

UNIVERSITY OF CALIFORNIA SAN DIEGO

SAN DIEGO STATE UNIVERSITY

Stress as a spectrum: Associations of stress and trauma with disordered eating

A Dissertation submitted in partial satisfaction of the requirements
for the degree Doctor of Philosophy

in

Clinical Psychology

by

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2024

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Chair

University of California San Diego

San Diego State University

2024

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Chapter 1, in full, is a reprint of the material as it appears in the journal *Appetite*. I, Alexandra D. Convertino (primary author of this material), would like to thank all coauthors — Drs. Blashill, Helm, and Pennesi and Mr. Gonzales IV — for their contributions to this work. I also would like to thank *Appetite* for accepting this work for publication and Elsevier for publishing this work.

Chapter 2, in full, is a reprint of the material as it appears in the journal *International Journal of Eating Disorders*. I, Alexandra D. Convertino (primary author of this material), would like to thank all coauthors — Drs. Blashill and Morland — for their contributions to this work. I also would like to thank the *International Journal of Eating Disorders* for accepting this work for publication and John Wiley & Sons for publishing this work.

Chapter 3, in full, is a reprint of the material as it has been submitted to the *Journal of Affective Disorders*. I, Alexandra D. Convertino (primary author of this material), would like to thank all coauthors — Ms. Mendoza and Dr. Blashill — for their contributions to this work.

VITA

EDUCATION AND TRAINING

San Diego State University/University of California San Diego Joint Doctoral Program in Clinical Psychology, San Diego, CA

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Thesis: An Integrated Model of Eating Disordered Behavior in Sexual Minority Young Adults

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BS in Psychology

Senior Honors Thesis: Body Dissatisfaction Following Exposure to Media Images: The Role of Photoshopping

PUBLICATIONS

*denotes a mentored author

Peer-Reviewed Journal Articles

30. Brown, T. A., Klimek-Johnson, P., Siegel, J. A., **Convertino, A. D.**, Douglas, V. J., Pachankis, J. E., & Blashill, A. J. (2024). Promoting resilience to improve disordered eating (PRIDE): A case series of an eating disorder treatment for sexual minority individuals. *International Journal of Eating Disorders*.
29. *Mendoza, R. R., **Convertino, A. D.**, & Blashill, A. J. (2023). A longitudinal study of potentially traumatic events and binge-purge eating disorder onset in children. *Appetite*. <https://doi.org/10.1016/j.appet.2023.107132>
28. Sahlan, R. N., Saunders, J. F., Klimek-Johnson, P., **Convertino, A. D.**, Lavender, J. M., Fitzsimmons-Craft, E. E., & Nagata, J. M. (2023). Validation of a Farsi version of the Eating Disorder Examination Questionnaire (F-EDE-Q) in adolescents and university students from Iran. *Journal of Eating Disorders*, *11*(1), 105. <https://doi.org/10.1186/s40337-023-00830-y>
27. **Convertino, A. D.**, & Mendoza, R. R. (2023). Posttraumatic stress disorder, traumatic events, and longitudinal eating disorder treatment outcomes: A systematic review. *International Journal of Eating Disorders*, *56*(6), 1055–1074. <https://doi.org/10.1002/eat.23933>

26. **Convertino, A. D.**, Elbe, C. I., *Mendoza, R. R., Calzo, J. P., Brown, T. A., Siegel, J. A., Jun, H., Corliss, H. L., & Blashill, A. J. (2022). Internalization of muscularity and thinness ideals: Associations with body dissatisfaction, eating disorder symptoms, and muscle dysmorphic symptoms in at risk sexual minority men. *International Journal of Eating Disorders*, *55*(12), 1765-1776. <https://doi.org/10.1002/eat.23829>
25. Claudat, K., Reilly, E. E., **Convertino, A. D.**, Trim, J., Cusack, A. & Kaye, W. H. (2022). Integrating evidence-based PTSD treatment into intensive eating disorders treatment: a preliminary investigation. *Eating and Weight Disorders*, *27*, 2599-3607. <https://doi.org/10.1007/s40519-022-01500-9>
24. **Convertino, A. D.**, Morland, L. A., & Blashill, A. J. (2022). Trauma exposure and eating disorders: Results from a United States nationally representative sample. *International Journal of Eating Disorders*, *55*(8), 1079-1089. <https://doi.org/10.1002/eat.23757>
23. Frederick, D. A., Tylka, T. L., Rodgers, R. F., Pennesi, J.-L., **Convertino, L.**, Parent, M. C., Brown, T. A., Compte, E. J., Cook-Cottone, C. P., Crerand, C. E., Malcarne, V. L., Nagata, J. M., Perez, M., Pila, E., Schaefer, L. M., Thompson, J. K., & Murray, S. B. (2022). Pathways from sociocultural and objectification constructs to body satisfaction among women: The U.S. Body Project I. *Body Image*, *41*, 195–208. <https://doi.org/10.1016/j.bodyim.2022.02.001>
22. Frederick, D. A., Hazzard, V. M., Schaefer, L. M., Rodgers, R. F., Gordon, A. R., Tylka, T. L., Pennesi, J.-L., **Convertino, L.**, Parent, M. C., Brown, T. A., Compte, E. J., Cook-Cottone, C. P., Crerand, C. E., Malcarne, V. L., Nagata, J. M., Perez, M., Pila, E., Thompson, J. K., & Murray, S. B. (2022). Sexual orientation differences in pathways from sociocultural and objectification constructs to body satisfaction: The U.S. Body Project I. *Body Image*, *41*, 181–194. <https://doi.org/10.1016/j.bodyim.2022.02.002>
21. Frederick, D. A., Schaefer, L. M., Hazzard, V. M., Rodgers, R. F., Tylka, T. L., Ong, L. Q., Pennesi, J.-L., **Convertino, L.**, Parent, M. C., Brown, T. A., Compte, E. J., Cook-Cottone, C. P., Crerand, C. E., Malcarne, V. L., Nagata, J. M., Perez, M., Pila, E., Kevin Thompson, J., & Murray, S. B. (2022). Racial identity differences in pathways from sociocultural and objectification constructs to body satisfaction: The U.S. Body Project I. *Body Image*, *41*, 140–155. <https://doi.org/10.1016/j.bodyim.2022.01.019>
20. Frederick, D. A., Gordon, A. R., Cook-Cottone, C. P., Brady, J. P., Reynolds, T. A., Alley, J., Garcia, J. R., Brown, T. A., Compte, E. J., **Convertino, L.**, Crerand, C. E., Malcarne, V. L., Nagata, J. M., Parent, M. C., Pennesi, J.-L., Perez, M., Pila, E., Rodgers, R. F., Schaefer, L. M., ... Murray, S. B. (2022). Demographic and sociocultural predictors of sexuality-related body image and sexual frequency: The U.S. Body Project I. *Body Image*, *41*, 109–127. <https://doi.org/10.1016/j.bodyim.2022.01.010>
19. Frederick, D. A., Tylka, T. L., Rodgers, R. F., **Convertino, L.**, Pennesi, J.-L., Parent, M. C., Brown, T. A., Compte, E. J., Cook-Cottone, C. P., Crerand, C. E., Malcarne, V. L., Nagata, J. M., Perez, M., Pila, E., Schaefer, L. M., Thompson, J. K., & Murray, S. B. (2022). Pathways from sociocultural and objectification constructs to body satisfaction among men: The U.S. Body Project I. *Body Image*, *41*, 84–96. <https://doi.org/10.1016/j.bodyim.2022.01.018>
18. Frederick, D. A., Crerand, C. E., Brown, T. A., Perez, M., Best, C. R., Cook-Cottone, C. P., Compte, E. J., **Convertino, L.**, Gordon, A. R., Malcarne, V. L., Nagata, J. M., Parent, M. C., Pennesi, J.-L., Pila, E., Rodgers, R. F., Schaefer, L. M., Thompson, J. K., Tylka, T. L., &

- Murray, S. B. (2022). Demographic predictors of body image satisfaction: The U.S. Body Project I. *Body Image*, *41*, 17–31. <https://doi.org/10.1016/j.bodyim.2022.01.011>
17. Frederick, D. A., Pila, E., Malcarne, V. L., Compte, E. J., Nagata, J. M., Best, C. R., Cook-Cottone, C. P., Brown, T. A., **Convertino, L.**, Crerand, C. E., Parent, M. C., Pennesi, J.-L., Perez, M., Rodgers, R. F., Schaefer, L. M., Thompson, J. K., Tylka, T. L., & Murray, S. B. (2022). Demographic predictors of objectification theory and tripartite influence model constructs: The U.S. Body Project I. *Body Image*, *40*, 182–199. <https://doi.org/10.1016/j.bodyim.2021.12.012>
 16. **Convertino, A. D.**, & Blashill, A. J. (2022). Psychiatric comorbidity of eating disorders in children between the ages of 9 and 10. *Journal of Child Psychology and Psychiatry*, *63*(5), 519–526. <https://doi.org/10.1111/jcpp.13484>
 15. **Convertino, A. D.**, Helm, J. L., Pennesi, J.-L., Gonzales IV, M., & Blashill, A. J. (2021). Integrating minority stress theory and the tripartite influence model: A model of eating disordered behavior in sexual minority young adults. *Appetite*, *163*. <https://doi.org/10.1016/j.appet.2021.105204>
 14. **Convertino, A. D.**, Brady, J. P., Grunewald, W., & Blashill, A. J. (2021). Intimate partner violence and muscularity-building behavior in Latino sexual minority men. *Eating Disorders: The Journal of Treatment & Prevention*, *29*(3), 245–259. <https://doi.org/10.1080/10640266.2021.1891371>
 13. Klimek, P., **Convertino, A. D.**, Pennesi, J., Gonzales, M., Roesch, S. C., Nagata, J. M., & Blashill, A. J. (2021). Confirmatory factor and measurement invariance analyses of the Eating Disorder Examination Questionnaire in sexual minority men and women. *International Journal of Eating Disorders*, *54*(5), 745–754. <https://doi.org/10.1002/eat.23488>
 12. Klimek, P., **Convertino, A. D.**, Gonzales IV, M., Roesch, S. C., Blashill, A. J. (2021). Confirmatory factor and measurement invariance analyses of the Drive for Muscularity Scale in sexual minority men and women. *Psychology of Sexual Orientation & Gender Diversity*, *9*(2), 236–243. <https://doi.org/10.1037/sgd0000472>
 11. **Convertino, A. D.**, Brady, J. P., Albright, C. A., Gonzales IV, M., & Blashill, A. J. (2021). The role of sexual minority stress and community involvement on disordered eating, dysmorphic concerns and appearance- and performance-enhancing drug misuse. *Body Image*, *36*, 53-63. <https://doi.org/10.1016/j.bodyim.2020.10.006>
 10. Rozzell, K. N., Carter, C., **Convertino, A. D.**, Gonzales IV, M., & Blashill, A. J. (2020). The Dysmorphic Concern Questionnaire: Measurement invariance by gender and race/ethnicity among sexual minority adults. *Body Image*, *35*, 201-206. <https://doi.org/10.1016/j.bodyim.2020.08.010>
 9. Grunewald, W., **Convertino, A. D.**, Safren, S. A., Mimiaga, M. J., O’Cleirigh, C., Mayer, K. H., & Blashill, A. J. (2020). Appearance discrimination and binge eating among sexual minority men. *Appetite*, *156*. <https://doi.org/10.1016/j.appet.2020.104819>
 8. Schumacher, L. M., Martinelli, M. K., **Convertino, A. D.**, Forman, E. M., & Butryn, M. L. (2020). Weight-related information avoidance prospectively predicts poorer self-monitoring and engagement in a behavioral weight loss intervention. *Annals of Behavioral Medicine*, *5*(22), 103-111. <https://doi.org/10.1093/abm/kaa034>

7. Remmert, J. E., **Convertino, A. D.**, Roberts, S. R., Godfrey, K. M., & Butryn, M. L. (2019). Stigmatizing weight experiences in health care: Associations with BMI and eating behaviours. *Obesity Science & Practice*, 5(6), 555–563. <https://doi.org/10.1002/osp4.379>
6. **Convertino, A. D.**, Gonzales IV, M., Malcarne, V. L., & Blashill, A. J. (2019). A psychometric investigation of the Sociocultural Attitudes Towards Appearance Questionnaire-4-Revised among sexual minority adults in the U.S. *Body Image*, 31, 96–101. <https://doi.org/10.1016/j.bodyim.2019.08.013>
5. Call, C. C., Schumacher, L. M., Rosenbaum, D. L., **Convertino, A. D.**, Zhang, F., & Butryn, M. L. (2019). Participant and interventionist perceptions of challenges during behavioral weight loss treatment. *Journal of Behavioral Medicine*, 42(2), 353–364. <https://doi.org/10.1007/s10865-018-9965-0>
4. Kerrigan, S. G., Clark, M., **Convertino, A.**, Forman, E. M., & Butryn, M. L. (2018). The association between previous success with weight loss through dietary change and success in a lifestyle modification program. *Journal of Behavioral Medicine*, 41(2), 152–159. <https://doi.org/10.1007/s10865-017-9883-6>
3. Rosenbaum, D. L., Clark, M. H., **Convertino, A. D.**, Call, C. C., Forman, E. M., & Butryn, M. L. (2018). Examination of nutrition literacy and quality of self-monitoring in behavioral weight loss. *Annals of Behavioral Medicine*, 52(9), 809–816. <https://doi.org/10.1093/abm/kax052>
2. **Convertino, A. D.**, Rodgers, R. F., Franko, D. L., & Jodoin, A. (2016). An evaluation of the Aerie Real campaign: Potential for promoting positive body image? *Journal of Health Psychology*, 24(6), 726–737. <https://doi.org/10.1177/1359105316680022>
1. Rodgers, R. F., Wilking, C., Gottlieb, M., Daynard, R., Lovering, M., Matsumoto, A., Luk, S., Naab, P., Iannuccilli, A., Shoemaker, H., **Convertino, A.**, & Franko, D. L. (2016). A qualitative study of the decision to engage in tanning behaviors among female college students. *Revue Européenne de Psychologie Appliquée/European Review of Applied Psychology*, 66(1), 1–8. <https://doi.org/10.1016/j.erap.2015.11.003>

Book Chapters

1. **Convertino, A. D.**, Albright, C. A., & Blashill, A. J. (2021). Eating disorders and related symptomatology in sexual minority men and boys. In J. M. Nagata, T. A. Brown, S. B. Murray, & J. M. Lavender (Eds.), *Eating Disorders in Boys and Men* (1st ed.). Springer.

Under Review

1. **Convertino, A. D.**, *Mendoza, R. R., & Blashill, A. J. (2024). *Eating disorder and posttraumatic stress symptomology: An intensive time-series psychological network analysis* [Manuscript submitted for publication]. Psychology Department, San Diego State University.

POSTERS AND SYMPOSIA PRESENTATIONS

* denotes mentee

41. Henriksen, H. A., Askew, A., Pennesi, J-L., **Convertino, A. D.**, Rozzell-Voss, K., & Blashill, A. J. (2024, August 15-17). *Muscularity-oriented attitudes and behaviors among college-aged women of different sexual orientations* [Poster presentation]. 2024 National LGBTQ Health Conference, Atlanta, Georgia, United States.

40. **Convertino, A. D.** & Luzier, J. L. (2024, March 14-16). *The association of PTSD with symptom severity in outpatient treatment for eating disorders* [Poster presentation]. 2024 International Conference for Eating Disorders, New York City, New York, United States.
39. **Convertino, A. D.** & Luzier, J. L. (2024, March 14-16). *The associations of fear of negative and positive evaluation with eating pathology* [Poster presentation]. 2024 International Conference for Eating Disorders, New York City, New York, United States.
38. **Convertino, A. D.**, & Blashill, A. J. (2023, November 16-19). *Eating disorder and posttraumatic stress disorder symptoms: A network analysis replication* [Poster presentation]. Association for Behavioral and Cognitive Therapies 57th Annual Convention, Seattle, WA, United States.
37. **Convertino, A. D.**, Pennesi, J-L., & Blashill, A. J. (2023, November 16-19). *Difficulties in emotion regulation moderate the longitudinal association between negative affect and dieting behavior* [Poster presentation]. Association for Behavioral and Cognitive Therapies 57th Annual Convention, Seattle, WA, United States.
36. *Mendoza, R. R., **Convertino, A. D.**, & Blashill, A. J. (2023, November 16-19). *Borderline personality disorder symptoms and eating disorder diagnoses: Findings from a nationally representative survey of US adults (NESARC-III)* [Poster presentation]. Association for Behavioral and Cognitive Therapies 57th Annual Convention, Seattle, WA, United States.
35. **Convertino, A. D.** & Luzier, J. L. (2023, October 20-21). *PTSD diagnosis predicts reduced early change in eating disorder symptoms during outpatient treatment for eating disorders* [Poster presentation]. West Virginia Psychological Association Fall Conference 2023, Julian, WV, United States.
*1st Place Poster Award
34. *Mendoza, R. R., **Convertino, A. D.**, & Blashill A. J. (2023, June 1-3). *Differences between bisexual and gay men in associations between sociocultural constructs and eating pathology* [Poster presentation]. 2023 International Conference for Eating Disorders, Washington, DC, United States.
*Top 10 Highest Ranked Poster
33. *Mendoza, R. R., **Convertino, A. D.**, & Blashill A. J. (2023, March 3-4). *Examining associations of borderline personality disorder symptoms and eating disorder diagnoses* [Oral presentation]. San Diego State University Student Symposium (S³), San Diego, CA, United States.
32. Brown, T. A., Klimek-Johnson, P., **Convertino, A. D.**, Douglas, V. J., Pachankis, J., & Blashill, A. J. (2022, November 17-20). A pilot intervention of a novel eating disorder treatment for sexual minority men and women. In T. Myers (Chair), *Eating pathology in sexual and gender minority individuals: Prevalence, related sociocultural factors, and treatment* [Symposium]. Association for Behavioral and Cognitive Therapies 56th Annual Convention, New York, NY, United States.
31. *Mendoza, R. R., **Convertino, A. D.**, & Blashill A. J. (2022, October 27-29). *Muscular ideal internalization and muscularity-oriented eating behaviors: The role of negative affect* [Poster presentation]. National Diversity in STEM Conference, San Juan, Puerto Rico.

30. *Mendoza, R. R., **Convertino, A. D.**, & Blashill A. J. (2022, October 21). *Muscular ideal internalization and muscularity-oriented eating behaviors: The role of negative affect* [Oral presentation]. San Diego State University Undergraduate Research Symposium, San Diego, CA, United States.
29. *Mendoza, R. R., **Convertino, A. D.**, & Blashill A. J. (2022, April 6-9). *Traumatic life events and eating disorder prevalence in children between the ages of 9 and 10* [Poster presentation]. Society of Behavioral Medicine 43rd Annual Meeting and Scientific Sessions, Baltimore, MD, United States.
28. *Mendoza, R. R., **Convertino, A. D.**, & Blashill A. J. (2022, March 4-5). *A longitudinal study of trauma and the development of eating disorders in children* [Conference session]. San Diego State University Student Research Symposium, San Diego, CA, United States.
27. *Mendoza, R. R., **Convertino, A. D.**, & Blashill A. J. (2022, January 14-15). *Traumatic life events and eating disorder prevalence in children between the ages of 9 and 10* [Poster presentation]. Diversifying Clinical Psychology Conference, San Diego, CA, United States.
*Poster Presentation Award
26. **Convertino, A. D.**, Brown, T. A., Calzo, J. P., Pennesi, J-L., & Blashill, A. J. (2021, November 18-21). Internalization of muscularity and thinness: Associations with body dissatisfaction, cognitive restraint, and muscle dysmorphic symptoms in sexual minority men. In **A. D. Convertino** (Chair), *Body image and eating pathology in sexual and gender minority individuals: Risk and protective factors* [Symposium]. Association for Behavioral and Cognitive Therapies 55th Annual Convention, New Orleans, LA, United States.
25. *Mendoza, R. R., **Convertino, A. D.**, & Blashill A. J. (2021, November 10-13). *Muscularity-oriented eating pathology in college men: Associations with body ideal internalization and appearance pressures* [Poster presentation]. Annual Biomedical Research Conference for Minority Students, Washington, DC, United States.
*Poster Presentation Award
24. *Mendoza, R. R., **Convertino, A. D.**, & Blashill A. J. (2021, October 15) *Muscular-Oriented Eating Pathology in College Men: Associations with body ideal internalization and appearance pressures* [Oral presentation]. San Diego State University Undergraduate Research Symposium, San Diego, CA.
23. **Convertino, A. D.** & Blashill, A. J. (2021, June 10-12). *Psychiatric comorbidity of eating disorders in children between the ages of 9 and 10* [Paper presentation]. 2021 International Conference on Eating Disorders, Monterrey, NL, Mexico.
22. **Convertino, A. D.**, Brady, J. P., Grunewald, W., & Blashill, A. J. (2021, June 10-12). *Intimate partner violence and muscularity-building behavior in Latino sexual minority men* [Paper presentation]. 2021 International Conference on Eating Disorders, Monterrey, NL, Mexico.
21. **Convertino, A. D.**, Gonzales IV, M., & Blashill, A. J. (2021, March 18-19). Muscularity dissatisfaction and behaviors in sexual minority individuals: The role of community involvement. In C. Crerand (Chair), *Understanding risk and maintenance factors for body image disturbance in diverse psychiatric and non-psychiatric samples* [Symposium]. Anxiety and Depression Association of America Annual Conference, Boston, MA, United States.

