PA) who had low PA variability were more likely to have higher Ab levels in contrast to those who had high mean PA and high PA variability. Although NA variability did not interact with mean negative affect (NA), it did influence Ab levels, whereby those with less NA variability mounted a more robust immune response. Chapter 4 addressed some of the limitations of past methods for calculating variability by employing recurrence quantification analysis (RQA). RQA allowed for the assessment of affect predictability. Using RQA to establish whether affect over time was more or less predictable, the studies in Chapter 4 found that affect variability, predictability, and mean levels all had important implications for both psychological (e.g., depressive symptoms and well-being) and physical (e.g., self-reported somatic symptoms) health. Taken together, these studies addressed the three limitations outlined in Chapter 2's review of the health and affect variability research: 1. the limited use of physical health markers as outcome variables, 2. the lack of assessment of

the interaction between affect variability and mean levels of affect, and 3. the overuse of variability metrics that do not take into account patterning of affect over time. Although these studies have begun to address gaps in the literature, there are still limitations to this work. Additionally, future research should help uncover mechanisms that explain why these associations exist. Therefore, in Chapter 5, I expand upon the overall conclusions of the present research, the limitations of this work, and future directions of research in the area of affect variability and health.

Taken together, the studies in this dissertation have led to a number of consistent findings. First, as demonstrated in past research (e.g., Eid & Diener, 1999; Hardy & Segerstrom, 2016; Röcke, Li, & Smith, 2009; Steptoe, Leigh, & Kumari, 2011), in all cases PA variability was greater than NA variability but both were positively correlated. Second, in most cases, NA mean was positively associated with both NA and PA variability while PA mean was negatively associated with NA and PA variability. Third, greater variability tended to be associated with worse outcomes (lower Ab response, higher depressive and somatic symptoms, and lower well-being) but there were many mean by variability interactions such that at some levels of mean affect, affect variability was beneficial for health. When did greater affect variability tend to be helpful or harmful at different levels of mean affect? In Chapter 3, at high levels of mean PA, greater PA variability was associated with worse Ab production. Similarly, in Chapter 4, at high levels of mean PA, greater PA variability was associated with more depressive symptoms. In contrast, in Chapter 4, greater NA variability tended to be associated with fewer depressive symptoms and higher well-being. As a whole, there appear to be different consequences of the interaction between mean and variability for NA vs. PA. As hypothesized in Chapter 2, someone who is extremely high on NA may benefit from having a larger standard deviation because this

will necessarily indicate that they have more occurrences of low NA (see Figure 1 light grey line). Despite having more occurrences of high NA (this is necessarily the case compared to the individual with lower variability because mean level is held constant), these “breaks” seem to be associated with beneficial effects. The reverse reasoning can be used for the PA findings. An individual with high mean PA does not benefit from “breaks” in PA and such “breaks” represented by higher variability are associated with worse outcomes.



*Figure 1.* Two individuals with the same mean negative affect but with different standard deviations. M = mean; SD = standard deviation.

To better interpret these overall findings, there are several limitations of these new empirical studies that should be noted. First, all study the participants (aside from the simulation study) were college students. Therefore, these findings may not translate to different populations. Indeed, affect variability has been shown to be greater in younger versus older individuals (Brose, Scheibe, & Schmiedek, 2013). However, because younger individuals have greater affect variability as compared to older individuals, it may be that studying young adults may provide contexts in which more affect variability is likely to occur and subsequently the health effects that follow. Second, as is often the case in affect research, reverse causality concerns may be an

issue when determining the correlates of affect variability and health. It is possible that mental status may be the cause or be due to affect variability. For example, poor mental health (clinical or non-clinical) might lead a person to have more affect variability. However, this question has not been tested. Similarly, biological activity in the body (e.g., inflammation, microbiome activity) is known to influence affective states (Moloney, Desbonnet, Clarke, Dinan, & Cryan, 2014), so even an ongoing infectious illness could radically alter felt affect and therefore affect variability. Although reverse causality is a concern in this work, it should not be surprising that affect variability could lead to health consequences. Mean levels of affect have been shown to causally influence a slew of psychological and physical health outcomes (Burton & King, 2004; Emmons & McCullough, 2003; Sin & Lyubomirsky, 2009) and so it may be the case that variability in levels could lead to changes in health as well.

The research area of affect variability and health can benefit from a number of research advances. First, replication is needed in every area of this dissertation to solidify our understanding of 1. the association between affect variability and physical health and health-relevant biomarkers, 2. how affect variability interacts with mean levels to influence psychological and physical health, and 3, how RQA can help researchers better understand the predictability of affect.

In addition to replication of the present research, future research on *how* affect variability influences health is needed. In other words, why is affect variability related to health? Although the goal of this dissertation was not to test such mechanisms, future research can build upon the findings of this dissertation to explore what may link the association between affect variability and health. To better understand *how* affect variability influences health (i.e., the mechanisms of this association), it is beneficial to investigate some of the prominent variables that play a role in

the relationship between mean levels of affect and health outcomes (Ong, 2010). For example, health behaviors (e.g., sleep), physiological activity (e.g., HPA-axis functioning), affect regulation, and social support may all help explain these associations. For example, sleep is a health behavior that is influenced by affect (Brummett et al., 2006; Kalmbach, Pillai, Roth, & Drake, 2014) and has psychological and physical health consequences (Ayas et al., 2003a; Ayas et al., 2003b; Cribbet et al., 2014; Lovato & Gradisar, 2014; Patel et al., 2004; Prather, Janicki-Deverts, Hall, & Cohen, 2015). If it is that greater affect variability leads to worse sleep, this may be one pathway that can explain the affect variability and health association.

Physiological activity may be an additional pathway by which affect variability may lead to health outcomes. For example, the HPA axis has been found to be a pathway by which mean affect leads to health outcomes (McEwen, 1998). Therefore, an association between affect variability and HPA axis activity may present similar effects on health. A single study examining PA variability (measured using the standard deviation approach) and cortisol, a marker of HPA axis functioning, found that extremely high and extremely low levels of PA variability were associated with worse daily cortisol profiles (i.e., higher overall levels, less steep slopes; Human et al., 2015).

In addition to behavioral (e.g., sleep) and physiological pathways, social and psychological factors may be at play as well. One explanation may be connected to the influence of affective well-being on social relationships. High affect variability may be indicative of poor affect regulation; poor affect regulation is indicative of poor social relationship quality (Gross & Oliver, 2003); and poor relationship quality is predictive of poor health (Cohen & Willis, 1985). In this way, the process of affect instability leading to undesirable health outcomes can be theoretically linked. All of these biopsychosocial factors (e.g., health behaviors, physiological

activity, social support, psychological resources) may play roles in the association between affect variability and health. It is up to future research to explore these questions of mechanisms.

Despite the need for additional research in several avenues, this dissertation emphasizes the importance of studying the affective experience beyond just mean levels of affect. Had affect variability not been taken into consideration during these studies, several findings would have gone unnoticed. For example, only examining mean levels of affect in Chapter 3 would have shown no association between affect and Ab response. Additionally, studying affect predictability (Chapter 4) provides a much larger focus of the affective experience. Not only does affect vary but it may also vary in predictable versus unpredictable ways. These findings demonstrate that affect variability is constantly important and has implications for health.

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