20. **Convertino, A. D.**, Helm, J. L., Pennesi, J-L., Gonzales IV, M., & Blashill, A. J. (2020, November 19-22). *An integrated model of eating disordered behavior in sexual minority young adults* [Poster presentation]. Association for Behavioral and Cognitive Therapies 54th Annual Convention, Philadelphia, PA, United States.
19. Pennesi, J-L., Helm, J. L., **Convertino, A. D.**, & Blashill, A. J. (2020, November 19-22). *Test and extension of the dual-pathway model for bulimic pathology and muscle-building behavior in men: A daily diary study* [Poster presentation]. Association for Cognitive and Behavioral Therapies 54th Annual Convention, Philadelphia, PA, United States.
18. Rozzell, K. N., Carter, C., **Convertino, A. D.**, Gonzales IV, M., & Blashill, A. J. (2020, November 19-22). *An assessment of the Dysmorphic Concern Questionnaire: Measurement invariance by gender and race/ethnicity among sexual minority adults* [Poster presentation]. Association for Behavioral and Cognitive Therapies 54th Annual Convention, Philadelphia, PA, United States.
17. Grunewald, W., **Convertino, A. D.**, Safren, S. A., Mimiaga, M. J., O'Cleirigh, C., Mayer, K. H., & Blashill, A. J. (2020, April 1-4). *Association between appearance-based discrimination and binge eating disorder among sexual minority men* [Poster presentation]. Society of Behavioral Medicine 41st Annual Meeting and Scientific Sessions, San Francisco, CA, United States.
16. **Convertino, A. D.**, & Blashill, A. J. (2019, November 21-24). *Association of everyday discrimination and eating pathology in a sample of sexual minority men* [Poster presentation]. Association for Behavioral and Cognitive Therapies 53rd Annual Convention, Atlanta, GA, United States.
15. **Convertino, A. D.**, Gonzales IV, M., & Blashill, A. J. (2019, April 16-21). *Sexual minority stress and community influence on disordered eating, dysmorphic concerns and appearance- and performance-enhancing drug misuse* [Conference session]. International Conference on Body Image and Health, Puerto Vallarta, JA, Mexico.
14. Remmert, J. E., **Convertino, A. D.**, Roberts, S. R., Godfrey, K. M., & Butryn, M. L. (2019, March 6-9). *The relationship between stigmatizing weight experiences in healthcare, BMI, and eating behaviors* [Paper presentation]. Society of Behavioral Medicine 40th Annual Meeting and Scientific Sessions, Washington D.C, United States.
13. Schumacher, L. M., Martinelli, M., **Convertino, A. D.**, Forman, E. M., & Butryn, M. L. (2019, March 6-9). *Weight-related information avoidance as a predictor of self-monitoring compliance in a behavioral weight management program* [Poster presentation]. Society of Behavioral Medicine 40th Annual Meeting and Scientific Sessions, Washington DC, United States.
12. Call, C. C., Sininsky, S., **Convertino, A. D.**, Roberts, S. R., & Butryn, M. L. (2018, August 9-12). *Behavioral and psychological factors that counteract physical activity during behavioral weight loss* [Poster presentation]. 2018 American Psychological Association Annual Convention, San Francisco, CA, United States.
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ABSTRACT OF THE DISSERTATION

Stress as a spectrum: Associations of stress and trauma with disordered eating

by

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Objective: Multiple stressors are critical in the development of eating disorders (EDs).

The most widely studied forms of stress associated with EDs are childhood trauma, sexual assault, weight-related criticism, and pressure to obtain the appearance ideals. However, it is unclear how relatively underexplored forms of stress (i.e., sexual minority stress, non-interpersonal trauma, and posttraumatic stress) are associated with EDs in vulnerable

populations. **Methods:** The tripartite influence model of EDs proposes that body dissatisfaction and subsequent ED behavior are the result of social pressure, thin ideal internalization, and social appearance comparison. *Study 1* integrated sexual minority stressors into the tripartite influence model in a sample of young adult sexual minority men ($n = 479$) and women ($n = 483$) to determine whether integrating chronic stressors improved model explanation of variance. *Study 2* examined the association of traumatic events with eating disorders, specifically to differentiate whether war-related trauma, non-interpersonal trauma, non-sexual interpersonal trauma, or sexual interpersonal trauma were associated with EDs in a nationally representative sample of 36,309 US adults. *Study 3* examined the longitudinal associations in a network analysis of ED symptoms and posttraumatic stress disorder (PTSD) symptoms in a sample of US undergraduates ($N = 50$) with elevated symptoms of these disorders. **Results:** *Study 1* demonstrated that the tripartite influence model and minority stress theory could be successfully integrated and explained more variance in ED outcomes as compared to models that do not incorporate both theories. *Study 2* found that sexual interpersonal trauma was significantly positively associated with anorexia nervosa and binge eating disorder (BED). BED was found to be equally associated with sexual interpersonal, other interpersonal, and non-interpersonal trauma. *Study 3* found that loss of control eating, concentration impairment related to shape and weight, purging, fear of weight gain, negative alterations in cognition and mood, and body dissatisfaction demonstrated the strongest associations in the network. **Discussion:** *Studies 1, 2, and 3* confirmed that minority stress, traumatic events, and PTSD symptoms are associated with eating pathology. The consideration of these stressors in explanatory models of EDs could help refine prevention and intervention efforts with various vulnerable populations.

INTRODUCTION

Eating disorders are serious mental health conditions associated with high risk of mortality (Smink et al., 2012), impaired quality of life (Jenkins et al., 2011), serious medical complications (Mehler et al., 2018; J. E. Mitchell, 2016), and complex psychiatric comorbidities (Hudson et al., 2007). Even subclinical levels of eating pathology are associated with impaired quality of life, with impairment increasing as eating pathology severity increases (Jenkins et al., 2011). While eating disorders and disordered eating behaviors are a significant health risk, individuals with eating disorders experience a high rate of relapse after treatment, with approximately one-third of patients experiencing a recurrence of symptoms (Berends et al., 2018; Eddy et al., 2017; Olmsted et al., 2015). Furthermore, among randomized controlled trials of individuals that receive enhanced cognitive behavioral therapy, the front-line treatment for adults with eating disorders, the treatment dropout rate was 26% (Linardon et al., 2018). Therefore, eating disorders are a significant public health concern, with substantial work to still be conducted in improving retention and outcomes for eating disorder treatment.

Research in eating disorders has commonly emphasized the role of stress in the onset of disordered eating (Brewerton, 2015; Monteleone et al., 2018). In line with the diathesis-stress model (Gottesman, 1991), stressful life events precede the onset of eating pathology (Degortes et al., 2014; Machado et al., 2014; Pike et al., 2006; Rojo et al., 2006). Some of the most widely studied and well-connected forms of stress associated with the onset of eating pathology are childhood trauma (Caslini et al., 2016; Pignatelli et al., 2017), sexual assault (Madowitz et al., 2015), weight- and appearance-related commentary (Lie et al., 2019), and pressure to obtain the appearance ideals (Cafri et al., 2006).

Stress has also been theorized to play a role in the maintenance of eating pathology. As an example, stressful life events predict relapses following initial remission from an eating disorder (Grilo et al., 2009, 2012). Eating disordered behaviors may serve to regulate stress; the escape model of binge eating specifies that negative self-evaluation causes emotional distress and negative affect, which individuals with binge eating attempt to alleviate or avoid by the narrowed attention characteristic of a binge episode (Heatherton & Baumeister, 1991). Therefore, when individuals who engage in binge eating experience stressors that lead to self-critical thoughts, they are more likely to engage in binge eating, which provides them an escape from their thoughts, reinforcing that behavior and leading to greater likelihood of binge eating occurring in the future. This relationship has been supported by experimental studies of eating pathology, where women with binge eating disorder were more likely to eat larger amounts of food when exposed to social stressors, as compared to women without binge eating disorder and women with binge eating disorder who were not exposed to the stressor (Schulz & Laessle, 2012). However, there still remain significant questions about the role of stress in the maintenance of eating pathology that have yet to be answered.

One question is whether the inclusion of chronic stress in existing models of eating pathology provides incremental improvements to eating disorder-specific explanatory models. Some of the most widely utilized sociocultural models of eating pathology do not include chronic stressors as a potential influence on eating pathology. Given that these sociocultural models are important for the development of eating disorder interventions (Pennesi & Wade, 2016), omitting important influences on eating disordered behavior is likely to lead to less effective interventions.

To address this omission in the literature, **Study 1** sought to utilize sexual minority stress as a specific type of chronic stressor that has been found in previous studies to confer greater risk for eating pathology (e.g., Bayer et al., 2017; Wang & Borders, 2017; Watson et al., 2016). However, it is unclear whether sexual minority stress will explain additional variance when including eating disorder-specific etiology. As compared to their heterosexual peers, sexual minority (SM) individuals – including gay, lesbian, bisexual, and other non-heterosexual individuals, individuals who report same-gender attraction, and/or individuals who report having same-gender sexual contact (Institute of Medicine, 2011) – are at greater risk for body image concerns and eating pathology (Calzo et al., 2017; Frederick & Essayli, 2016).

Eating disorder development and risk can be conceptualized through sociocultural theories (e.g., Tiggemann, 2011). One of the most well-investigated theories is the tripartite influence model (Thompson, Covert, et al., 1999; Thompson, Heinberg, et al., 1999), which proposes that body dissatisfaction and subsequent disordered eating are the result of social pressure towards body ideals, internalization of the thin ideal, and social appearance comparison. This model was originally developed and tested in primarily heterosexual girls and women (e.g., Girard et al., 2018; Hazzard et al., 2019; Johnson et al., 2015; Lovering et al., 2018), but has also been adapted for samples of primarily heterosexual men (Karazsia & Crowther, 2009; Tylka, 2011), gay men, and lesbian/bisexual women (Hazzard et al., 2019; Huxley et al., 2015; Tylka & Andorka, 2012), providing preliminary support for these extended models. However, these models do not consider SM-specific contributions to eating disorder risk.

When considering SM-specific variables, most researchers have conceptualized SM individuals' greater risk through minority stress theory. Minority stress theory (I. H. Meyer, 1995, 2003) posits that SM groups experience increased stressors as a result of their minority

status, which leads to negative physical and mental health outcomes. Researchers have found significant associations minority stressors and disordered eating for SM populations (e.g., internalized homophobia; Bayer et al., 2017; Swearingen, 2006; Torres, 2008; Wang & Borders, 2017), suggesting that minority stress may play a role in eating disorder risk among SM groups. Thus, we build on this initial evidence to explore a comprehensive, integrated model combining the tripartite influence model (Thompson, Covert, et al., 1999; Thompson, Heinberg, et al., 1999) and minority stress theory (I. H. Meyer, 1995, 2003) to explain eating pathology in SM young adults.

While **Study 1** attempted to examine the association between chronic stress that may or may not be traumatic, **Study 2** endeavored to examine the association between different types of traumatic events and eating pathology. An estimated 80% of individuals that engage in disordered eating also report traumatic event exposure (K. S. Mitchell et al., 2012). Among individuals with eating disorders, exposure to traumatic events has been associated with more severe disordered eating (Backholm et al., 2013; Scharff, Ortiz, Forrest, & Smith, 2021; Tagay et al., 2014) and premature termination from eating disorder treatment (Anderson et al., 1997; Carter et al., 2006; Rodríguez et al., 2005). Therefore, traumatic stressors may be an important area of study within the field of eating disorders to improve eating disorder symptoms and treatment outcomes.

Specific types of trauma that have been linked to eating disorders include sexual assault (Arditte Hall et al., 2017, 2018; Blais et al., 2017; Madowitz et al., 2015), childhood abuse (Arditte Hall et al., 2017, 2018; Molendijk et al., 2017), and combat exposure (Arditte Hall et al., 2017, 2018; Breland et al., 2018), but other traumatic experiences (e.g., life-threatening illness, car accident, general physical assault) are under-investigated. For example, sexual assault, a form

of interpersonal trauma (i.e., assault or abuse that are perpetrated by another person; Forbes et al., 2012; Ford et al., 2011) has been examined numerous times with fairly conclusive findings, but other forms of interpersonal trauma (e.g., physical assault, stalking) are more rarely investigated and with mixed results (e.g., Arditte Hall et al., 2018; L. K. Meyer & Stanick, 2018; Quilliot et al., 2019). Similarly, few previous studies have examined non-interpersonal trauma (i.e., adverse events that do not necessarily involve another person) with mixed results (e.g., Gomez et al., 2021; L. K. Meyer & Stanick, 2018). Therefore, **Study 2** examined relatively understudied traumatic event exposure types with eating disorder diagnoses.

Further, **Study 2** also examined these four trauma types in a single, multivariable model to account for exposure to different trauma types. Since multiple trauma events are common (Kilpatrick et al., 2013), researchers have now begun to examine trauma exposure simultaneously in single models to examine differential relationships between trauma event types and eating disorders (e.g., Breland et al., 2018). If specific types of traumatic events are more likely to be associated with eating disorders, this may lead to novel insights for the prevention of eating disorders in individuals with traumatic event exposure, and potentially modified screening and care for these individuals.

Study 2 therefore examined the association of trauma classes, specifically sexual interpersonal (both childhood and adult), other interpersonal (e.g., physical assault, stalking), war/combat (e.g., active military combat, relief worker), and non-interpersonal (e.g., natural disasters, serious injury/illness) traumas, with eating disorders in a large, nationally representative survey of US adults. By including these trauma classes into one model, we were able to examine of the unique association of specific trauma classes with eating pathology.

Finally, **Study 3** sought to examine the association between posttraumatic stress disorder (PTSD) symptoms and eating disorder symptoms. While many people experience traumatic events (approximately 90%; Kilpatrick et al., 2013), only about 8% of US population is estimated to meet criteria for PTSD (Kilpatrick et al., 2013) and, among individuals with eating disorders, 18.35-24.59% have comorbid PTSD (Ferrell et al., 2022). In addition, individuals diagnosed with both an eating disorder and PTSD display more severe eating pathology than those diagnosed with an eating disorder alone (Scharff, Ortiz, Forrest, Smith, et al., 2021; Tagay et al., 2014; Vierling et al., 2015), and appear to be more likely to drop out of eating disorder treatment and/or relapse after treatment (Convertino & Mendoza, 2023). Therefore, individuals with comorbid PTSD and an eating disorder display higher clinical severity and lower treatment engagement/efficacy as compared to their single diagnosis peers.

Researchers have typically theorized that both PTSD and eating disorder symptoms must be addressed in order for remission from either disorder (Brewerton, 2007). This theory is based on cross-sectional research findings that suggest bidirectional, dependent relationships between symptoms. For example, emotion dysregulation – a common correlate of PTSD – has been associated with eating pathology in multiple analyses and, at least in cross-sectional analyses, has been found to mediate the association between traumatic event exposure and eating disordered behavior (Mills et al., 2015; K. S. Mitchell & Wolf, 2016; Moulton et al., 2015; Racine & Wildes, 2015). These findings suggest that eating pathology may serve as an emotion regulation tool to cope with PTSD-related symptoms, but this relationship has not been examined longitudinally.

Network analysis is a feasible, innovative data-driven approach to identify the longitudinal relationships between PTSD and eating disorder symptoms. The network

perspective of psychopathology posits that mental disorders are best conceptualized as a system of interacting symptoms (Borsboom & Cramer, 2013). Therefore, instead of an underlying entity of “mental disorder” causing all symptoms, symptoms tend to co-occur because they are causally related. This causal interplay of symptoms is what constitutes the disorder (Borsboom & Cramer, 2013). While previous studies have examined symptoms of PTSD and eating disorders through network analysis (e.g., Liebman et al., 2021; Nelson et al., 2022; Vanzhula et al., 2019), they have all use a cross-sectional design. These symptoms must be examined longitudinally in order to identify important symptom relationships for intervention. This type of study is ill-suited to examine the theorized longitudinal causal relationships between symptoms. Longitudinal network analysis can lead to important insights such as the most influential symptoms to target during treatment to inform later intervention development.

This dissertation broadly examined stress as it relates to eating pathology. While some specific stressors have been widely documented in their association with eating pathology (e.g., childhood trauma, sexual assault, weight- and appearance-related commentary, and pressure to obtain the appearance ideals), other forms of stress have not been examined, or have not been examined when accounting for other important influences. For example, **Study 1** sought to examine minority stress as a well-documented and defined form of chronic stress in the context of one of the most prominent sociocultural models of eating pathology. Prior to this study, no known study has examined a full integration of minority stress theory into the tripartite influence model. **Study 2** sought to examine traumatic stress in its relation to eating disorders to determine which forms of traumatic stress are more likely to be associated with eating disorders when accounting for other trauma experiences. Finally, **Study 3** sought to identify longitudinal relationships between PTSD symptoms and how they relate to eating disorder symptoms. This

study has implications for effective interventions to address the comorbidity between PTSD and an eating disorder. This dissertation sought to address some weaknesses of the current literature on eating disorders and stress by addressing the relationships that exist between minority stress, trauma experiences, PTSD and eating disorders.

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CHAPTER 1: STUDY 1

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Abstract

Eating disorders are serious mental illnesses associated with high mortality rates and health complications. Prior research has found increased rates of eating pathology in sexual minority (SM; e.g., lesbian, gay, bisexual) individuals compared to sexual majority (i.e., heterosexual) individuals. Two prominent models have potential to explain these differences: the tripartite influence model and minority stress theory. While both models separately have promise for explaining the pathway of eating disordered behavior in SM individuals, research has indicated that both models have unexplained variance. Therefore, a comprehensive, integrative model could further explain unique variance. 479 men and 483 women between 18 and 30 years were recruited through Qualtrics; all participants endorsed attraction to same-gender partners. Two models were estimated by gender using structural equation modeling. For men and women, community involvement accelerated the positive association of heterosexist discrimination with internalized homophobia. Minority stressors were associated with dissatisfaction and muscularity behavior, indicating the importance of incorporating minority stress. For women, community involvement accelerated both the association of pressures with muscularity internalization and the association of muscularity-based dissatisfaction with muscle building behaviors. If confirmed by prospective studies, this model could help refine prevention and intervention efforts with this vulnerable population.

Integrating Minority Stress Theory and the Tripartite Influence Model: A Model of Eating Disordered Behavior in Sexual Minority Young Adults

Eating disorders, and associated eating disordered behaviors, are a serious mental health concern with significant life-threatening medical and psychiatric morbidity and lower quality of life (Ágh et al., 2016; Arcelus et al., 2011; Klump et al., 2009; Mitchell & Crow, 2006). Indeed, mortality rates for eating disorders are among the highest rates for all psychiatric disorders (Arcelus et al., 2011). Disordered eating can affect individuals regardless of sociodemographic characteristics; however, risk for eating pathology is disproportionately higher for some groups.

Eating Pathology in Sexual Minority Individuals

One group at particular risk for eating pathology is sexual minority (SM) individuals (Calzo et al., 2017), which includes gay, lesbian, bisexual, and other non-heterosexual individuals, individuals who report same-gender attraction, and/or individuals who report having same-gender sexual contact (Institute of Medicine, 2011). Recent evidence suggests that SM individuals are at increased risk for body image concerns and eating pathology in comparison to their heterosexual peers (Alvy, 2013; Calzo et al., 2017; Feldman & Meyer, 2007; Frederick & Essayli, 2016). SM men (SMM) are shown to have higher rates of eating disorders (Diemer et al., 2015; Matthews-Ewald et al., 2014), and are consistently at greater risk for eating disorder symptomatology such as body image concerns, binge eating, restrictive dieting, and anabolic steroid misuse (Blashill et al., 2017; Calzo et al., 2013; Frederick & Essayli, 2016; Matthews-Ewald et al., 2014) compared with heterosexual men. Evidence of eating disorder risk among SM women (SMW) is less conclusive (Feldman & Meyer, 2007; Matthews-Ewald et al., 2014). A recent systematic review found that SMW have higher rates of eating disorders, binge eating, and purging behavior, but lower body dissatisfaction and drive for thinness, compared with

heterosexual women (Meneguzzo et al., 2018). Some studies (Feldman & Meyer, 2007; Matthews-Ewald et al., 2014; Morrison et al., 2004; Peplau et al., 2009) report little to no differences between SMW and heterosexual women on body image concerns and eating disorder prevalence. Other studies have speculated that SM status may even be protective against eating disorder symptomatology in women (Gettelman & Thompson, 1993; Huxley et al., 2015; Lakkis et al., 1999), although research only partially supports this idea. Despite these inconsistent findings among SMW, evidence largely indicates that SM individuals overall are at elevated risk for developing disordered eating behavior. Understanding the underlying theoretical mechanisms of elevated eating disorder risk for SM individuals through examination of etiological models is essential to understand this differential risk and, subsequently, inform future effective prevention and intervention programs.

Sociocultural Models

Sociocultural theories offer a framework for understanding how dominant sociocultural influences contribute to body image concerns and disordered eating (e.g., Tiggemann, 2011). One model that has received considerable attention is the tripartite influence model (Thompson, Covert, et al., 1999; Thompson, Heinberg, et al., 1999), which proposes that pressure from family, friends, and the media to conform to dominant sociocultural appearance ideals leads to body dissatisfaction and restrained eating via internalization of the thin ideal and social appearance comparison. Later modifications of the model included a fourth source of pressure called “significant others” which includes pressure from romantic partners, teachers, and coaches to achieve appearance ideals (thus a “quadripartite” model that includes pressures from four sources; Schaefer et al., 2017). This model has received widespread empirical support with samples of primarily heterosexual women (e.g., Girard et al., 2018; Hazzard et al., 2019; Johnson

et al., 2015; Lovering et al., 2018), and modified versions of the model for the development of muscle-building behaviors and dual body image pathways representing both muscularity and body fat dissatisfaction have received some support with samples of primarily heterosexual men (Karazsia & Crowther, 2009; Tylka, 2011). Researchers have recently begun to explore the model with gay men and lesbian/bisexual samples (Hazzard et al., 2019; Huxley et al., 2015; Tylka & Andorka, 2012), with research providing preliminary support for these extended models. Therefore, sociocultural models have promise for explaining the contribution of culture to body image concerns and eating disordered behaviors in SM individuals. However, previous research has found that existing sociocultural models do not explain all variance in disordered eating and muscle-building behaviors. For instance, the tripartite influence model in gay men explained 33.5% of the variance in muscle-building behaviors and 47.1% of the variance in disordered eating behaviors (Tylka & Andorka, 2012). Therefore, there is still additional variance to be explained beyond that found in previous sociocultural models.

Minority Stress Models

Researchers have also begun to integrate aspects of minority stress theory (Meyer, 1995, 2003), a prominent model for the development of negative physical and mental health outcomes for SM individuals, into the tripartite influence model. Minority stress theory posits that SM groups experience increased levels of stigma-related stress associated with their minority status, which can lead to higher rates of psychopathology, including disordered eating. The theory delineates distal stressors (e.g., discrimination, prejudice, stereotypes) and proximal stressors (e.g., internalized homophobia, expectations of rejection, concealment), as both distinct stressors and interdependent processes (Meyer, 2003). Some minority stressors have been included in models of disordered eating for SM populations, including internalized homophobia (Bayer et

al., 2017; Swearingen, 2006; Torres, 2008; Wang & Borders, 2017), sexual orientation concealment (Mason & Lewis, 2015), and heterosexist discrimination (Katz-Wise et al., 2015; Mason et al., 2017; Mason & Lewis, 2016; Watson et al., 2016), with recent evidence supporting the association between these stressors and eating pathology. Taken together, this body of research suggests that minority stress variables are important contributors to eating disorder risk among SM groups and should be considered in examination of eating disorder models. However, reported variance explained in disordered eating ranged from 17.4% to 38% (Mason et al., 2017; Mason & Lewis, 2015, 2016; Watson et al., 2016), indicating that minority stress may not completely explain eating disorder risk in this population.

Within the minority stress model in lesbian, gay and bisexual populations (Meyer, 2003), SM community involvement is considered a key moderating factor of negative physical and mental health outcomes, such that the SM community serves as a source of social support and coping that can ameliorate the adverse effects of minority stress. However, prior research has found that community involvement was positively related to muscularity enhancement behaviors, both directly and indirectly through internalization of the mesomorphic ideal and appearance comparison, suggesting that community involvement may promote gay men's engagement in muscularity-driven behaviors (Tylka & Andorka, 2012). Other research among SM groups found that community involvement may not be protective against negative outcomes, and was associated with higher disordered eating symptoms (Convertino et al., 2021; Davids & Green, 2011; Feldman & Meyer, 2007) and body dissatisfaction (Beren et al., 1996; Davids et al., 2015; Davids & Green, 2011), supporting the finding that community involvement may actually place SM individuals at greater risk for eating disorders.

A plausible alternative explanation for these findings might be provided by intraminority stress theory (Pachankis et al., 2020). This theory states that gay and bisexual men perceive status-based competitive pressures from within the community through their interactions with other SMM, including appearance-based pressures, which contribute to adverse mental health outcomes. It is therefore possible that community involvement may be associated with negative outcomes for SM community members. Prior research has identified similar processes in SMW, such that women who perceived they were falling short of community standards of appearance were at higher risk for depression and anxiety (Boyle & Omoto, 2014). However, the extent to which intraminority stress theory holds for SMW is unclear.

Current Study

Despite promising advances in SM mental health research, and recent improvements made to the tripartite influence model to include SM-specific variables, key differences in eating disorder risk that are defined by sexual orientation remain unexplained. While existing evidence shows that the tripartite influence model and variables included in the minority stress model have promise for explaining eating disorder risk in SM individuals, no existing study, to our knowledge, has tested these theories in an integrated model. Therefore, the integration of these theories in a comprehensive model for disordered eating in SM individuals could further explain unique variance, and may help to better clarify eating disorder disparities. In the current paper, we build on this initial evidence to explore a comprehensive, integrated model combining the tripartite influence model (Thompson, Covert, et al., 1999; Thompson, Heinberg, et al., 1999) and minority stress theory (Meyer, 1995, 2003) to explain eating pathology in SM young adults.

Aims and Hypotheses

The aim of the present study was to test an integrated model for eating disordered behavior among samples of SMM and SMW, by integrating the tripartite influence model and minority stress theory (see Figure 1). The current study provides a meaningful contribution to the examination of models for eating disorder risk in SM young adults in a number of ways. First, in line with recent extensions of the tripartite influence model to better represent appearance ideals for men and women that are both thin and muscular (Girard et al., 2018; Hazzard et al., 2019; Hoffmann & Warschburger, 2019; Rodgers et al., 2012; Tylka, 2011; Tylka & Andorka, 2012), we explored dual body image pathways to represent both thinness- and muscularity-based concerns. Second, we integrated minority stress theory variables (i.e., SM community involvement, internalized homophobia, sexual orientation concealment, heterosexist discrimination) within the tripartite influence model to assess eating disorder risk unique to SM young adults. This study is the first to empirically test a comprehensive integration of these two prominent risk models for eating disordered behavior in SM young adults. In doing so, we build on previous research on the health disparities between SM individuals and their non-SM counterparts (e.g., Plöderl & Tremblay, 2015). Third, we explored SM community involvement as a key moderating variable within our model, integrating these two theories. This is consistent with the pathways proposed in the minority stress model and subsequent tests of this model showing that community involvement may moderate the association between minority stressors and negative physical and mental health outcomes (Beren et al., 1996; Davids et al., 2015; Meyer, 2003). To examine this moderation, we utilized latent interaction modeling to better understand the associations between community involvement and variables within the minority stress and tripartite influence models, as theorized by the minority stress model (Meyer, 2003). Unlike more typical methods of examining interactions, examining latent interactions provides

the opportunity to control for measurement error in explanatory variables, which affords greater power to detect interaction effects when they exist (Bollen, 1989). This approach provides a novel addition to the literature of the association of community involvement with mental health outcomes, which has been largely unclear. Finally, we examined two eating disordered behavior variables (i.e., dietary restraint and muscle building behaviors) which may help to inform future transdiagnostic eating disorder interventions for SM groups.

The following hypotheses were made:

H1: Our integrated tripartite influence model and minority stress model would provide good fit to the data, separately for SMM and SMW. We expected sources of appearance pressure (i.e., family, peer, significant others, media) to load onto a latent pressures factor, which would be associated with both thin- and muscular-ideal internalization. We expected that thin- and muscular-ideal internalization would be associated with both thinness- and muscularity-based dissatisfaction, and further, that both thinness- and muscularity-based dissatisfaction would, in turn, be associated with our two examined eating disordered behavior variables (i.e., dietary restraint and muscle building behaviors). In line with previous research on the tripartite influence model (e.g., Hazzard et al., 2019; Huxley et al., 2015; Tylka, 2011) and in the interest of both comprehensiveness and parsimony, we included internalization of appearance ideals but not social comparisons.

H2: As predicted by minority stress theory, distal stressors (i.e., heterosexist discrimination) were expected to be associated with proximal stressors (i.e., internalized homophobia and sexual orientation concealment), such that greater number of heterosexist experiences would be associated with greater internalization of negative societal attitudes and sexual orientation concealment. Together, these minority stressors were expected to be

associated with greater negative outcomes (i.e., appearance-based dissatisfaction variables and eating disordered behavior variables). Further, we expected sexual orientation concealment and internalized homophobia to be associated with greater eating disordered behavior variables both directly and indirectly through appearance-based dissatisfaction variables.

H3: To integrate the tripartite influence model and minority stress theory, we expected SM community involvement to moderate the associations between pressures and internalization of appearance ideals, and between appearance-based dissatisfaction and eating disordered behavior variables. We also expected SM community involvement to moderate the association between heterosexist discrimination and both sexual orientation concealment and internalized homophobia. Based on the aforementioned theories indicating contradictory hypotheses for the association between community involvement and disordered eating, no directional hypotheses were generated regarding the simple slopes of these interaction terms.

Method

Participants and Procedures

Participants were 483 SMW and 479 SMM aged 18-30 years ($M_{age} = 23.7$, $SD = 3.7$) recruited from across the United States through Panels, a service provided by Qualtrics, an online survey-based software company. Individuals can create an account through Qualtrics and participate in surveys if they meet criteria set by a researcher/s. Participants who were: 1) between the ages of 18-30 years old; 2) self-identify as gay/lesbian/bisexual; 3) self-identify as i) African American, ii) Non-Hispanic White, iii) Asian American/Pacific Islander, iv) Native American/Alaska Native, or v) Hispanic with any other race; and 4) English speaking based on their Qualtrics profile, were invited to participate in the study. Following consent, participants completed a pre-screener to confirm eligibility, then completed a 15-20 minute survey.

Participants received the equivalent of \$4 US dollars in e-reward currency that can be redeemed within Qualtrics for gift cards or airline miles. All procedures were reviewed and approved by the university's Institutional Review Board. This dataset has been reported previously (Gonzales IV & Blashill, 2021); however, the other article is primarily focused on reporting race and ethnicity group differences in eating disorders, body dysmorphic disorder, drive for muscularity, and appearance- and performance-enhancing drug misuse. The current study is unique in that it tested a full structural model of eating disorder behavior.

Measures

Demographics

Sexual identity was assessed using a single, close-ended item, "How would you describe your sexual identity?" with response options: *Lesbian/Gay*, *Bisexual*, *Heterosexual*, *Asexual*, *I prefer not to specify*, and *Other* where individuals were provided with an open-ended response option. Ethnicity was assessed using a single, closed-ended item, "What is your ethnicity?" with response options: *Hispanic/Latino* or *Not Hispanic/Latino*. Race was assessed using a single, closed-ended item, "What is your race?" with response options: *White*, *Black or African American*, *Native American or American Indian*, and *Asian/Pacific Islander*.

Pressures

Sociocultural pressures to achieve the body ideal were measured using the Pressures subscales of the Sociocultural Attitudes Towards Appearance Questionnaire 4-Revised (SATAQ-4R; Schaefer et al., 2017), including Family, Peers, Significant Others, and Media. Items were scored on a 5-point scale ranging from 1 (*definitely disagree*) to 5 (*definitely agree*), with higher scores indicating greater pressures. Previous studies have found an internal consistency range of the pressures subscales of $\alpha = .89-.96$ in men and $\alpha = .92-.96$ in women

(Schaefer et al., 2017). The SATAQ-4R has been previously validated in SMM and SMW (Convertino et al., 2019). Internal consistency in the current study was $\alpha = .95$ for SMM and $\alpha = .93$ for SMW.

Thin-Ideal Internalization

Internalization of societal thinness-based appearance ideals was measured using the Internalization: Thin/Low Body Fat subscale of the SATAQ-4R (Schaefer et al., 2017). Items were scored on a 5-point scale ranging from 1 (*definitely disagree*) to 5 (*definitely agree*), with higher scores indicating greater internalization. Previous studies have found an internal consistency of $\alpha = .75$ in men and $\alpha = .82$ in women (Schaefer et al., 2017). The SATAQ-4R has been previously validated in SMM and SMW (Convertino et al., 2019). Internal consistency in the current study was $\alpha = .77$ for SMM and $\alpha = .81$ for SMW.

Muscular-Ideal Internalization

Internalization of societal muscularity-based appearance ideals was measured using the Internalization: Muscular subscale of the SATAQ-4R (Schaefer et al., 2017). Items were scored on a 5-point scale ranging from 1 (*definitely disagree*) to 5 (*definitely agree*), with higher scores indicating greater internalization. Previous studies have found an internal consistency of $\alpha = .87$ in men and $\alpha = .91$ in women (Schaefer et al., 2017). The SATAQ-4R has been previously validated in SMM and SMW (Convertino et al., 2019). Internal consistency in the current study was $\alpha = .88$ for SMM and $\alpha = .88$ for SMW.

Thinness-Based Dissatisfaction

Thinness-based body dissatisfaction was measured using the Eating Disorder Examination Questionnaire 6.0 gender invariant version (EDE-Q; Fairburn & Beglin, 2008; Rand-Giovannetti et al., 2020). Factor 3, Weight and Shape Concern, was utilized for these

analyses. This subscale includes 11 items scored on a 7-point scale ranging from 0 (*no days*) to 6 (*every day*) of the last 28 days, with higher scores indicating greater body dissatisfaction.

Previous studies have found an internal consistency of $\alpha = .86$ (Friborg et al., 2013). The internal consistency for the current study was $\alpha = .93$ for SMM and $\alpha = .93$ for SMW.

Muscularity-Based Dissatisfaction

Dissatisfaction with one's muscularity was measured using the 7-item Muscle-Oriented Body Image subscale of the Drive for Muscularity Scale (DMS; McCreary & Sasse, 2000). Items were scored on a 6-point scale ranging from 1 (*never*) to 6 (*always*), with higher scores indicating higher muscularity-based dissatisfaction. Previous studies that have validated the DMS in SMM (DeBlaere & Brewster, 2017) and SMW (Klimek et al., in press). Previous studies found an internal consistency of $\alpha = .93$ in SMM (DeBlaere & Brewster, 2017). The internal consistency in the current study was $\alpha = .91$ for SMM and $\alpha = .90$ for SMW.

Dietary Restraint

Dietary restraint was measured using the EDE-Q (Fairburn & Beglin, 2008; Rand-Giovannetti et al., 2020). Factor 1, Dietary Restraint, was utilized for these analyses. This subscale includes three items scored on a 7-point scale ranging from 0 (*no days*) to 6 (*every day*) of the last 28 days, with higher scores indicating greater dietary restraint. Previous studies have found an internal consistency of $\alpha = .78$ (Friborg et al., 2013). The internal consistency for the current study was $\alpha = .83$ for SMM and $\alpha = .84$ for SMW.

Muscle Building Behaviors

Engagement in behaviors associated with the desire to be muscular was measured using the 7-item Muscle-Oriented Behavior subscale of the DMS (McCreary & Sasse, 2000). Items were scored on a 6-point scale ranging from 1 (*never*) to 6 (*always*), with higher scores

indicating higher engagement in muscle building behaviors. Previous studies that have validated the DMS in SMM (DeBlaere & Brewster, 2017) and SMW (Klimek et al., in press). Previous studies found an internal consistency of $\alpha = .87$ in SMM (DeBlaere & Brewster, 2017). The internal consistency in the current study was $\alpha = .88$ for SMM and $\alpha = .88$ for SMW.

Internalized Homophobia

Internalized homophobia was measured using the five item Internalized Homophobia Scale-Revised (IHP-R; Herek et al., 2009). The IHP-R measures the degree to which an individual internalizes anti-gay societal attitudes towards them, scored on a 5-point scale ranging from 1 (*disagree strongly*) to 5 (*agree strongly*). Higher scores indicate greater internalization of negative self-attitudes. Previous research has found an internal consistency of $\alpha = .83$ for SMM and $\alpha = .71$ for SMW (Herek et al., 2009). The internal consistency in the current study was $\alpha = .87$ for SMM and $\alpha = .87$ for SMW.

Heterosexist Discrimination

Heterosexist discrimination was measured using the 14-item Heterosexist Harassment, Rejection, and Discrimination Scale (HHRDS; Szymanski, 2006). The HHRDS measures SM individuals' experiences with a variety of heterosexist events within the previous year, scored on a 6-point scale ranging from 1 (*never*) to 6 (*almost all of the time*). Higher scores indicate greater heterosexist experiences. Previous research has identified an internal consistency of $\alpha = .90$ in SMW (Szymanski, 2006) and $\alpha = .91$ in SMM (Szymanski, 2009). The internal consistency for the total score in the current study was $\alpha = .95$ for SMM and $\alpha = .95$ for SMW.

Sexual Orientation Concealment

Sexual orientation Concealment was measured using the 6-item Sexual Orientation Concealment Scale (SOCS; Jackson & Mohr, 2016). The SOCS measures the degree to which an

individual attempts to conceal their own sexual identity within the previous two weeks. Items were scored on a 5-point scale ranging from 1 (*not at all*) to 5 (*all the time*), with higher scores indicating greater sexual orientation concealment. Previous research has found an internal consistency of $\alpha = .78$ in a mixed gender sample (Jackson & Mohr, 2016). The internal consistency in the current study was $\alpha = .87$ for SMM and $\alpha = .88$ for SMW.

Sexual Minority Community Involvement

SM community involvement was measured using 6-items adapted from the Social Justice Sexuality Project (SJP; Harris et al., 2013), which is a national survey of lesbian, gay, bisexual, and transgender (LGBT) people. The items measure the degree to which an individual participates in the LGBT community (e.g., used the internet, participated in social or cultural events, read newspapers or magazines) within the previous year. Items were scored on a 5-point scale ranging from 1 (*never*) to 5 (*about once a week*), with higher scores indicating greater community involvement. The internal consistency in the current study was $\alpha = .77$ for men and $\alpha = .80$ for women.

Statistical Analysis

Latent variable structural equation modeling (SEM) via R package lavaan (Rosseel, 2012) with full information maximum likelihood estimation was employed to estimate parameter in both models. Due to differing items for SMM and SMW on the SATAQ-4R, models were estimated separately by gender because these models could not be integrated into a single model without untenable measurement assumptions.

Parceling Strategy

Items from the peers, family, significant others, and media subscales were averaged within subscales and then used as indicators of a latent pressures factor. For dietary restraint

(with three items) and for thin-ideal internalization in men (with two items), a latent variable was created by allowing its items to estimate it (i.e., a parceling strategy was not used). For all other latent variables, the single factor analysis parceling strategy was used to create three parcels per latent variable (Landis et al., 2000). This approach was chosen because an exploratory factor analysis (EFA) indicated that for each theorized latent variable, the items comprised a single factor and previous research has indicated that this approach performs well when items are unidimensional (Landis et al., 2000; Rogers & Schmitt, 2004). In this approach, the item with the highest loading was paired with the item with the lowest loading, the next highest and the next lowest are paired, continuing until six items are paired into three parcels. If there were more than six items, the seventh was placed on the third parcel, the eighth on the second parcel, continuing until all items are allocated to a parcel.

Interaction Specification and Testing

Interactions were specified using the product-indicator approach as specified by Kenny and Judd (1984). The parcels of each latent variable that were theorized to interact were multiplied by each other such that all possible products were created. These cross-products were then specified to load onto a latent interaction variable. For each significant interaction variable, a simple slopes analysis (Aiken & West, 1991) was conducted to compare the relationships between the moderator and the criterion at low (-1 SD) and high ($+1$ SD) levels of community involvement.

Model Fit

Model fit was determined via consensus among three indices: Comparative Fit Index (CFI), standardized root mean square residual (SRMR), and root mean square error of approximation (RMSEA). Guidelines for acceptable fit within SEM are an ongoing discussion

amongst statisticians. Current guidance suggests that CFI values should meet or exceed .95, SRMR values should be less than or equal to .08, and RMSEA values should be less than or equal to .06 to indicate that a model provides an adequate fit to the data (Hu & Bentler, 1999; Marsh et al., 2004). However, these same authors suggest that when sample sizes are less than 500 and models are complex, less stringent criteria should be used; specifically that CFI values greater than or equal to .90, RMSEA values less than or equal to .10, and SRMR values less than or equal to .10 indicate adequate fit (Hu & Bentler, 1999; Marsh et al., 2004). Therefore, the less stringent criteria was utilized for the current study because of the model complexity as well as sample sizes less than 500 for both men and women.

Results

Model for SMM

Means, standard deviations, and correlations for study variables are included in Table 3. All variables had less than 1% missing data. Data were examined for normality. All items and parcel indicators utilized in the current model were lower than the skewness (>3) and kurtosis (>10) values recommended by Kline (2010), and were therefore not transformed (skewness range = -0.49 to 0.92; kurtosis range = -1.09 to -0.01). Data was examined for outliers using Mahalanobis distance (Leys et al., 2018). When potential outliers were removed from the sample, parameter estimates were within .03 of the results with outliers included.. Therefore, analyses with the full sample were retained. Parcels and items were internally consistent (α range = .77 to .95; α average = .86) and moderately correlated (r range = .14 to .73, r average = .35). The current study ($N = 479$) exceeded the number cases recommended for internally consistent and moderately correlated indicators (≥ 200 ; Weston & Gore, 2006).

Examination of the measurement model

The measurement model for SMM provided an acceptable fit to the data (CFI = .937, SRMR = .070, RMSEA = .051 [95% CI: .048-.053]). All item/parcel loadings were significant ($p < .001$). See Figure 2 for loadings.

Examination of the structural model

The structural model for SMM provided an acceptable fit to the data (CFI = .928, SRMR = .092, RMSEA = .046 [95% CI: .044-.048]). The model explained 61.7% of the variance in dietary restraint and 58.4% of the variance in muscle-building behaviors. Of the interaction effects, only one significant path emerged: the path from the interaction of community involvement and heterosexist discrimination to internalized homophobia ($\beta = .216, p < .001$). Simple slope analyses indicated that the effect of heterosexist discrimination on internalized homophobia was strongest among SMM with community involvement 1 standard deviation above the mean ($\beta = .473, z = 5.955, p < .001$). Further, heterosexist discrimination was not significantly associated with internalized homophobia among SMM with community involvement 1 standard deviation below the mean ($\beta = .041, z = 0.880, p = .379$). While non-significant paths and interactions were not removed from the tested model, see Figure 3 for model with nonsignificant paths removed for parsimony.

Model for SMW

Means, standard deviations, and correlations for study variables are included in Table 4. All variables had less than 1% missing data. Data were examined for normality. All items and parcel indicators utilized in the current model were lower than the skewness (>3) and kurtosis (>10) values recommended by Kline (2010) and were therefore not transformed (skewness range = -0.38 to 1.57; kurtosis range = -1.17 to 1.81). Data was examined for outliers using Mahalanobis distance (Leys et al., 2018). When potential outliers were removed from the

sample, parameter estimates were within .03 of the results with outliers included. Therefore, analyses with the full sample were retained. Parcels and items were internally consistent (α range = .73 to .95; α average = .86) and moderately correlated (r range = .08 to .69, r average = .31). The current study ($N = 483$) exceeded the number cases recommended for internally consistent and moderately correlated indicators (≥ 200 ; Weston & Gore, 2006).

Examination of the measurement model

The measurement model for SMW provided an acceptable fit to the data (CFI = .927, SRMR = .123, RMSEA = .057 [95% CI: .054-.059]). All item/parcel loadings were significant ($p < .001$). See Figure 4 for loadings.

Examination of the structural model

The structural model for SMW provided an acceptable fit to the data (CFI = .922, SRMR = .126, RMSEA = .049 [95% CI: .047-.051]). The model explained 48.9% of the variance in dietary restraint and 75.6% of the variance in muscle-building behaviors. Of the interaction effects, four significant paths emerged. The path from the interaction of community involvement and pressures to muscular-ideal internalization was significant ($\beta = .184, p < .001$). Simple slope analyses indicated that the effect of pressures on muscular-ideal internalization was strongest among SMW with community involvement 1 standard deviation above the mean ($\beta = .542, z = 6.736, p < .001$). Pressures were not as strongly associated with muscular-ideal internalization among SMW with community involvement 1 standard deviation below the mean ($\beta = .175, z = 2.497, p = .013$). The path from the interaction of community involvement and heterosexist discrimination to internalized homophobia was significant ($\beta = .252, p < .001$). Simple slope analyses indicated that the effect of heterosexist discrimination on internalized homophobia was strongest among SMW with community involvement 1 standard deviation above the mean (β

= .571, $z = 7.002$, $p < .001$). Further, heterosexist discrimination was not significantly associated with internalized homophobia among SMW with community involvement 1 standard deviation below the mean ($\beta = .067$, $z = 1.124$, $p = .261$). The path from the interaction of community involvement and heterosexist discrimination to sexual orientation concealment was significant ($\beta = .107$, $p = .046$). Simple slope analyses indicated that the effect of heterosexist discrimination on sexual orientation concealment was strongest among SMW with community involvement 1 standard deviation above the mean ($\beta = .532$, $z = 6.994$, $p < .001$). Heterosexist discrimination was not as strongly associated with sexual orientation concealment among SMW with community involvement 1 standard deviation below the mean ($\beta = .319$, $z = 3.551$, $p < .001$). The path from the interaction of community involvement and muscularity-based dissatisfaction to muscularity behaviors was significant ($\beta = .170$, $p < .001$). Simple slope analyses indicated that the effect of muscularity-based dissatisfaction on muscularity behaviors was strongest among SMW with community involvement 1 standard deviation above the mean ($\beta = .768$, $z = 10.272$, $p < .001$). Muscularity-based dissatisfaction was not as strongly associated with muscularity behaviors among women with community involvement 1 standard deviation below the mean ($\beta = .428$, $z = 6.809$, $p < .001$). While non-significant paths and interactions were not removed from the tested model, see Figure 5 for model with nonsignificant paths removed for parsimony.

Discussion

The aim of the current study was to integrate the tripartite influence model (Thompson, Heinberg, et al., 1999) and minority stress theory (Meyer, 2003) into a cohesive theoretical framework for explaining eating pathology among SM individuals. Overall, the model explained 61.7% and 58.4% of the variance for dietary restraint and muscle-building behaviors,

respectively, in SMM and 48.9% and 75.6% of the variance for dietary restraint and muscle-building behaviors, respectively, in SMW. The current model explains greater variance than previously reported for the tripartite model (47.1% and 33.5% of the variance for disordered eating behaviors and muscle-building behaviors; Tylka & Andorka, 2012) and models utilizing minority stress theory (Mason et al., 2017; 17.4% to 38% in disordered eating; Mason & Lewis, 2015, 2016; Watson et al., 2016). Therefore, the current model demonstrated an improvement in variance explained, over and above previous models.

Findings Relevant to the Tripartite Influence Model

Aspects of the full model that are specific to the tripartite influence model as specified by Thompson et al. (1999) were supported, namely that paths from pressures to thin-ideal internalization, from thin-ideal internalization to thinness-based dissatisfaction, and from thinness-based dissatisfaction to dietary restraint were all significant, positive, and at least of moderate strength (.38-.65 in SMW and .41-.70 in SMM). These findings bolster prior research in SMM (Tylka & Andorka, 2012) and SMW (Hazzard et al., 2019) that found support for the tripartite influence model in SM individuals. The current study also found support for a modified version of the tripartite influence model that includes dual body image pathways to body change behaviors. This additional pathway was supported in both SMM and SMW; specifically, that paths from pressures to muscular-ideal internalization, from muscular-ideal internalization to muscularity-based dissatisfaction, and from muscularity-based dissatisfaction to muscle building behaviors were all significant, positive, and at least of moderate strength (.36-.69 in SMW and .43-.69 in SMM). Thus, these findings replicate prior work in SMM that found support for dual body image pathways (Tylka & Andorka, 2012). Furthermore, this is the first study, to our knowledge, to support the dual body image pathway modification in SMW. This finding is not

completely unexpected as prior research has noted an increase in the desire for a lean and toned body among young women (Robinson et al., 2017; Tiggemann & Zaccardo, 2018). This study provides preliminary evidence that these pathways also exist in SMW.

Findings Relevant to Minority Stress Theory

Aspects of minority stress theory (Meyer, 2003) were supported within the model as well. For SMM and SMW, the paths from heterosexist discrimination to sexual orientation concealment and internalized homophobia were significant, positive, and of small size among SMM and moderate size among SMW, indicating that greater distal stressors (i.e., heterosexist discrimination) are associated with greater proximal stressors (i.e., sexual orientation concealment and internalized homophobia), as hypothesized by minority stress theory. Furthermore, the paths from (1) internalized homophobia to muscle building behaviors in SMM and SMW, (2) heterosexist discrimination and sexual orientation concealment to thinness-based dissatisfaction in SMM, and (3) heterosexist discrimination to muscularity-based dissatisfaction in SMW were significant, positive, and of small size, even when including other eating- and body image-specific influences. This finding supports the hypothesis of minority stress theory that SM stressors will lead to poor mental health outcomes, and, further, is in line with prior research finding similar effects of SM stressors on eating and body image outcomes (e.g., Mason & Lewis, 2015; Wang & Borders, 2017; Watson et al., 2016).

However, minority stress theory also states that social support, including that of the SM community, would mitigate the negative mental health effects of minority stressors. There was no support for a mitigating effect of community involvement on the association between heterosexist discrimination and sexual orientation concealment. There was a significant interaction effect of community involvement and heterosexist discrimination on internalized

homophobia for both SMM and SMW, but this effect was such that for individuals that experience greater discrimination, involvement in the community is significantly associated with greater internalized homophobia with medium to large effects. In addition, there was a significant interaction between community involvement and heterosexist discrimination for SMW, such that for individuals that experience greater discrimination, involvement in the community increases sexual orientation concealment. This is contrary to minority stress hypotheses and has not been explored thus far in the literature. One potential explanation for this unexpected finding may be that individuals who endorse high discrimination may be experiencing the “black sheep effect.” The black sheep effect states that individuals of an ingroup judge their fellow ingroup members’ behavior more harshly than comparable outgroup members when the ingroup members’ behavior may reflect poorly on the individual (Marques et al., 1988). Utilizing this framework, it may be that individuals who experience discrimination reach out to the SM community for support. If they do not receive the expected support, and perhaps instead experience intraminority stressors, this could heighten internalized anti-LGBT attitudes that have been propagated and communicated through overt discrimination. The current study was not designed to examine such psychological processes; therefore, future research would benefit from formally testing the black sheep effect and other frameworks in better understanding the unexpected effect of community involvement in SM stressors.

The association between community involvement and body image-related outcomes was complicated by seemingly contradictory findings. In both men and women, community involvement was significantly and positively associated with greater dietary restraint and muscle building behaviors of small-to-medium size (range: .12-.30). Furthermore, the bivariate correlations indicated that both muscularity-based dissatisfaction and thinness-based

dissatisfaction were significantly and positively associated with community involvement of small-to-medium size (range: .14-.46). This may lead to the conclusion that community involvement is associated with greater body dissatisfaction and eating disordered behaviors. Contrarily, in the overall model, community involvement was negatively associated with muscularity-based dissatisfaction in SMM with moderate strength and negatively associated with thinness-based dissatisfaction in SMM and SMW (path coefficients were -.36 and -.23 respectively). This somewhat contradictory finding can most likely be attributed to the variance explained by the other latent variables in the model. Bivariate correlations are the total association between two variables, or their association without accounting for other effects that might influence this relationship (Brown, 2015). When included in a structural equation model, the association between two variables is now the unique (i.e., partial) association, removing the effect of all other variables. Stated differently, when covarying out shared variance with all SM stress variables, and internalization of the appearance ideal and pressures to conform to that ideal, greater community involvement is significantly associated with lower muscularity-based dissatisfaction in men and women and lower thinness-based dissatisfaction in men. This may suggest that, in a hypothetical world where there are no effects of SM stressors or sociocultural pressures to appear a certain way, being more involved with the SM community is helpful for one's body satisfaction. Therefore, it appears that the association between community involvement and body dissatisfaction is complicated and, at least in the current study, varied substantially depending on the analytic approach.

Differences by Gender

Some findings relevant to the dual pathway model differed by gender. First of note, there were two significant paths in women that were non-significant in men: 1) a negative path

between muscular-ideal internalization and thinness-based dissatisfaction of small size and 2) a negative path between thinness-based dissatisfaction and muscle building behavior of small size. These two paths could exist for women and not for men because of differences in the body ideal and subsequent associated behaviors. Men are more likely to pursue a body ideal characterized by both muscularity and leanness, which is thought to improve the appearance of muscularity. Therefore, men may internalize the muscular ideal, but pursue both muscularity and thinness through simultaneous or quick alternating “bulking” and “cutting” phases (Murray et al., 2016), which are less likely in women.

There were also differences by gender for aspects relevant to minority stress theory. In men, there were two additional significant paths: 1) a small, positive main effect of community involvement on sexual orientation concealment, and 2) a small, positive main effect of community involvement on internalized homophobia (but this was qualified by a significant interaction, such that this association was not significant at low levels of community involvement; therefore, this main effect is somewhat less relevant). In women, there were no statistically significant main effects of community involvement on sexual orientation concealment or internalized homophobia. Rather, the interactions were significant, such that the association between heterosexist discrimination and sexual orientation concealment, as well as the association between heterosexist discrimination and internalized homophobia, were strongest with a large effect size at higher levels of community involvement. Therefore, while it appears that community involvement may serve to moderate these association for women, community involvement is associated with greater proximal stressors at high levels.

In women, there were two additional paths: 1) a small, positive path between community involvement and muscular-ideal internalization (this was qualified by a significant interaction,

described below), and 2) a small, positive path between heterosexist discrimination and muscle building behavior. There were also two additional interaction effects such that community involvement accelerated the association of pressures on muscular-ideal internalization and muscularity-based dissatisfaction on muscle building behaviors. Given the dearth of research on factors within the SMW community that may explain such an association, it is unclear why greater community involvement would lead to an acceleration of muscular-ideal internalization and muscle building behaviors. One explanation is that gender expression in SMW communities is diverse and, as such, there is more room for engagement in traditionally masculine building activities as compared to heterosexual communities. For example, prior research has found that greater levels of masculine/butch expression in SMW was associated with lower levels of thin ideal internalization (Henrichs-Beck & Szymanski, 2017). It may be that masculine/butch SMW may identify with a more masculine ideal and thus engage in more muscle building behaviors when they feel that these behaviors and ideals are acceptable within the SMW community. Though these results were not significant in men, it should be noted that results were trending in the same direction as women, such that men who were highly involved in the community and endorsed high pressures experienced greater muscular-ideal internalization and men with high muscularity-based dissatisfaction who were highly involved in the community experienced greater muscle building behaviors. Therefore, it is possible that these interaction effects exist for men but there was such a large effect size for pressures and muscularity-based dissatisfaction, respectively, that there was not much additional variance to predict. Overall, it appears that community involvement may accelerate the association of pressures on internalization and dissatisfaction on muscle building behaviors for both SMM and SMW, but this finding was not supported statistically in men.

Importance of Community Involvement for SM Adults

In light of the moderation effects found in the current study, examining community involvement within body image models for SM populations is of paramount importance. Examinations of community involvement are often complicated by the complexity of the construct. Indeed, researchers have tried to parse out the community construct into *community connectedness*, reflecting cognitive identification and solidarity with the community, and *community participation*, reflecting concrete behaviors such as attending social events or professional groups (Frost & Meyer, 2012). Prior research in SM populations has indicated a stronger association between community participation and substance use as opposed to between community connectedness and substance use (Demant et al., 2018; Demant & Oviedo-Trespalacios, 2019); however, it is unclear whether the association between community participation and eating disordered behavior would be similarly stronger than community connectedness and eating disordered behavior. Since the items in the current study are behaviorally focused, future research may consider examining how these results may change when considering community connectedness as opposed to behavioral participation.

Limitations

The current study has some limitations of note. First, the cross-sectional nature of the study's design precludes conclusions of temporality. Future studies should address this limitation by examining this model with longitudinal research designs. Second, while the current study was only designed to integrate two theories, there are other relevant theories and mechanisms that may be relevant for future work. For example, objectification theory (Fredrickson & Roberts, 1997) may be relevant given prior findings on objectification experiences within the SM community (Davids et al., 2015). Finally, the current study relied on self-report. Future research

may integrate clinician-based measures to establish the validity of eating pathology reported.

Despite these limitations, the current study may also offer important considerations to the field.

Implications for Theory in Eating Disorders

The current study has implications for theoretical work in eating disorders. First, the current study follows calls from researchers to create a comprehensive, integrated model of body image and disordered eating in SM individuals (Mason et al., 2018). Including both the tripartite model and minority stress theory has promise for explaining disparities in disordered eating by sexual orientation (Calzo et al., 2017). The integration provided in the current study can therefore inform future investigations of disordered eating within this population. This study also highlights the importance of including minority stress theory within investigations of eating pathology for SM individuals. Second, the current study complicates the association between SM communities and mental health outcomes, as stated in minority stress theory. While SM communities can no doubt have a mitigating effect for some mental health outcomes (e.g., Griffin et al., 2018; Lambe et al., 2017; Salfas et al., 2019), it should be noted that this does not appear to be the case for eating pathology in this sample. Uniformly, greater community involvement was associated with greater dietary restraint and muscle building behaviors with mostly small effect sizes. This finding is in direct contrast to minority stress theory, which considers community involvement to be a protective factor against psychopathology (Meyer, 2003). When considering the interaction effects observed in the current study, community involvement does not seem buffer against negative outcomes and, in some cases, may actually contribute to the negative effect of distal stressors on proximal stressors. Third, and somewhat more broadly, this study highlights the importance of testing latent interactions in structural equation modeling. Without testing these interactions, the accelerative association of community

involvement on outcomes would not have been observed. Therefore, failing to examine interaction effects in these models could result in misspecified models and may have implications for clinical work. For example, if heterosexist discrimination is only associated with internalized homophobia for those with high community involvement, then targeting an intervention widely in the community for internalized homophobia may be less fruitful than targeting an intervention with only those who participate regularly.

Conclusion

The current research was the first known study to empirically integrate the tripartite influence model and minority stress theory into a cohesive, testable model in SMM and SMW. Minority stressors were positively associated with greater thinness-based dissatisfaction (in men only), muscularity-based dissatisfaction, and muscle building behaviors. Furthermore, SM community involvement was associated with greater dietary restraint and muscle building behaviors in men and women, and accelerated the association of muscularity-based dissatisfaction with muscle building behaviors in SMW. Future research should examine this model longitudinally to ascertain if theoretical causal pathways are supported.

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Tables

Table 1.1: *Demographics of the sample*

Characteristic	Total (N = 962) N (%)	Men (n = 479) N (%)	Women (n = 483) N (%)
Sexual Identity			
Gay/Lesbian	336 (34.9)	239 (49.9)	97 (20.1)
Bisexual	564 (58.6)	206 (43.0)	358 (74.1)
Asexual	20 (2.1)	10 (2.1)	10 (2.1)
Other ^a	42 (4.4)	24 (5.0)	18 (3.7)
Sexual Attraction			
Only Same Sex	295 (30.7)	203 (42.4)	92 (19.0)
Mostly Same Sex	142 (14.8)	89 (18.6)	53 (11.0)
Equally Opposite and Same Sex	525 (54.6)	187 (39.0)	338 (70.0)
Race^b			
White	371 (38.6)	184 (38.4)	187 (38.7)
Black/African American	294 (30.6)	146 (30.5)	148 (30.6)
Native American/American Indian	23 (2.4)	13 (2.7)	10 (2.1)
Asian/Pacific Islander	272 (28.3)	134 (28.0)	138 (28.6)
Ethnicity			
Hispanic/Latino	234 (24.3)	120 (25.1)	114 (23.6)
Non-Hispanic/Latino	728 (75.7)	359 (74.9)	369 (76.4)

^a Other reported sexual identities included pansexual, panromantic, omnisexual, demisexual, queer, and straight. ^b Two men (0.4% of the sample) did not report race.

Table 1.2: Means and standard deviations [*M* (*SD*)] of study variables by gender and sexual orientation

	Men (n = 479)				Women (n = 483)			
	Gay	Bisexual	Asexual	Other	Lesbian	Bisexual	Asexual	Other
Pressure	2.84 (1.01)	2.74 (0.91)	2.77 (0.67)	2.58 (0.84)	2.68 (1.02)	2.84 (1.00)	2.24 (0.97)	2.87 (0.87)
Heterosexist Discrimination	2.46 (1.18)	2.24 (1.14)	2.05 (0.92)	2.38 (1.20)	2.58 (1.27)	2.08 (1.16)	2.53 (1.20)	2.04 (1.00)
Community Involvement	2.91 (1.14)	2.83 (1.02)	2.93 (0.76)	2.64 (1.11)	2.91 (1.23)	2.72 (1.10)	3.37 (1.08)	2.64 (1.05)
Sexual Orientation Concealment	2.62 (1.13)	2.71 (1.00)	2.43 (0.64)	2.37 (1.02)	2.25 (1.09)	2.32 (1.09)	2.38 (1.16)	1.89 (0.93)
Internalized Homophobia	2.13 (1.07)	2.34 (1.10)	2.34 (0.92)	1.89 (0.88)	1.80 (0.96)	1.89 (1.01)	1.74 (1.28)	1.68 (0.72)
Thin-Ideal Internalization	3.13 (1.16)	2.93 (1.11)	3.20 (1.16)	2.52 (1.03)	3.11 (1.01)	3.30 (1.04)	3.10 (0.72)	3.66 (1.11)
Muscular-Ideal Internalization	3.28 (1.13)	3.10 (1.03)	2.63 (1.06)	2.52 (1.12)	2.97 (1.10)	2.45 (1.05)	2.35 (1.11)	2.40 (1.06)
Thinness-Based Dissatisfaction	2.71 (1.76)	2.68 (1.63)	3.48 (1.35)	2.65 (1.88)	2.75 (1.78)	3.29 (1.73)	2.53 (1.84)	4.02 (1.73)
Muscularity-Based Dissatisfaction	3.55 (1.37)	3.39 (1.30)	3.64 (1.45)	2.92 (1.57)	2.97 (1.34)	2.52 (1.22)	3.00 (1.91)	2.30 (1.22)
Dietary Restraint	1.96 (1.87)	1.91 (1.57)	3.00 (1.87)	1.75 (1.73)	2.05 (1.89)	2.20 (1.82)	2.33 (1.98)	2.17 (2.02)
Muscle Building Behavior	2.84 (1.01)	2.74 (0.91)	2.77 (0.67)	2.58 (0.84)	2.35 (1.16)	1.97 (1.06)	2.10 (1.61)	1.49 (0.71)

Note. M and SD are used to represent mean and standard deviation, respectively.

Table 1.3: Means, standard deviations, and correlations among study variables for sexual minority men

Variable	M	SD	1	2	3	4	5	6	7	8	9	10
1. Pressures	2.78	0.96										
2. Heterosexist Discrimination	2.35	1.16	.45**									
3. Community Involvement	2.86	1.08	.38**	.44**								
4. Sexual Orientation Concealment	2.64	1.06	.39**	.38**	.26**							
5. Internalized Homophobia	2.21	1.07	.39**	.39**	.31**	.52**						
6. Thin-Ideal Internalization	3.01	1.14	.46**	.20**	.16**	.23**	.17**					
7. Muscular-Ideal Internalization	3.15	1.10	.44**	.14**	.20**	.26**	.23**	.29**				
8. Thinness-Based Dissatisfaction	2.71	1.70	.48**	.35**	.24**	.30**	.20**	.54**	.28**			
9. Muscularity-Based Dissatisfaction	3.45	1.35	.49**	.25**	.28**	.28**	.28**	.21**	.73**	.31**		
10. Dietary Restraint	1.95	1.74	.35**	.32**	.29**	.27**	.23**	.37**	.19**	.65**	.24**	
11. Muscle Building Behavior	2.55	1.20	.42**	.43**	.46**	.34**	.47**	.16**	.43**	.22**	.53**	.32**

Note. M and SD are used to represent mean and standard deviation, respectively. * indicates $p < .05$. ** indicates $p < .01$.

Table 1.4: Means, standard deviations, and correlations among study variables for sexual minority women

Variable	M	SD	1	2	3	4	5	6	7	8	9	10
1. Pressures	2.80	1.00										
2. Heterosexist Discrimination	2.19	1.19	.35**									
3. Community Involvement	2.77	1.13	.29**	.52**								
4. Sexual Orientation Concealment	2.29	1.09	.39**	.41**	.25**							
5. Internalized Homophobia	1.86	1.00	.32**	.39**	.26**	.56**						
6. Thin-Ideal Internalization	3.27	1.03	.50**	.15**	.16**	.22**	.15**					
7. Muscular-Ideal Internalization	2.50	1.07	.28**	.31**	.19**	.25**	.25**	.25**				
8. Thinness-Based Dissatisfaction	3.19	1.76	.52**	.17**	.15**	.17**	.12**	.57**	.10*			
9. Muscularity-Based Dissatisfaction	2.61	1.27	.30**	.40**	.25**	.30**	.30**	.21**	.74**	.21**		
10. Dietary Restraint	2.17	1.84	.36**	.18**	.17**	.14**	.08	.35**	.12**	.56**	.18**	
11. Muscle Building Behavior	2.03	1.10	.31**	.56**	.46**	.38**	.49**	.14**	.49**	.12**	.67**	.23**

Note. M and SD are used to represent mean and standard deviation, respectively. * indicates $p < .05$. ** indicates $p < .01$.

Figures

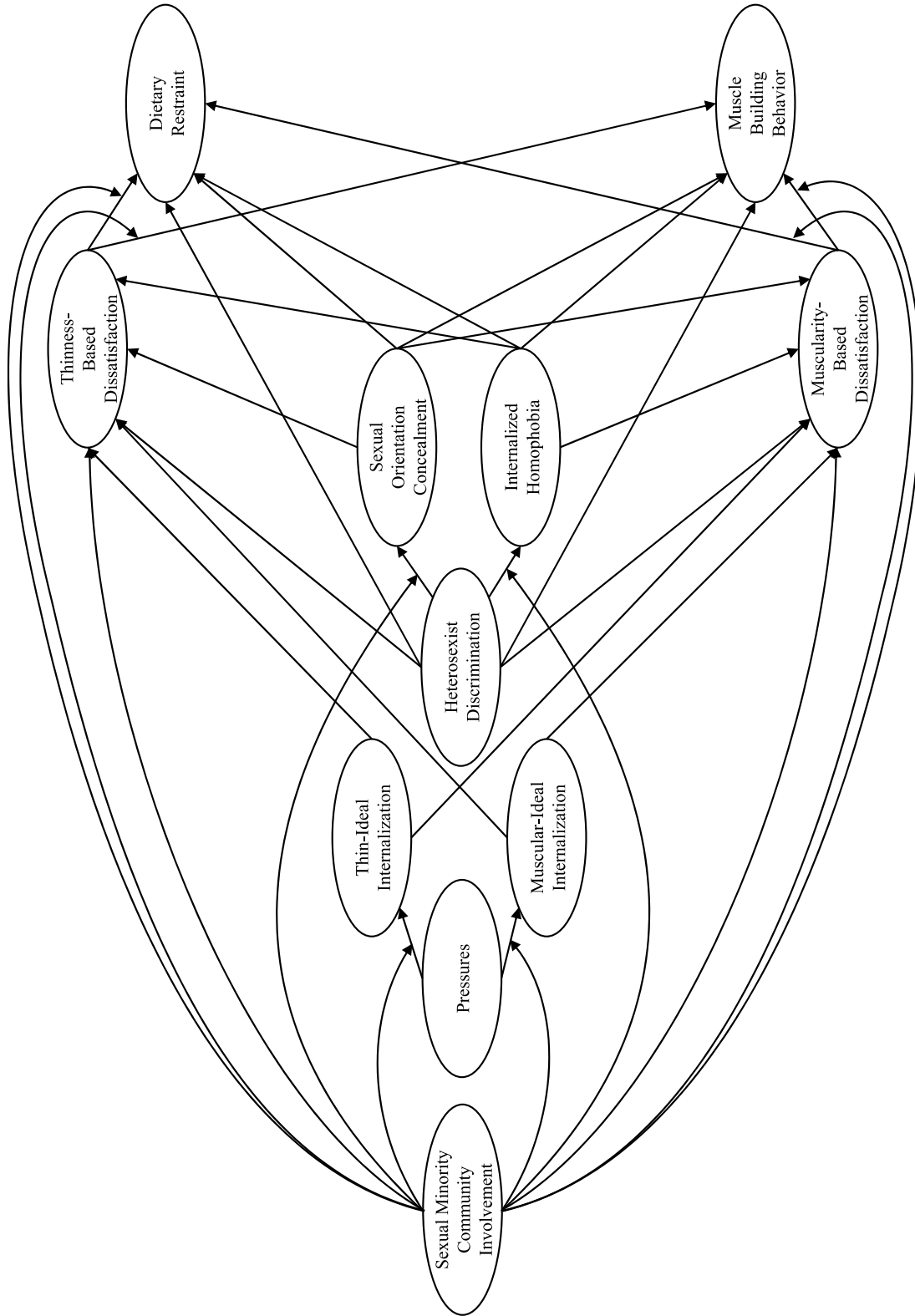


Figure 1.1: Theoretical integrated model of eating pathology in sexual minority men and women

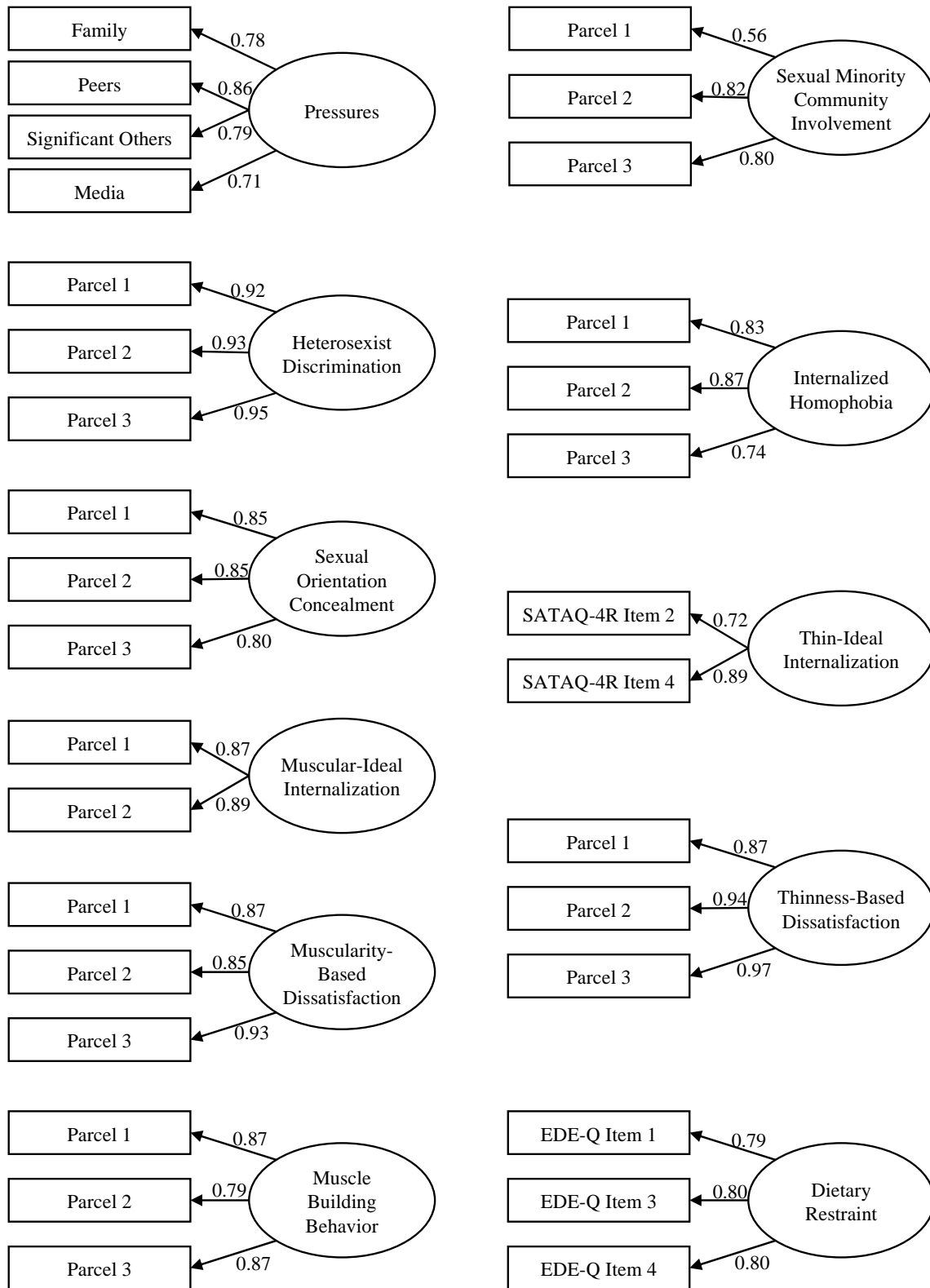


Figure 1.2: Factor loadings for sexual minority men

Note. All loadings $p < .001$.

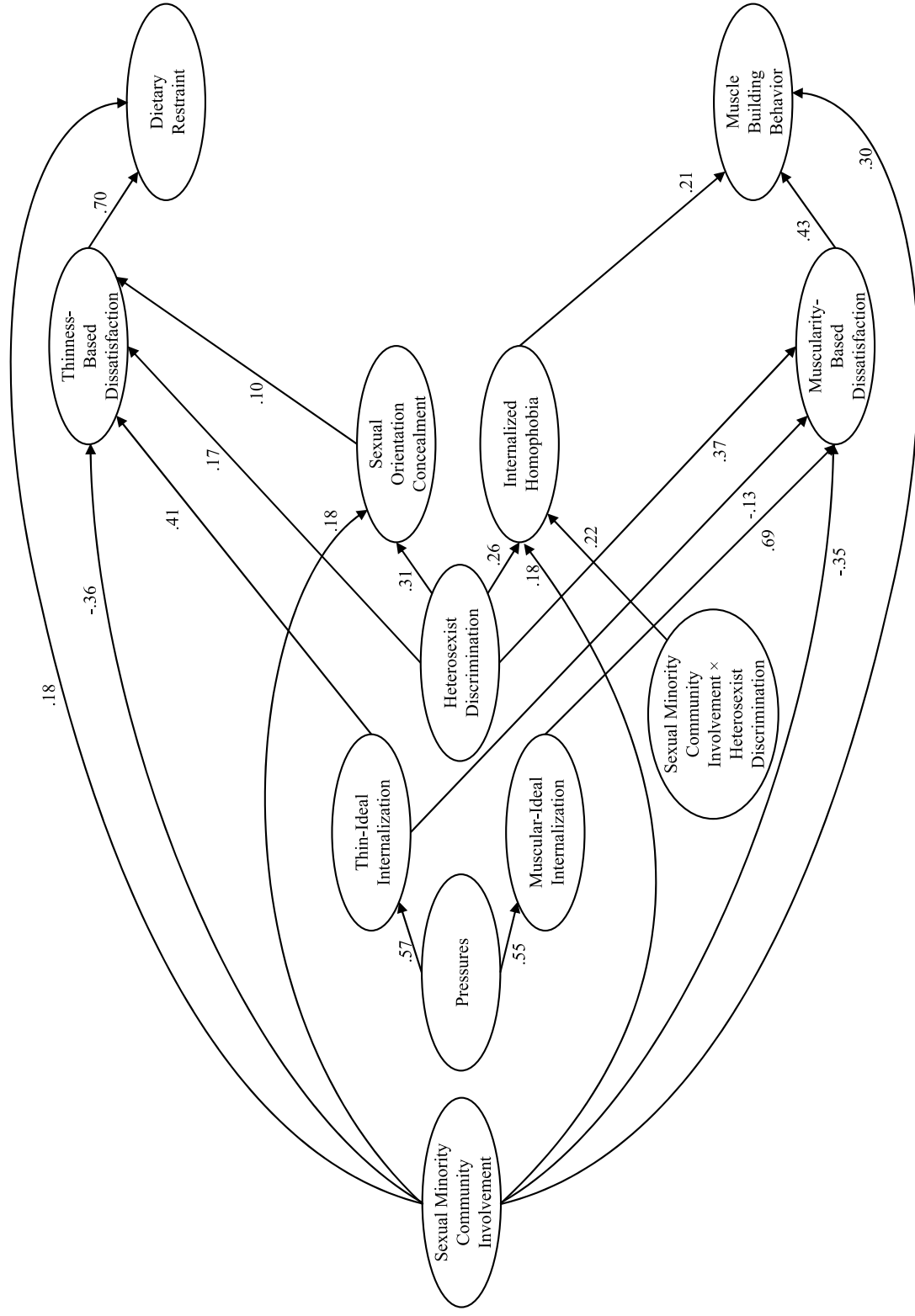


Figure 1.3: Structural equation modeling results in sexual minority men

Note. Non-significant tested paths are removed from the figure for parsimony, but are not removed from overall model. All paths shown are significant at $p > .05$.

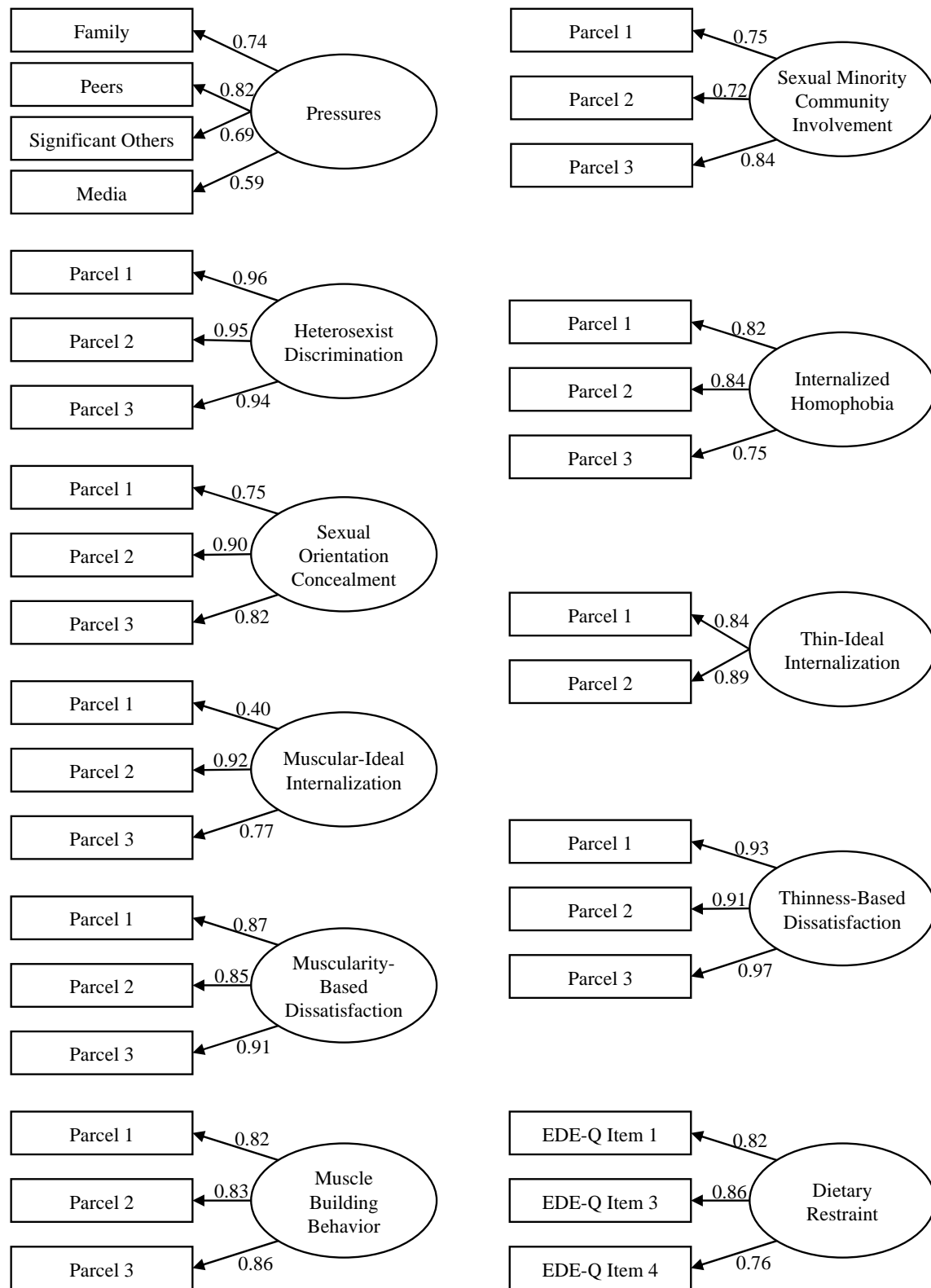


Figure 1.4: Factor loadings for sexual minority women

Note. All loadings $p < .001$

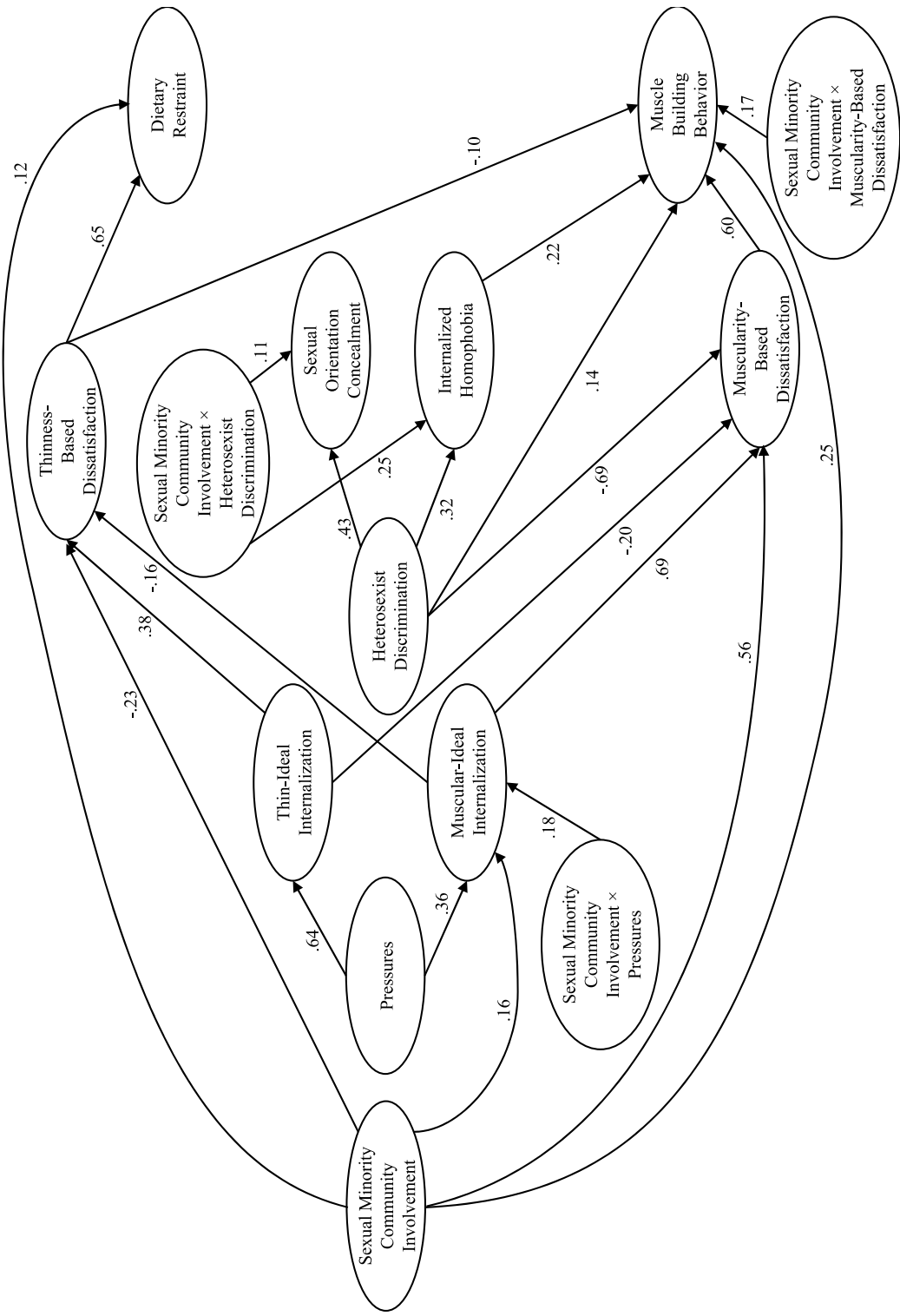


Figure 1.5: Structural equation modeling results in sexual minority women

Note. Non-significant tested paths are removed from the figure for parsimony, but are not removed from overall model. All paths shown are significant at $p > .05$.

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CHAPTER 2: STUDY 2

The content within this section, titled “Chapter 2: Study 2” reflects material from a paper that has been published in the journal *International Journal of Eating Disorders* by John Wiley & Sons. The formal citation is as follows:

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Abstract

Objective: Sexual assault, child abuse, and combat have been linked to eating disorders (EDs). However, non-interpersonal trauma is relatively understudied, and therefore it is unknown whether non-interpersonal trauma is associated with EDs. Furthermore, most previous studies do not account for multiple trauma exposures, or the relative association of traumatic events with EDs in the same statistical model. **Method:** Multinomial regression was used to examine the association of lifetime ED diagnosis (anorexia nervosa [AN], bulimia nervosa [BN], binge eating disorder [BED]) with trauma type (sexual interpersonal, other interpersonal, war/combat, and non-interpersonal) in a nationally representative dataset of US adults in bivariate and multivariable (i.e., with all trauma types) models. **Results:** Sexual interpersonal trauma was significantly positively associated with AN and BED in bivariate and multivariable models. In the multivariable model, only BED was found to be equally associated with sexual interpersonal, other interpersonal, and non-interpersonal trauma. **Discussion:** These results indicate a strong positive association between sexual trauma and EDs, even when controlling for experiences of other trauma events. Future research should examine longitudinal mediators between trauma and EDs, especially sexual trauma, to identify what factors may explain this relationship.

Trauma Exposure and Eating Disorders: Results from a United States Nationally Representative Sample

Traumatic events have been linked to eating pathology, such that exposure to traumatic events has been associated with later eating disorder symptoms (Zelkowitz et al., 2021). Furthermore, exposure to traumatic events may have deleterious effects on severity of eating pathology and eating disorder treatment efficacy. Exposure to traumatic events has been associated with more severe disordered eating (Backholm et al., 2013; Scharff et al., 2021; Tagay et al., 2014) and premature termination from eating disorder treatment (Anderson et al., 1997; Carter et al., 2006; Rodríguez et al., 2005). Therefore, investigating the association of specific trauma events and eating pathology to determine potential mechanisms conferring this greater risk are of paramount importance.

While most research examining traumatic events in relation to eating pathology has focused on sexual assault (Madowitz et al., 2015), childhood abuse (e.g., Afifi et al., 2017; Forrest et al., 2021), and combat exposure (Jacobson et al., 2009), this leaves a range of traumatic experiences (e.g., life-threatening illness, car accident, general physical assault) uninvestigated. Previous research (e.g., Chung & Breslau, 2008; Huang et al., 2017; Thomas et al., 2021) has differentiated trauma events into two broad categories: interpersonal and non-interpersonal trauma. Interpersonal trauma can be defined as assault or abuse that are perpetrated by another person (e.g., sexual assault, physical abuse) as compared to non-interpersonal trauma that are adverse events that do not necessarily involve another person (e.g., accidents, disasters, illness; Forbes et al., 2012; Ford et al., 2011). These two broad trauma classifications have been supported by findings that interpersonal traumas lead to more adverse psychological outcomes as compared to non-interpersonal trauma (Charuvastra & Cloitre, 2008; Kessler et al., 2005;

Kilpatrick et al., 2013). The current study will therefore examine both interpersonal and non-interpersonal traumas in their association with eating pathology.

Few previous studies examined the association between non-interpersonal trauma and eating pathology with contradictory results; some have found that only sexual assault was associated with eating pathology in multivariable models (e.g., Gomez et al., 2021) and some have found that other non-interpersonal events were associated with eating pathology in bivariate models (e.g., Lie et al., 2021; Meyer & Stanick, 2018). In addition, studies only rarely examine whether other interpersonal traumas (e.g., physical assault, stalking) were associated with eating pathology. Again, the results are largely mixed such that some find a positive association with disordered eating (e.g., Hazzard et al., 2019) and some do not (e.g., Arditte Hall et al., 2018; Lie et al., 2021; Quilliot et al., 2019). Therefore, the literature examining associations of non-sexual interpersonal and non-interpersonal trauma has found mixed results depending on methods.

In addition, most previous studies rely on the bivariate association between specific traumatic events and eating disorders. While this is informative, a history of multiple trauma events are common (Kilpatrick et al., 2013) and these studies do not examine whether specific trauma experiences are more likely to be associated with eating disorders. For example, while combat exposure has been found to be associated with eating pathology (Arditte Hall et al., 2017, 2018), another study found that combat exposure was not associated with eating pathology when accounting for the association between military sexual trauma and eating pathology (Breland et al., 2018). This finding demonstrates the importance of examining trauma experiences in their association with eating disorders, when including other trauma events in the same model. If specific trauma events or classes of events are more likely to be associated with eating disorders, this highlights the importance of early intervention for such trauma events.

It is theoretically feasible that sexual assault alone would be associated with eating pathology. Previous research in childhood sexual assault has found a robust relationship with eating disorder diagnoses in both men and women (Afifi et al., 2017; Micali et al., 2017). Specific mechanisms have been proposed to explain this association include body dissatisfaction and shame (Madowitz et al., 2015). For example, body dissatisfaction has been found to mediate the association between childhood sexual assault and eating disorder symptoms (Preti et al., 2006). Given that victims of sexual assault frequently develop body dissatisfaction post-trauma (Kremer et al., 2013), individuals may seek to alleviate their negative body image by altering their body with disordered eating. Victims of sexual assault also routinely endorse feelings of shame after assault (Feiring & Taska, 2005; Negrao et al., 2005; Sable et al., 2006) and shame has been implicated in the onset of disordered eating (Blythin et al., 2020). These two proposed mechanisms may not occur solely following sexual-based trauma but may be more likely to occur in sexual trauma as compared to other trauma types. Interpersonal traumas such as physical assault can lead to body dissatisfaction (Kremer et al., 2013), but this effect has been less robustly found in the literature and more well-documented among individuals who experience residual injury after physical assault (Weaver & Resick, 2014). Therefore, sexual assault alone may be associated with eating pathology, but it is also possible that interpersonal traumas broadly may be associated with eating pathology through similar purported mechanisms. Therefore, in the current study, sexual interpersonal trauma was separated from other interpersonal trauma for analyses, both for comparison with previous studies (e.g., Breland et al., 2018; Gomez et al., 2021) and for the aforementioned theoretical reasons.

The primary aims of the current study were to: 1) assess the independent association of sexual interpersonal (both childhood and adult), other interpersonal (e.g., physical assault,

stalking), war/combat (e.g., active military combat, relief worker), and non-interpersonal (e.g., natural disasters, serious injury/illness) traumas and eating disorder diagnoses and 2) assess the association of the aforementioned trauma types with eating disorder diagnoses when including all trauma types in a single model (i.e., accounting for exposure to other trauma types) to determine which trauma types are more likely to be associated with eating disorders. Based on prior research, we hypothesize that all trauma types will be associated with eating disorders in bivariate models, but only sexual interpersonal trauma will be associated with eating pathology in multivariable models.

Method

Design

The current study used a publicly available dataset from the National Epidemiologic Survey of Alcohol and Related Conditions-III (NESARC-III; National Institute on Alcohol Abuse and Alcoholism, 2019), a nationally representative sample of 36,309 US non-institutionalized adults (Grant et al., 2014, 2016). The NESARC-III respondents were selected through multistage probability sampling. Primary, secondary, and tertiary sampling units were counties or groups of contiguous counties, groups of Census-defined blocks, and households, respectively. Black, Asian, and Hispanic household members were oversampled such that, within each household, two respondents were selected. The household response rate was 72%, and the person-level response rate was 84%, for an overall response rate of 60.1% (Grant et al., 2016). Data were adjusted for nonresponse and weighted to represent the civilian US population based on the 2012 American Community Survey.

Measures

Sociodemographic Characteristics

Respondents provided sociodemographic information, including age, race/ethnicity, sex assigned at birth, census region, country of birth, sexual orientation, household income, and highest education level achieved. See table 1 for sociodemographic characteristics by lifetime eating disorder diagnosis.

Eating Disorder Diagnoses

Eating disorder diagnoses were given based on interviews conducted using the NIAAA Alcohol Use Disorder and Associated Disabilities Interview Schedule-5 (AUDADIS-5; Grant et al., 2011). Interviewers were 970 lay assessors with an average of 5 years of field experience with health-related and other surveys. Given that prior researchers have found errors in the coding of eating disorder diagnoses (Udo & Grilo, 2018), diagnoses were recoded based on the procedures established by Udo and Grilo (2018) for meeting DSM-5 criteria for anorexia nervosa (AN), bulimia nervosa (BN), and binge eating disorder (BED). Since this study examined lifetime diagnoses of an eating disorder, categorization for an eating disorder was based on previous studies (Udo & Grilo, 2018) and the well-established diagnostic “hierarchy” of AN>BN>BED (i.e., lifetime BN excludes those with lifetime AN, lifetime BED excludes those with lifetime AN/BN). Other Specified Feeding and Eating Disorder (OSFED) diagnoses were not examined because the relevant items for assessing these diagnoses were not administered per NESARC protocol (e.g., if participants were never low weight, they were not administered the other items for assessing anorexia nervosa symptoms).

Trauma Exposure

As part of the interview protocol, all participants were asked whether they had experienced 20 specific and potentially traumatizing events. In an effort to facilitate comparison of different types of traumatic events, the events were grouped into four theoretically derived

categories (1. sexual interpersonal; 2. other interpersonal; 3. non-interpersonal; and 4. war/combat).

Analysis

Analyses were performed using IBM SPSS Version 28 and accounted for the sample design of the NESARC-III by using the complex sampling with the procedures with the Taylor series Linearization variance estimation method. Weighted frequencies and cross-tabulations were conducted for eating disorder diagnoses and trauma exposure. For all regression analyses, the reference group for each trauma exposure type was no exposure to that trauma type; therefore, trauma groupings are not mutually exclusive. Binary logistic regression was used to calculate if the odds of experiencing each type of trauma was higher in those diagnosed with any eating disorder, with no eating disorder diagnosis as the reference group. Multinomial logistic regression was used to calculate odds ratios, adjusting for the NESARC complex sampling design, to compare the risk of lifetime diagnosis for each type of eating disorder as a function of trauma type. The outcome was lifetime diagnosis of each type of eating disorder; no eating disorder diagnosis was the reference group. For bivariate multinomial model results, each trauma exposure type was entered as an independent variable in four different multinomial logistic regression models. For multivariable multinomial model results, all trauma exposure types were entered as independent variables into the one multinomial logistic regression model. If more than one trauma type was significant in multivariable model, the strength of the association between the trauma type and eating disorder outcome was compared. Point estimates for the difference between parameters were calculated and then the delta method (Greene, 2012, pp. 1083–1084) was used to calculate the standard error of this difference. Z-score was calculated by dividing the point estimate by its standard error. Bonferroni corrections for multiple testing were applied by

dividing 0.05 by the overall number (92) of comparisons. The level of significance was set at $p < .00154$. Effect sizes are discussed using Funder and Ozer (2019)'s metrics of effect size for psychological research which are converted to odds ratios as follows: OR = 1.20 as very small, OR = 1.44 as small, OR = 2.10 as medium, OR = 3.13 as large, and OR = 4.87 as very large.

Potential demographic covariates were reviewed for inclusion based on guidance from prior research, such that confounding variables (i.e., variables that may connote a common cause for both the independent variable and outcome) were controlled for, but collider variables (i.e., variables that are caused by both the independent variable and outcome) and mediator variables were not (see Rohrer, 2018 for further explanation). Sex assigned at birth was included as a covariate, given that men report greater trauma exposure as compared to women (Frans et al., 2005) and women are more often diagnosed with eating disorders than men (Hudson et al., 2007; Udo & Grilo, 2018). Sexual orientation was also included as a covariate as sexual minority individuals are more likely to experience trauma (Roberts et al., 2010) and eating pathology (Calzo et al., 2017; Kamody et al., 2020) as compared to their heterosexual counterparts. Age was included as a covariate because older individuals are more likely to be diagnosed with BED (Mangweth-Matzek & Hoek, 2017) and are more likely to accumulate traumatic experiences as they age (Ogle et al., 2013). Sex, sexual orientation, and age are included as covariates in bivariate and multivariate models.

Results

Prevalence of Trauma Events by Eating Disorder Diagnosis

In the current sample, 43.8% of individuals diagnosed with an eating disorder and 24.9% of individuals without an eating disorder diagnosis reported more than one traumatic event. In terms of trauma exposure types, 35.8% of individuals diagnosed with an eating disorder and

16.7% of individuals without an eating disorder diagnosis reported trauma of more than one type. The most common type of trauma was non-interpersonal. Approximately 71% of individuals diagnosed with AN, 67% of individuals diagnosed with BN, and 75% of individuals diagnosed with BED had experienced a non-interpersonal trauma. The least common trauma type was war-related. Approximately 2% of individuals diagnosed with AN, 2.2% of individuals diagnosed with BN, and 1.4% of individuals diagnosed with BED had experienced war-related trauma. See table 2 for full prevalence of traumatic events by eating disorder diagnosis.

Association of Trauma with Eating Disorder Diagnoses

Bivariate Results

When examining the bivariate association between trauma classes and eating disorder diagnoses, war-related trauma was not significantly associated with lifetime diagnosis of AN (OR = 1.00, $p = .999$), BN (OR = 0.98, $p = .977$), or BED (OR = 0.48, $p = .378$). Sexual interpersonal trauma was significantly associated with higher odds of lifetime diagnosis of AN (OR = 3.23, $p < .001$) and BED (OR = 3.04, $p < .001$), but not BN (OR = 1.97, $p = .032$). Other non-sexual interpersonal trauma was significantly associated with higher odds of lifetime diagnosis of AN (OR = 2.11, $p < .001$) and BED (OR = 2.66, $p < .001$), but not BN (OR = 2.20, $p = .008$). Non-interpersonal trauma was associated with higher odds of lifetime diagnosis of AN (OR = 1.78, $p = .001$) and BED (OR = 2.05, $p < .001$), but not BN (OR = 1.23, $p = .450$). See table 3 for full results.

Multivariable Results

When including all trauma classes in a single model, sexual interpersonal (OR = 2.64, $p < .001$) was significantly associated with higher odds of AN lifetime diagnosis, but war-related (OR = 0.80, $p = .618$), non-sexual interpersonal (OR = 1.45, $p = .039$), and non-interpersonal

(OR = 1.49, $p = .018$) traumas were not. Lifetime BN diagnosis was not associated with any trauma class, including war-related (OR = 0.86, $p = .831$), sexual interpersonal (OR = 1.55, $p = .230$), non-sexual interpersonal trauma (OR = 1.95, $p = .062$), and non-interpersonal trauma (OR = 1.04, $p = .903$). Finally, war-related trauma was not associated with lifetime diagnosis of BED (OR = 0.38, $p = .246$), but sexual interpersonal (OR = 2.22, $p < .001$), non-sexual interpersonal (OR = 1.96, $p < .001$), and non-interpersonal trauma were (OR = 1.72, $p < .001$). For BED, the difference in odds ratios was compared, given that more than one trauma type was significant. The difference in odds ratios between sexual interpersonal trauma and non-sexual interpersonal trauma ($z = 1.53$, $p = .125$), sexual interpersonal trauma and non-interpersonal trauma ($z = 1.22$, $p = .223$), and non-sexual interpersonal trauma and non-interpersonal trauma ($z = 0.23$, $p = .821$) was not significant. See table 3 for full results.

Discussion

The primary aims of the current study were to: 1) assess the bivariate association of four classes of trauma exposure and eating disorder diagnoses and 2) assess the association of these trauma classes with eating disorder diagnoses to account for multiple trauma type exposures. Results indicated that war-related trauma was not associated with any eating disorder diagnosis. Non-interpersonal trauma was associated with diagnosis of AN and BED in bivariate models with a small effect size, but only BED with a small effect size when controlling for other trauma types. Non-sexual interpersonal trauma was associated with diagnosis of AN and BED in bivariate models with a medium effect size, but only BED with a small effect size when controlling for other trauma types. Finally, sexual interpersonal trauma was associated with AN and BED with a large and medium effect size respectively in bivariate models, but not BN. In

multivariate models AN and BED were associated with sexual interpersonal trauma with a medium effect size, but BN was not.

In line with most research (Breland et al., 2018; Gomez et al., 2021; Madowitz et al., 2015), sexual trauma was found have a medium association with eating pathology (controlling for sex, sexual identity, and age), both in bivariate models and within the context of other trauma exposure for AN and BED, though not with BN. Given that prior meta-analyses have found that the association between childhood sexual assault is less robust for AN as compared to BN and BED (Solmi et al., 2021), this finding provides further support that sexual assault is associated with AN diagnosis, but also contradicts previous findings that binge and/or purge-spectrum behaviors may be more likely associated with eating pathology (Caslini et al., 2016; Molendijk et al., 2017). However, the current study had a relatively small sample size of individuals with BN. Further, given our hierarchical classification of eating disorders, the AN category may have included individuals that were also diagnosed with BN or BED at some point during their life. Therefore, the current null findings should be considered tentative. Regarding sexual trauma, Madowitz et al. (2015) hypothesized two potential etiological pathways that may explain the association between sexual assault and eating disorders: 1) body perceptions, including potential mediators such as body dissatisfaction (Prete et al., 2006) and sexual dysfunction (Castellini et al., 2013); and 2) management of psychological difficulties, including potential mediators such as emotion regulation (Burns et al., 2012) and impulsivity (Dworkin et al., 2014). Another potential mediator that might explain this relationship is PTSD as sexual trauma survivors are at higher conditional risk of developing PTSD as compared to other trauma survivors (Hetzl-Riggin & Roby, 2013) and previous studies have found that posttraumatic stress is a significant mediator in the relationship between sexual trauma and eating disorder symptoms cross-

sectionally (Holzer et al., 2008). Future research should examine the association between sexual trauma and eating disorders longitudinally to test the theorized mediation paths.

War-related trauma was not associated with any eating disorder diagnosis, both in bivariate models and accounting for other trauma types. However, caution is warranted in interpreting the current findings due to potential concerns about statistical power given the low unweighted sample size of individuals who both had an eating disorder and war-related trauma. It may be that individuals with eating disorders are less likely to experience war-related trauma, or the NESARC-III dataset may not fully capture the war-related experiences of the general population. Prior research examining eating pathology in veteran populations found that combat trauma was significant when accounting for experiences of sexual assault in men (Arditte Hall et al., 2017) but not in women (Breland et al., 2018). Therefore, future research may seek to replicate the findings of Arditte Hall et al. (2017) and Breland et al. (2018) in a sample with greater exposure to war-related trauma as well as examining perhaps a moderating role of gender.

In the current study, non-interpersonal trauma was found to be associated with diagnosis of AN and BED with a small effect size, controlling for covariates, in bivariate models but was only associated with BED with a small effect size when accounting for other trauma exposures. This contradicts prior work that finds no significant association between non-interpersonal trauma and eating pathology (Gomez et al., 2021). Of note, the Gomez et al. (2021) study examined the association of non-interpersonal trauma (i.e., wreck, crash, accident; serious body-related accident; and life-threatening illness or injury) with an overall measure of eating pathology (Eating Disorder Diagnostic Scale; Stice et al., 2004), accounting for combat exposure and sexual trauma. Given the use of an overall eating pathology measure, diagnosis-specific

differences such as those observed in the current study may have been obscured. Indeed, a recent study by Lie et al. (2021) found a significant association between experiences of a life-threatening illness or disease and lifetime diagnosis of AN binge/purge subtype as well as mixed eating disorder history (i.e., a history of AN and BN and/or BED diagnosis). However, Lie et al. (2021) did not account for the experience of multiple trauma types; therefore, it is unknown whether this association would have remained significant after accounting for other trauma types such as sexual assault.

Interestingly, sexual interpersonal, non-sexual interpersonal, and non-interpersonal trauma were associated with BED diagnosis in bivariate models and multivariable models, and with relatively equal association strength (small to medium). This finding is similar to previous studies that have found that any potentially traumatic event is associated with BED (Brewerton, 2007; Brewerton et al., 2014). This may indicate that the etiology of trauma in BED functions differently than AN and BN. A potential mechanism of interest in conferring this association may be overvaluation of shape and weight, which is required for AN or BN diagnosis but not BED (American Psychiatric Association, 2013). However, prior research has suggested that overvaluation of shape and weight is similar between BED and BN patients (Ahrberg et al., 2011). Future research should examine the association between trauma and BED longitudinally to identify potential mechanisms of interest to explain the observed differences by diagnosis.

There are some important limitations of the current study to note. Given that this study was cross-sectional, no casual conclusions about the association between various trauma types and eating disorder onset can be made. Future research is needed to investigate the prospective association between trauma events and eating disorders, particularly to identify important mediators between trauma and eating pathology. In addition, results regarding war-related

trauma should be considered tentative due to the potential low power afforded by the small, unweighted sample size of individuals with both an eating disorder and war-related trauma. Future research should consider replicating the current study with a larger sample size at the cross-section of eating disorders and war-related trauma exposure. Another limitation of note is the potential for underreporting of trauma events. Due to the sensitive nature of trauma, individuals in the current study may not have felt comfortable disclosing said trauma (Andresen & Blais, 2019). Alternatively, research has found that trauma memories are subject to significant recall bias (Hirst et al., 2015) which may also lead to underreporting. Therefore, trauma in the current study may be a lower estimate of actual exposure. A further potential limitation is that the current study was not able to separate exposure to trauma as a child as compared to adult exposure. A robust literature has found a strong relationship between childhood trauma and eating pathology (Caslini et al., 2016; Molendijk et al., 2017; Pignatelli et al., 2017). Prior research has found differences in eating pathology based on timing of trauma exposure (Vidaña et al., 2020) as well as the effect of cumulative trauma over time (Molendijk et al., 2017); therefore, future research should consider disentangling these associations by eating disorder diagnosis. Finally, the sample size for AN and BED were much larger than that of BN. This may have led our analyses to be underpowered to detect effects in the BN group, as compared to the AN and BED groups; therefore, the null results in BN should be considered with caution. Despite these limitations, we consider the current study to be an important addition to the existing literature.

The current study demonstrates a meaningful extension of prior work in multiple ways. First, this study examines similar questions of interest to Breland et al. (2018), Gomez et al. (2021), and Lie et al. (2021) but employs a nationally representative sample of US adults, as

compared to samples of predominantly White female veterans, food insecure individuals, and Swedish nationals respectively. Second, the current study builds upon Lie et al. (2021) by including comparisons of trauma exposure in a single model, which is important for determining whether specific types of trauma are more likely to be associated with eating disorders.

The present study has implications for clinical work in the field of eating disorders. Individuals with eating disorders are more likely to have experienced traumatic events than the general population. Therefore, clinicians that work with eating disorders should consider incorporating trauma assessment into their intake evaluations to ascertain an accurate clinical picture and determine whether trauma-informed care is needed. Given that trauma has been found to predict premature treatment termination (Anderson et al., 1997; Carter et al., 2006; Rodríguez et al., 2005), clinicians should be aware of this possibility and work to adapt treatment to these patients' needs. Conversely, given the higher rate of trauma exposure in eating disorder populations, these individuals may also present to trauma clinics. Clinicians that work in the field of PTSD and trauma should consider assessing for eating pathology.

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Table 2.1: Lifetime prevalence of DSM-5 eating disorder diagnosis by sociodemographic characteristics

Sociodemographic Characteristic	AN		BN		BED		Any ED		No ED Diagnosis	
	n	% (SE)	n	% (SE)	n	% (SE)	n	% (SE)	n	% (SE)
Age, M (SE)	41.83	(0.96)	39.05	(2.45)	45.26	(1.21)	42.82	(0.83)	46.60	(0.19)
Total	276	0.8 (0.1)	77	0.2 (0.1)	247	0.7 (0.1)	600	1.7 (0.1)	35,709	98.3 (0.1)
Sex										
Male	23	7.5 (2.2)	11	15.3 (4.9)	62	28.1 (3.6)	96	16.6 (1.9)	15766	48.6 (0.3)
Female	253	92.5 (2.2)	66	84.7 (4.9)	185	71.9 (3.6)	504	83.4 (1.9)	19943	51.4 (0.3)
Race or ethnicity										
Non-Hispanic White	206	79.2 (2.8)	42	68.4 (7.0)	160	72.9 (3.5)	408	75.3 (2)	18,786	66 (0.8)
Non-Hispanic Black	17	2.8 (0.7)	12	10.3 (3.7)	33	9.1 (2.2)	62	6.2 (1.0)	7,704	11.9 (0.7)
Non-Hispanic American Indian/Alaska Native	8	5.5 (2.3)	2	2.1 (1.5)	2	0.9 (0.7)	12	3.3 (1.2)	499	1.5 (0.1)
Non-Hispanic Asian/Native Hawaiian/Other Pacific Islander	9	4.1 (1.5)	2	2.3 (1.7)	9	3.9 (1.8)	20	3.8 (1.1)	1,781	5.8 (0.5)
Hispanic	36	8.5 (1.5)	19	16.8 (5.3)	43	13.2 (2.9)	98	11.4 (1.5)	6,939	14.8 (0.7)
Born in the United States										
Yes	247	91.1 (1.9)	71	94.3 (2.8)	220	89.4 (2.1)	538	90.9 (1.2)	29,358	83.9 (0.5)
No ^a	29	8.9 (1.9)	6	5.7 (2.8)	27	10.6 (2.1)	62	9.1 (1.2)	6,342	16.1 (0.5)
Unknown	0	0	0	0	0	0	0	0	9	0 (0.0)
Census Region										
Northeast	39	17.1 (3.3)	11	18.4 (7.0)	22	14.3 (2.7)	72	16.2 (2.2)	5,108	18.3 (0.5)
Midwest	57	22.4 (3.4)	15	18.3 (4.9)	63	24.9 (3.3)	135	22.9 (2.3)	7,431	21.5 (0.4)
South	88	30.5 (4.1)	20	26.7 (5.4)	84	32.1 (3.5)	192	30.6 (2.4)	14,340	37.2 (0.9)
West	92	30 (4.1)	31	36.6 (6.0)	78	28.7 (3.5)	201	30.4 (2.5)	8,830	23.1 (0.9)

Tables

Table 2.2: Lifetime prevalence of DSM-5 eating disorder diagnosis by sociodemographic characteristics, continued

Sociodemographic Characteristic	AN		BN		BED		Any ED		No ED Diagnosis	
	n	% (SE)	n	% (SE)	n	% (SE)	n	% (SE)	N	% (SE)
Sexual Identity										
Heterosexual or straight	256	92.8 (2.0)	66	87.3 (4.2)	231	94.3 (1.6)	553	92.7 (1.3)	34,091	96 (0.2)
Gay or lesbian	5	1.5 (0.8)	2	3.1 (2.5)	7	3.1 (1.3)	14	2.3 (0.7)	572	1.4 (0.1)
Bisexual	10	4 (1.4)	5	5.9 (2.7)	5	1.4 (0.7)	20	3.2 (0.9)	546	1.3 (0.1)
Not sure	4	1.6 (1.1)	3	3 (2.1)	4	1.2 (0.7)	11	1.6 (0.7)	188	0.5 (0.0)
Unknown	1	0.2 (0.2)	1	0.7 (0.7)	0	0	2	0.2 (0.1)	312	0.8 (0.1)
Education Level										
Less than high school	22	7.7 (1.5)	13	11.7 (3.9)	32	12.3 (2.3)	67	10 (1.2)	5,423	13.1 (0.4)
High school or GED	48	15.4 (2.4)	16	20.5 (6.8)	53	23.2 (3.4)	117	19.1 (1.9)	9,682	25.9 (0.5)
Some college or higher	206	76.9 (2.9)	48	67.8 (7.2)	162	64.5 (3.9)	416	70.9 (2.1)	20,604	61 (0.8)
Household Income Level										
<\$25,000	75	19.5 (2.5)	26	26.3 (5.8)	92	28.6 (2.8)	193	23.9 (1.8)	12,627	27.4 (0.6)
\$25,000–39,999	47	14.1 (2.5)	21	21.6 (4.8)	44	17.4 (2.5)	112	16.3 (1.8)	6,736	17.1 (0.3)
\$40,000–69,999	59	25.3 (3.0)	12	13.3 (4.2)	57	21.5 (2.9)	128	22.3 (2.1)	7,742	22.8 (0.3)
≥\$70,000	95	41.1 (3.8)	18	38.8 (8.0)	54	32.6 (4.2)	167	37.5 (2.8)	8,604	32.7 (0.6)

Note. All analyses adjusted for complex survey design of the National Epidemiologic Survey of Alcohol and Related Conditions-III study. SE, standard error; M, mean; AN, anorexia nervosa; BN, bulimia nervosa; BED, binge eating disorder; ED, eating disorder. ^aIn the eating disorder group, the most common other country of birth was Mexico ($n = 21$). Forty-one participants identified other countries of birth; no one birth country was represented by more than 5 participants.

Table 2.3: Lifetime trauma exposure prevalence by eating disorder diagnosis

Traumatic Event	AN (n = 276)		BN (n = 77)		BED (n = 247)		Any ED (n = 600)		No ED Diagnosis (n = 35,709)		ED vs. No ED Diagnosis ^a	
	n	% (SE)	n	% (SE)	n	% (SE)	n	% (SE)	n	% (SE)	AOR	95% CI
Any war-related trauma	8	2.0 (0.8)	3	2.2 (1.5)	2	1.4 (1.2)	13	1.8 (0.6)	1,272	3.9 (0.2)	0.45	0.22; 0.94
Active military combat	3	1.0 (0.6)	0	0	1	1.1 (1.1)	4	0.9 (0.5)	856	2.7 (0.1)	0.33	0.10; 1.07
Peacekeeper/relief worker	1	0.1 (0.1)	0	0	0	0	1	0.0 (0.0)	158	0.4 (0.0)	0.09	0.01; 0.72
Civilian in war zone/place of terror	4	1.1 (0.6)	3	2.2 (1.5)	0	0	7	0.8 (0.3)	194	0.6 (0.0)	1.38	0.61; 3.15
Refugee	2	0.5 (0.3)	1	0.4 (0.4)	0	0	3	0.3 (0.2)	112	0.4 (0.0)	0.77	0.22; 2.65
Prisoner of war	0	0	0	0	1	0.3 (0.3)	1	0.1 (0.1)	54	0.2 (0.0)	0.68	0.90; 5.04
Any sexual interpersonal trauma	85	34.0 (3.0)	25	24.3 (5.8)	64	27.2 (3.4)	174	30.1 (2.0)	3,378	9.1 (0.2)	4.32	3.58; 5.21*
Sexually abused before age 18	68	26.7 (2.8)	19	17.7 (4.6)	52	22.6 (3.2)	139	23.9 (1.8)	2,817	7.7 (0.2)	3.80	3.11; 4.64*
Sexually assaulted as an adult	30	11.2 (2.3)	10	9.2 (3.4)	18	6.4 (1.3)	58	9.1 (1.2)	864	2.1 (0.1)	4.59	3.40; 6.19*
Any other non-sexual interpersonal trauma	99	34.2 (3.5)	29	35.6 (6.8)	94	37.8 (3.4)	222	35.8 (2.4)	6,627	17.7 (0.3)	2.60	2.12; 3.20*
Physically abused before age 18	22	6.5 (1.4)	9	15.2 (6.4)	29	13.9 (3.0)	60	10.5 (1.6)	1,258	3.6 (0.1)	3.17	2.27; 4.43*
Beaten up by spouse/romantic partner	50	18.2 (2.8)	14	14.3 (4.1)	34	10.7 (1.8)	98	14.8 (1.6)	2,337	5.6 (0.2)	2.92	2.23; 3.82*
Beaten up by someone else	19	6.1 (1.8)	7	14.0 (6.5)	13	6.4 (1.9)	39	7.2 (1.5)	1,523	4.2 (0.1)	1.76	1.14; 2.70
Kidnapped/held hostage	5	2.3 (1.3)	3	2.4 (1.5)	6	2.1 (0.8)	14	2.2 (0.7)	252	0.6 (0.0)	3.78	1.96; 7.31*
Stalked	20	6.6 (1.6)	4	3.8 (2.1)	17	6.5 (1.8)	41	6.2 (1.1)	983	2.4 (0.1)	2.67	1.81; 3.94*
Mugged, held up, threatened with a weapon or assaulted in any other way	21	6.7 (2.0)	3	2.3 (1.5)	21	9.1 (1.8)	45	7.1 (1.3)	2,007	5.4 (0.2)	1.34	0.90; 1.98

Table 2.2: Lifetime trauma exposure prevalence by eating disorder diagnosis, continued

Traumatic Event	AN (n = 276)		BN (n = 77)		BED (n = 247)		Any ED (n = 600)		No ED Diagnosis (n = 35,709)		ED vs. No ED Diagnosis ^a	
	n	% (SE)	n	% (SE)	n	% (SE)	n	% (SE)	n	% (SE)	n	% (SE)
Any non-interpersonal trauma	195	71.2 (3.1)	55	66.9 (7.1)	200	82.8 (2.8)	450	75.2 (2.1)	21,097	62.2 (0.7)	1.85	1.47; 2.31*
Serious or life-threatening injury	33	10.8 (1.8)	7	7.4 (3.4)	40	16.2 (2.9)	80	12.5 (1.5)	3,806	11.2 (0.3)	1.13	0.86; 1.48
Serious or life-threatening illness	44	16.1 (2.7)	13	19.4 (5.4)	44	17.2 (2.7)	101	16.9 (1.7)	4,260	13.1 (0.3)	1.35	1.05; 1.74
Saw a dead body or body parts	56	24.4 (3.4)	11	11.7 (4.0)	71	28.8 (3.4)	138	24.5 (2.1)	6,253	18.5 (0.4)	1.42	1.13; 1.79
Injured in a terrorist attack	0	0	0	0	2	1.8 (1.3)	2	0.7 (0.5)	158	0.4 (0.0)	1.57	0.36; 6.87
Natural disaster, like flood, fire, earthquake, hurricane	35	13.1 (2.9)	8	9.1 (3.6)	20	7.9 (1.9)	63	10.5 (1.6)	3,170	9.6 (0.5)	1.11	0.79; 1.56
Juvenile detention or jail	9	3.3 (1.1)	5	4.1 (2.1)	15	6.4 (1.9)	29	4.6 (1.1)	1,196	3.1 (0.1)	1.50	0.92; 2.42
Number of trauma events	86	28.8 (2.5)	26	35.2 (6.0)	63	26.2 (3.3)	175	28.6 (1.7)	18,449	50.5 (0.6)		
0	74	28.1 (3.6)	16	25.4 (6.9)	71	27.6 (3.7)	161	27.6 (2.4)	8,572	24.5 (0.3)		
1	44	17.4 (2.7)	15	21.5 (7.3)	54	21.4 (2.9)	113	19.5 (1.9)	4,338	12.8 (0.3)		
2	28	11.3 (2.6)	9	6.8 (2.7)	31	12.2 (2.2)	68	11.1 (1.6)	2,390	6.7 (0.2)		
3	44	14.4 (2.4)	11	11.1 (3.3)	28	12.6 (2.7)	83	13.1 (1.6)	1,960	5.4 (0.2)		
4+ ^b	86	28.8 (2.5)	26	35.2 (6.0)	63	26.2 (3.3)	175	28.6 (1.7)	18,449	50.5 (0.6)		
Number of trauma event types	86	28.8 (2.5)	26	35.2 (6.0)	63	26.2 (3.3)	175	28.6 (1.7)	18,449	50.5 (0.6)		
0	93	35.2 (3.7)	22	36.4 (8.1)	92	35.8 (3.9)	207	35.6 (2.6)	11,224	32.7 (0.5)		
1	66	25.3 (3.6)	22	20.9 (5.5)	75	29.2 (3.1)	163	26.3 (2.3)	4,861	13.6 (0.3)		
2	30	10.4 (2.3)	7	7.6 (2.9)	17	8.7 (2.5)	54	9.4 (1.4)	1,573	4.2 (0.1)		
3	1	0.3 (0.3)	0	0	0	0	1	0.1 (0.1)	37	0.1 (0.0)		

Note. All analyses adjusted for complex survey design of the National Epidemiologic Survey of Alcohol and Related Conditions-III (NESARC) study. AN, anorexia nervosa; BN, bulimia nervosa; BED, binge eating disorder; ED, eating disorder; SE, standard error; AOR, adjusted odds ratio.

^a Odd ratios are the odds of experiencing the specific trauma event for individuals with an eating disorder as compared to individuals without an eating disorder. Reference group for the specific trauma event is no exposure to that specific trauma event.

^b In the NESARC study design, only four potentially traumatic events are inquired about. Therefore, individuals that may have had four or more trauma events are classified into a single group.

* $p < .001$; Significant finding following Bonferroni correction.

Table 2.4: Lifetime exposure to potentially traumatizing event by eating disorder

Traumatic event	Bivariate Adjusted Odds Ratios (95% CI)			Multivariable Adjusted Odds Ratios (95% CI)		
	AN	BN	BED	AN	BN	BED
Any war-related trauma	1.00 (0.42, 2.37)	0.98 (0.24, 3.95)	0.48 (0.09, 2.52)	0.80 (0.34, 1.92)	0.86 (0.21, 3.46)	0.38 (0.07, 1.98)
Any sexual interpersonal trauma	3.23 (2.44, 4.27)*	1.97 (1.06, 3.66)	3.04 (2.13, 4.34)*	2.67 (1.95, 3.64)*	1.55 (0.75, 3.21)	2.22 (1.54, 3.22)* ^{ab}
Any other non-sexual interpersonal trauma	2.11 (1.55, 2.85)*	2.20 (1.23, 3.94)	2.67 (2.00, 3.55)*	1.45 (1.02, 2.06)	1.95 (0.97, 3.94)	1.96 (1.46, 2.62)* ^{ac}
Any non-interpersonal trauma	1.78 (1.31, 2.42)*	1.23 (0.72, 2.12)	2.05 (1.56, 2.70)*	1.49 (1.07, 2.07)	1.04 (0.56, 1.91)	1.72 (1.30, 2.26)* ^{bc}

Note. All analyses control for age, sexual identity, and sex assigned at birth, as well as the complex survey design of the National Epidemiologic Survey of Alcohol and Related Conditions-III study. Effect sizes are interpreting using Funder and Ozer (2019)'s metrics of effect size for psychological research which are converted to odds ratios as follows: OR = 1.20 as very small, OR = 1.44 as small, OR = 2.10 as medium, OR = 3.13 as large, and OR = 4.87 as very large. CI, confidence interval; AN, anorexia nervosa; BN, bulimia nervosa; BED, binge eating disorder.

^aFor individuals with binge eating disorder, there is no significant difference between the odds of experiencing sexual interpersonal trauma and the odds of experiencing non-sexual interpersonal trauma ($z = -0.50, p = .618$).

^bFor individuals with binge eating disorder, there is no significant difference between the odds of experiencing sexual interpersonal trauma and the odds of experiencing non-interpersonal trauma ($z = -1.05, p = .296$).

^cFor individuals with binge eating disorder, there is no significant difference between the odds of experiencing non-sexual interpersonal trauma and the odds of experiencing non-interpersonal trauma ($z = 0.63, p = .531$).

* $p < .001$; Significant finding following Bonferroni correction.

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CHAPTER 3: STUDY 3

The content within this section, titled “Chapter 3: Study 3” reflects material from a paper that is under review in the Journal of Affective Disorders by Elsevier. The formal citation is as follows:

Convertino, A. D., Mendoza, R. R., & Blashill, A. J. (2024). *Eating disorder and posttraumatic stress symptomology: An intensive time-series psychological network analysis*
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Abstract

Background: It is estimated that about 8% of the US population meets criteria for posttraumatic stress disorder (PTSD) in their lifetime; however, individuals with eating disorders (EDs) are more likely to be diagnosed with PTSD than those without EDs. Patients with comorbid ED and PTSD are more likely to drop out of and relapse after ED treatment. It has been theorized that ED and PTSD symptoms demonstrate a bidirectional relationship, such that symptoms of one disorder maintain the other. Thus, the current study seeks to examine the association between ED and PTSD symptoms longitudinally. **Methods:** The current study examined 50 undergraduate students with elevated PTSD and ED symptoms in a time-series network analysis with an ecological momentary assessment design. Participants completed surveys six times a day for fourteen days. **Results:** In the temporal network, loss of control eating, concentration impairment related to shape and weight, and negative alterations in cognition and mood were the strongest temporal links. In the contemporaneous network, fear of weight gain demonstrated the strongest association with ED and PTSD symptoms at the same timepoint. Body dissatisfaction and arousal were the symptoms with the strongest associations across diagnostic clusters. **Limitations:** The current study cannot speak to causality and participants were primarily white women, limiting generalizability to other groups. **Conclusions:** Interventions designed to target both ED and PTSD symptoms may wish to consider targeting these symptoms as they demonstrated the strongest associations in the network.

Eating Disorder and Posttraumatic Stress Symptomology: An Intensive Time-Series Psychological Network Analysis

While many people experience traumatic events in their lifetime (approximately 90%; Kilpatrick et al., 2013), only about 8% of the US populace is estimated to meet criteria for posttraumatic stress disorder (PTSD; Kilpatrick et al., 2013). When examining individuals with an eating disorder (ED) however, prevalence estimates of PTSD are estimated to be 18.35% when weighted by sample size and 24.59% when weighted by study quality (Ferrell et al., 2022). In addition, individuals diagnosed with both an ED and PTSD display more severe eating pathology than those diagnosed with an ED alone (e.g., Scharff et al., 2021). When considering treatment outcomes, a recent systematic review concluded that there was tentative evidence that PTSD was associated with higher dropout rates from ED treatment and – though individuals with PTSD achieved equivalent ED symptom reduction as compared to their undiagnosed peers at the end of treatment – patients with PTSD experienced greater ED symptom relapse rates after treatment (Convertino & Mendoza, 2023). Therefore, individuals with PTSD and an ED are arguably in great need of innovations aimed towards treatment retention and relapse prevention.

Researchers have theorized that there is a bidirectional, functional relationship between ED and PTSD symptoms (Mitchell et al., 2021; Trottier et al., 2016). Specifically, ED behaviors such as restriction, binge eating, and purging are thought to serve a self-medication or self-soothing function for PTSD symptoms (Mitchell et al., 2021; Trottier et al., 2016). This assertion has been supported by multiple cross-sectional studies finding that emotion regulation mediated the relationship between PTSD or trauma event exposure and ED symptoms (e.g., Mitchell & Wolf, 2016; Racine & Wildes, 2015). Additionally, negative alterations in cognition as a symptom of PTSD are theorized to lead increased negative beliefs about the self and body,

particularly regarding the valuation of shape and weight, perfectionism, and low self-esteem (Mitchell et al., 2021; Trottier et al., 2016). Therefore, existing theory suggests longitudinal associations between PTSD and ED symptoms that have yet to be examined.

Network analysis is a feasible, innovative method of evaluating the association between PTSD and ED symptoms. The network perspective of psychopathology posits that mental disorders are best conceptualized as a system of interacting symptoms (Borsboom & Cramer, 2013). In psychological network analyses, symptoms (i.e., nodes) are examined for their unique correlation (i.e., edges) with other symptoms in the same model. Network analysis also identifies “central symptoms” or symptoms that have the strongest relationships with all other symptoms. Identifying these central symptoms is important because these are the symptoms that, should they be disrupted through treatment, theoretically lead to the most improvement in mental health (Borsboom & Cramer, 2013), though this claim has not been conclusively proven (Bringmann et al., 2019, 2022).

Furthermore, the network perspective is particularly well-suited to address comorbidity between psychopathology because, rather than assuming that mental health conditions are different clusters of symptoms separately occurring simultaneously, the network perspective hypothesizes that symptoms of both disorders interact (Fried et al., 2017). Symptoms in mental illnesses that lead to activation of symptoms in another mental illness are conceptualized as “bridge symptoms.” Targeting these bridge symptoms in treatment is theorized to be the most effective for disrupting maladaptive relationships between symptoms, effectively addressing comorbid disorders simultaneously (Fried et al., 2017). Therefore, identifying these bridge symptoms between PTSD and EDs is an important first step in developing a new intervention for the comorbidity between these disorders.

Previous network analyses examining both PTSD and ED symptoms in the same network have been conducted with cross-sectional data, highlighting insights about central and bridge symptoms in this comorbidity. Central ED symptoms that have been identified are cognitive restraint (Liebman et al., 2021), binge eating, fear of weight gain, and desire for a flat stomach (Vanzhula et al., 2019). PTSD symptoms of re-experiencing/intrusion (Liebman et al., 2021; i.e., disturbing dreams, being upset at reminders of trauma, Vanzhula et al., 2019) and negative beliefs/negative emotions were also identified as central (Nelson et al., 2022). Binge eating was a common bridge symptom and has been linked to PTSD symptoms of irritability (Vanzhula et al., 2019), trouble experiencing positive feelings, and concentration difficulties (Nelson et al., 2022). Similarly, re-experiencing/intrusion symptoms have been linked to ED symptoms (Liebman et al., 2021), and in a separate sample, the re-experiencing symptom of disturbing dreams was associated with body dissatisfaction (Vanzhula et al., 2019). Taken together, previous cross-sectional findings have highlighted important central and bridge symptoms to consider for future research replication.

Psychological network analysis also has the capacity to conduct longitudinal analyses of time series data (Epskamp et al., 2018). Time series data allow examination of temporal dynamics between symptoms and can establish sequential ordering – a precondition of inferring causality. Time series network analysis produces three separate network structures: 1) a temporal network, 2) a contemporaneous network, and 3) a between-subjects network. The temporal network represents how symptoms precede one another in time. The contemporaneous network represents symptoms that occur at the same time, or within the time scale of the assessment window, accounting for temporal effects that have already been modeled. Both the temporal and contemporaneous networks are driven by intra-individual processes. Finally, the between-

subjects network represents the association between average symptoms across the time period of study across all participants, thus driven by cross-individual differences. To our knowledge, there are no studies that examine PTSD and ED symptoms in the same network. Ecological momentary assessments (EMA) are a feasible and acceptable way to assess the theoretically longitudinal relationships between symptoms over short time windows. Prior studies have utilized EMA to study longitudinal symptomology in different samples of PTSD (Walz et al., 2014) patients and ED (Engel et al., 2016) patients and has led to important new insights into the relationships between symptoms.

The primary aim of the current study was to assess longitudinal associations between ED and PTSD symptoms, particularly central and bridge symptoms that may indicate pathways that maintain this comorbidity. The secondary aim of the current study was to assess concurrent associations between ED and PTSD symptoms to illuminate either symptoms that occur simultaneously or in quick succession. Finally, an exploratory aim of the current study was to identify central and bridge symptoms across participants, which would serve to potentially replicate existing cross-sectional findings. Based on previous cross-sectional research, central symptoms of the ED and PTSD comorbidity network were hypothesized to be restriction, binge eating, fear of weight gain, re-experiencing symptoms, and negative alterations in cognitions and mood. Bridge symptoms were hypothesized to be binge eating and re-experiencing symptoms.

Method

Sample

Undergraduates from US colleges and universities were recruited through the research pool, listservs, and flyers at San Diego State University as well as social media advertisements from late 2021 to spring 2023. Inclusion criteria for the current study were the following: 1) self-

reported age of 18-30 years, 2) have elevated ED symptoms (one standard deviation below the clinical norm or one standard deviation above the community norm; see measures for details), 3) meet PTSD criterion A with an event at least 1 month ago, 4) have elevated PTSD symptoms (one standard deviation above the norm for college students; see measures for details), 5) own a smartphone that could download the EMA application, and 6) read and speak English.

Procedures

Participants were recruited via two different sources. First, as part of a parent study that recruited via advertisement on the research participation pool website at the first author's institution, a subsection of the participants that had elevated PTSD and ED symptoms were invited to participate in the current study. Second, individuals were also recruited through listservs and flyers at the first author's institution and the social media platforms Facebook, Instagram, and Twitter. Potential participants from these sources were screened with an initial survey that assessed ED and PTSD symptoms. Individuals that screened positive, passed three attention check items (e.g., "For this question, select moderately"), and provided a valid college or university email address were invited to participate in the current study. Prior to completing EMA surveys, participants completed a one-time orientation visit session via video. During the orientation visit, participants were consented into the study, completed baseline questionnaires and demographic information (i.e., age, grade, race, ethnicity, height, weight, sex assigned at birth, gender identity, and sexual orientation), downloaded the EMA phone application, and completed test questions with a researcher to demonstrate understanding. Once the orientation visit was completed, participants began receiving surveys the next day.

This EMA protocol encompassed multiple reporting instances throughout the day, following the recommendations for EMA identified by previous studies (Burke et al., 2017). The

SEMA3 application was used to administer surveys (Koval et al., 2019). Participants received six notifications daily inviting them to complete a survey. Each survey took approximately 3 minutes to complete. If participants did not respond to the first notification, they received two additional reminders: one fifteen minutes after the initial notification and one 2.5 minutes prior to expiry. Signal times were determined by randomly selecting times around “anchor points” that subdivided the day into six approximately equal blocks of time: 8:30 a.m., 10:40 a.m., 12:50 p.m., 3:00 p.m., 5:20 p.m., and 7:30 p.m. The signal times were randomly distributed around these anchor times in a normal distribution with a standard deviation of 30 minutes to provide assessments evenly across the waking hours of the day. Participants were asked about their experiences “since the last assessment”; this is done so that there is no overlap of assessment data. The first assessment of the day also included two additional items: 1) experiences of nightmares overnight and 2) trouble falling or staying asleep. If participants were recruited through the participant pool, they were offered the option of compensation of up to four research credits or up to \$40 in Amazon gift card value. Participants recruited through other sources were compensated with up to \$40 in Amazon gift card value. Specifically, participants were informed that they would receive 1 credit or \$5 if they completed less than 25% of daily surveys, 2 credits or \$15 if they completed 25-50% of daily surveys, 3 credits or \$25 if they completed 51-75% of daily surveys, and 4 credits or \$40 if they completed greater than 75% of daily surveys. This study was approved by the Institutional Review Board at San Diego State University (HS-2021-0172).

Measures

Eating Pathology

During screening, participants completed the Eating Disorder Examination Questionnaire (EDE-Q; Fairburn & Beglin, 2008), a 28-item self-report measure. Participants responded on a 7-point Likert scale, ranging from 0 (no days) to 6 (every day). Participants one standard deviation below the mean for a clinical ED sample on the EDE-Q were considered to have elevated ED symptoms for cisgender men (1.38; Smith et al., 2017) and women (2.86; Welch et al., 2011). Of note, these values are similar to values of one standard deviation above the mean in normative samples of college women and men (Lavender et al., 2010; Luce et al., 2008). Since these norms differ by gender identity and clinical norms for transgender and gender-expansive individuals have not been established, participants one standard deviation above the mean for community samples were considered elevated for these gender identities. The following mean scores were considered elevated: 3.11 for transgender women (Nagata, Murray, et al., 2020), 3.12 for transgender men (Nagata, Murray, et al., 2020), and 3.09 for gender-diverse individuals (Nagata, Compte, et al., 2020).

EMA measurement of ED behavior followed prior research (e.g., Engel et al., 2013; Fuller-Tyszkiewicz et al., 2020). Participants were asked to report the following symptoms: body dissatisfaction, fear of weight gain, concentration impairment related to shape and weight, loss of control eating, restrictive eating, and self-induced vomiting or use of laxatives/diuretics (Engel et al., 2013; Fuller-Tyszkiewicz et al., 2020). Participants were asked to answer how much they have engaged in these behaviors since their last check-in on a scale of 0 (not at all) to 4 (extremely).

Posttraumatic Stress Symptoms

During screening, posttraumatic stress symptomology was measured with the PTSD Checklist for DSM-5 (PCL-5; Blevins et al., 2015) with the brief criterion A assessment

(Weathers et al., 2018). Participants responded on a 5-point Likert scale ranging from 0 (not at all) to 4 (extremely). Responses were reviewed by the first author to determine whether they would meet for criterion A. In unclear cases, the first author consulted with a senior expert in PTSD to determine eligibility. Participants that obtained a total score of 28 (Blevins et al., 2015) on the PCL-5 were considered to have elevated PTSD symptoms, approximately one standard deviation above the mean in two college samples (Blevins et al., 2015). EMA measurement of PTSD symptoms followed prior research (Short et al., 2017), such that 10 items were administered that load most strongly onto the four symptom clusters of PTSD in an undergraduate sample (Ashbaugh et al., 2016); items were averaged into scores by symptom cluster. The prompt for the PCL-5 items was altered to ask about how much these symptoms have bothered the participant since their last check-in on a scale of 0 (not at all) to 4 (extremely).

Data Analysis

Analyses were conducted in R version 4.2.2 (R Core Team, 2022). Participants were excluded from analyses if they completed less than 25% of the surveys (less than 21 total surveys). Missing time-series data was handled using the Kalman filter based on the state-space model (Hamaker & Grasman, 2012) as implemented in the *imputeTS* package (Moritz & Bartz-Beielstein, 2017). The Kalman filtering method of imputation has been found to perform well with small sample sizes and moderate (10-25%) levels of missing data (Mansueto et al., 2022). To assess the assumption of stationarity in vector autoregression (VAR) models, augmented Dickey-Fuller tests were conducted for selected network variables (Dickey & Fuller, 1979; Trapletti & Hornik, 2023). All tests were significant ($p < .01$) and therefore were not detrended.

Network Estimation

To address the aims of the current study, networks were estimated using multilevel vector autoregression (Epskamp et al., 2018) implemented in the package *mlVAR* (Epskamp et al., 2021) with the two-step estimation procedure (i.e., “lmer”). Centrality was determined using outgoing expected influence in the directed network (i.e., temporal network) and expected influence for undirected networks (i.e., contemporaneous and between-subjects networks; Robinaugh et al., 2016) in the *qgraph* package (Epskamp et al., 2012). Bridge symptoms were determined using 1-step expected influence (Jones et al., 2021) using the *networktools* package (Jones, 2022). Expected influence is a metric that aims to quantify the strength and direction of a node’s influence on a network to estimate how a node may help activate, maintain, or deactivate a network and was specifically developed for psychopathological networks (Robinaugh et al., 2016).

Results

Participants

A total of 52 participants completed the orientation visit and were enrolled in the study. Of these participants, 2 were excluded from analyses due to completing less than 25% of all surveys, for an analytic sample of 50 participants. Average compliance after removing these participants was 78.05% (SD = 14.73%), which is comparable to most other EMA studies (Wrzus & Neubauer, 2023). The majority of participants identified as women ($n = 40$, 80.0%), white ($n = 38$, 76.0%), and non-Hispanic/Latino ($n = 36$, 72.0%). More than half of participants ($n = 27$) identified their criterion A trauma event as sexual assault or other unwanted sexual experiences. See Table for demographic information and descriptive statistics.

Temporal Network

See Figure 3.1 for a visual depiction of the temporal network including directed edges and edge weights and Table 3.2 for a summary of the network analysis. Loss of control eating ($z = 1.44$) demonstrated the greatest outgoing expected influence centrality index and was associated with subsequent concentration impairment related to shape and weight (fixed effect coefficient = .07, $SE = .02$), fear of weight gain (fixed effect coefficient = .06, $SE = .02$), body dissatisfaction (fixed effect coefficient = .06, $SE = .02$), negative alterations in cognition and mood (fixed effect coefficient = .05, $SE = .01$), and re-experiencing symptoms (fixed effect coefficient = .03, $SE = .02$). The ED symptoms of loss of control eating ($z = 1.40$) and concentration impairment related to weight and shape ($z = 1.37$) demonstrated the highest bridge expected influence indices within the ED community. Loss of control eating was most strongly associated with negative alterations in cognition and mood (fixed effect coefficient = .05, $SE = .01$) and concentration impairment related to shape and weight was most strongly associated with avoidance (fixed effect coefficient = .08, $SE = .03$). The negative alterations in cognition and mood ($z = 1.08$) symptom demonstrated the highest bridge expected influence index in the PTSD community and was most strongly associated with concentration impairment related to shape and weight in the ED community (fixed effect coefficient = .07, $SE = .03$). See Figure 3.2 for a visual depiction of the temporal network node centrality and bridge indices.

Contemporaneous Network

See Figure 3.1 for a visual depiction of the contemporaneous network including edge weights and Table 3.3 for a summary of the network analysis. The fear of weight gain ($z = 1.27$) demonstrated the greatest expected influence centrality and was associated with the concomitant symptoms of concentration impairment related to shape and weight (partial $r = .50$), body dissatisfaction (partial $r = .25$), loss of control eating (partial $r = .12$), and re-experiencing

(partial $r = .07$). Body dissatisfaction ($z = 1.74$) demonstrated the highest bridge expected influence in the ED symptom community and arousal symptoms ($z = 1.43$) demonstrated the highest bridge expected influence in the PTSD symptom community, meaning that these symptoms had the strongest direct influence on symptoms of the other community at the same timepoint. Body dissatisfaction was most strongly associated with the negative alterations in cognition and mood (partial $r = .08$) and arousal (partial $r = .08$) symptoms of PTSD. See Figure 3.3 for a visual depiction of the contemporaneous network node centrality and bridge indices.

Between-Subjects Network

See Figure 3.1 for a depiction of the between-subjects network including edge weights and Table 3.4 for a summary of the network analysis. Fear of weight gain ($z = 0.95$), arousal symptoms ($z = 0.92$), and negative alterations in cognition and mood ($z = 0.85$) demonstrated the highest expected influence centrality. Fear of weight gain was associated with body dissatisfaction (partial $r = .50$) and concentration impairment related to weight and shape (partial $r = .47$), arousal symptoms were associated with negative alterations in cognition and mood (partial $r = .63$) and restriction (partial $r = .32$), and negative alterations in cognition in mood were associated with arousal (partial $r = .63$) and body dissatisfaction (partial $r = .30$). The symptoms of arousal ($z = 1.36$) and negative alterations in cognition and mood ($z = 1.22$) demonstrated the strongest bridge expected influences in the PTSD cluster and body dissatisfaction ($z = 1.22$) demonstrated the strongest bridge expected influence in the ED cluster. Arousal was associated with the ED symptom of restriction (partial $r = .32$) and negative alterations in cognition and mood symptoms were associated with ED symptom of body dissatisfaction (partial $r = .30$). See Figure 3.4 for a visual depiction of the between-subjects network node centrality and bridge indices.

Discussion

The primary and secondary aims of the current study were to examine the within-person longitudinal and concurrent associations between ED and PTSD symptoms in a sample of individuals with elevated PTSD and ED symptoms through network analysis. As part of this examination, central symptoms in the temporal network (i.e., symptoms that had the most influence on later timepoints) and contemporaneous network (i.e., symptoms that had the most influence on the same timepoint) were identified. Bridge symptoms (i.e., symptoms in one diagnostic cluster that had the most influence on the other cluster) were also identified in both networks. As an exploratory aim, this study also examined associations between symptoms across participants in a between-subjects network to evaluate how this network compares to cross-sectional findings.

Longitudinal and Concurrent Symptom Associations

Loss of control eating was identified as the most central symptom in the temporal network. This finding is similar to cross-sectional findings identifying binge eating as central (Vanzhula et al., 2019). Loss of control eating was strongly associated with subsequent concentration impairment related to shape and weight, fear of weight gain, body dissatisfaction, and negative alterations in cognition and mood. Loss of control eating may therefore contribute to negative body-related thoughts, in line with prior theory (Fairburn, 2008). Loss of control eating may further serve to maintain PTSD-ED comorbidity by eliciting self-critical thoughts, bolstering common cognitively-mediated emotions in PTSD such as shame and guilt (LoSavio et al., 2017). The association of loss of control eating with subsequent negative alterations in cognition and mood may explain the differences in eating pathology presentation by traumatic exposure, as binge-purge subtype EDs have been more strongly linked to trauma as compared to

EDs characterized by restriction primarily (Caslini et al., 2016). Thus, loss of control eating appears to serve a central role in maintaining the PTSD and ED comorbidity, and should be targeted in treatment to avoid further activation of the network.

Fear of weight gain was identified as the most central symptom in the contemporaneous network, associated with concentration impairment related to shape and weight, body dissatisfaction, loss of control eating, and re-experiencing symptoms. This finding is perhaps unsurprising as fear of weight gain, body-related concentration impairment, and body dissatisfaction have all been characterized as attitudinal facets of body image disturbance (Garner & Garfinkel, 1982); however, these constructs have also been found to have differing relationships with ED psychopathology, indicating that they are not synonymous concepts (Linardon et al., 2018; Mitchison et al., 2017). Further, fear of weight gain was identified as a central symptom in cross-sectional work (Vanzhula et al., 2019), also while including body-related concentration impairment and body dissatisfaction in the network, indicating its unique salience in maintaining the ED-PTSD network. Prior research has suggested that fear of weight gain during mealtimes is associated with binge eating behavior (Ralph-Nearman et al., 2024), indicating that this may be a common link between symptoms for people with eating disorders. However, the finding linking fear of weight gain with re-experiencing symptoms is novel. Given that more than half the sample identified their index trauma as sexual-related, perhaps participants were experiencing intrusion symptoms that prompted fear of weight gain. Sexual trauma in particular has been linked to distorted body image (Madowitz et al., 2015), though distorted body image can also occur after other forms of trauma. Regardless, fear of weight gain may be an important symptom to target in intervention to reduce comorbid PTSD and ED symptomology.

Concentration impairment related to shape and weight was identified as a strong bridge influences from ED to PTSD symptoms in the temporal network, similar to previous network analyses (Nelson et al., 2022; Vanzhula et al., 2019). Body-related concentration impairment was most strongly associated with subsequent avoidance symptoms in the PTSD cluster. This association may indicate that thinking about shape and weight may lead to avoidance, perhaps because of the association of weight and shape-related thoughts with trauma-related thoughts and cues. For instance, individuals with traumatic experiences relating to the body (e.g., physical assault, sexual assault, physical injury), often have distorted body image (Preti et al., 2006; Treuer et al., 2005; Weaver et al., 2007). Day to day body-related thoughts may therefore lead to thoughts about an associated traumatic event, and subsequent avoidance behaviors. Improving weight and shape-related cognitions may lead to decreases in avoidance behaviors.

Negative alterations in cognition and mood were also identified as an important bridge symptom in the temporal network and was most strongly related to concentration impairment related to weight and shape in the ED cluster. Bolstering this purported relationship, a pathway between body dissatisfaction and negative alterations in cognition and mood was identified as an important bridge in the contemporaneous network and between-subjects network, echoing cross-sectional findings (Vanzhula et al., 2019). Previously, researchers have theorized that PTSD symptoms such as negative alterations in cognition and mood can cause or exacerbate existing negative beliefs about the self, including the body, thus strengthening the ED (Mitchell et al., 2021; Trottier et al., 2016). Body dissatisfaction was also strongly associated with arousal symptoms in the contemporaneous network. Body dissatisfaction may serve as a relatively common daily stressor and therefore associated with increased manifestations of stress such as irritable behavior or difficulty concentrating, symptoms assessed by the arousal cluster in PTSD.

Alternatively, experimental studies have found that stress induced by social situations increases body dissatisfaction in women with binge ED (Naumann et al., 2018). Thus, other daily stressors may increase arousal symptoms and body dissatisfaction simultaneously.

Between-Subjects Associations

In the between-subjects network, fear of weight gain, arousal symptoms, and negative alterations in cognition and mood were the most central symptoms. The strongest bridge symptoms were arousal symptoms, negative alterations in cognition and mood and body dissatisfaction. The strongest bridge pathway was between and arousal symptoms and restriction. Arousal symptoms such as difficulties with sleep and recklessness have also been linked to restriction in previous network analyses (Nelson et al., 2022). The current findings are in line with theoretical work hypothesizing that ED symptoms (such as restriction) may serve to blunt the experience of arousal symptoms for individuals with PTSD (Mitchell et al., 2021; Trottier et al., 2016). Thus, restriction serve a self-medicating function for arousal symptoms individuals with PTSD.

Strengths and Limitations

The strengths of the current study include the use of time series data, which allows examination of short-term longitudinal associations between symptoms of EDs and PTSD. Examination of these symptoms longitudinally moves beyond cross-sectional examinations comparing individuals to examine dynamic relationships across time. Further, the current sample was recruited specifically with elevated pathology in both PTSD and EDs. Recruiting individuals with elevated symptoms may reveal associations between symptoms that do not exist as compared to individuals without elevated pathology. However, recruiting only individuals that meet both an ED and PTSD diagnostic criteria may artificially reduce variability in symptoms

and affect network results (see Terluin et al., 2016); thus, a sample with elevated pathology was preferred.

The current study has some limitations to note. First, the current study cannot conclude that observed symptom associations are causal. Second, the study sample consisted of undergraduate students who mostly identified as cisgender white women. The current results may not generalize to other groups as differences in post-trauma reactions (Patel & Hall, 2021) and eating pathology (Levinson & Brosf, 2016) are well-documented cross-culturally. Third, the current study used adapted items from validated measures for EMA; however, the psychometric properties of these items are currently unknown. While this is common in the EMA literature, future research should establish valid and reliable measurement that can be used in EMA studies.

Future Directions

The current study has implications for the theoretical overlap of PTSD and EDs. The current study bolstered evidence for some existing theorized associations between ED and PTSD symptoms. For example, PTSD has been theorized to cause or exacerbate body dissatisfaction, leading to downstream increases in eating pathology, through negative alterations in self-focused cognitions (Mitchell et al., 2021; Trottier et al., 2016), a pathway that was observed in the current study. Cross-sectional network analyses had not yet observed this association. However, other pathways were observed that have not yet been theorized, such as the associations between 1) concentration impairment related to shape and weight and avoidance symptoms in the temporal network and 2) body dissatisfaction and arousal symptoms in the contemporaneous network. As these are novel findings, future researchers should examine whether similar pathways emerge in future research.

The current study also has implication for future treatment development. Given that network analysis identifies the most influential symptoms in maintaining psychopathology, treatments focused on disrupting these symptoms and their relationships should be the most effective for addressing psychopathology (Smith et al., 2018). Targeting these symptoms may maximally disrupt the relationships between symptoms and prevent further symptom activation (Smith et al., 2018), therefore, fully addressing empirically derived symptom relationships. Thus, future treatment development research targeting both ED and PTSD symptoms should target loss of control eating, concentration impairment related to shape and weight, negative alterations in cognition and mood, fear of weight gain, body dissatisfaction, and arousal symptoms as these were identified as important central and bridge symptoms in the temporal and contemporaneous networks. Treatments developed from network analyses should be theoretically more effective than treatments developed from clinical intuition alone.

Conclusion

Many researchers have postulated that ED and PTSD symptoms may have functional relationships that maintain this comorbidity. While these symptoms have been examined in prior cross-sectional network analyses, this study is the first to examine these associations in a longitudinal network analysis. Our results may offer insights into important maintaining symptoms for comorbid ED and PTSD. Our findings suggest that loss of control eating, concentration impairment related to shape and weight, and negative alterations in cognition and mood are important drivers of temporal changes in ED and PTSD symptoms.

Dissertation Author's Acknowledgements

I, Alexandra D. Convertino (primary author of this material), would like to thank all coauthors — Ms. Mendoza and Dr. Blashill — for their contributions to this work.

Tables

Table 3.1: *Participant demographic information and descriptive statistics*

Characteristic	Analytic Sample (N=50)
Age (years)	
Mean (<i>SD</i>)	20.06 (2.12)
Range	18.00 - 29.00
PCL-5 (sum)	
Mean (<i>SD</i>)	46.24 (13.98)
Range	28.00 - 80.00
EDE-Q (mean)	
Mean (<i>SD</i>)	4.28 (0.86)
Range	1.59 - 5.73
	<i>N</i> (%)
Provisional PTSD Diagnosis	
Yes	35 (70.0%)
No	15 (30.0%)
Positive Eating Disorder Screen	
Yes	38 (76.0%)
No	12 (24.0%)
Grade	
Freshman	15 (30.0%)
Sophomore	15 (30.0%)
Junior	7 (14.0%)
Senior	8 (16.0%)
Senior (5+ years)	5 (10.0%)
Sex Assigned at Birth	
Male	4 (8.0%)
Female	46 (92.0%)
Intersex	0 (0.0%)
Gender ^a	
Man	6 (12.0%)
Woman	40 (80.0%)
Nonbinary	3 (6.0%)
Self-described: transmasculine	1 (2.0%)
Race ^b	
White	38 (76.0%)
Black or African American	1 (2.0%)
Native American or American Indian	0 (0.0%)
Asian or Pacific Islander	12 (24.0%)
Different race ^c	2 (4.0%)

Table 3.1: *Participant demographic information and descriptive statistics, continued*

Characteristic	Analytic Sample (N=50)
Ethnicity	
Non-Hispanic/Latino	36 (72.0%)
Hispanic/Latino	14 (28.0%)
Sexual Orientation	
Heterosexual or straight	21 (42.0%)
Gay or Lesbian	3 (6.0%)
Bisexual	15 (30.0%)
Pansexual	4 (8.0%)
Queer	5 (10.0%)
Different sexual orientation ^d	2 (4.0%)
Type of Criterion A Event Identified	
Natural disaster	1 (2.0%)
Transportation accident	4 (8.0%)
Serious accident at work, home, or during recreational activity	1 (2.0%)
Physical assault	3 (6.0%)
Assault with a weapon	2 (4.0%)
Sexual assault	26 (52.0%)
Other unwanted or uncomfortable sexual experience	1 (2.0%)
Life-threatening illness or injury	7 (14.0%)
Sudden violent death (for example, homicide, suicide)	3 (6.0%)
Sudden accidental death	1 (2.0%)
Any other very stressful event or experience: stalking	1 (2.0%)

Abbreviations: EDE-Q, eating disorder examination questionnaire; PCL-5, posttraumatic stress disorder checklist for the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition; SD, standard deviation.

^a Options also included agender, genderfluid, and genderqueer, but these options were not endorsed in the sample.

^b Categories are not mutually exclusive; participants were permitted to identify more than one race.

^c The two participants who self-described identified as: “ethnically native but not racially” and “mixed race.”

^d The two participants who self-described identified as demisexual and questioning.

Table 3.2: Summary of temporal network of eating disorder and posttraumatic stress disorder symptoms

Path		Fixed Effect Estimate	SE	p	Random Effects SD
From	To				
re-experiencing	re-experiencing	.49	.04	<.001	.24
re-experiencing	avoidance	.10	.03	.002	.15
re-experiencing	negative alterations in cognition and mood	.02	.03	.532	.10
re-experiencing	arousal	.08	.02	<.001	.08
re-experiencing	body dissatisfaction	-.02	.03	.382	.10
re-experiencing	fear of weight gain	.02	.03	.476	.11
re-experiencing	concentration impairment related to shape and weight	.00	.03	.998	.10
re-experiencing	restriction	.01	.02	.782	.03
re-experiencing	loss of control	-.01	.03	.652	.03
re-experiencing	purging	-.01	.04	.829	.09
avoidance	re-experiencing	-.10	.02	<.001	.10
avoidance	avoidance	.13	.04	<.001	.17
avoidance	negative alterations in cognition and mood	.02	.03	.509	.11
avoidance	arousal	-.06	.02	.002	.06
avoidance	body dissatisfaction	.01	.03	.710	.09
avoidance	fear of weight gain	-.03	.03	.362	.11
avoidance	concentration impairment related to shape and weight	-.01	.03	.749	.08
avoidance	restriction	-.02	.02	.501	.07
avoidance	loss of control	.00	.03	.980	.11
avoidance	purging	.03	.04	.421	.12
negative alterations in cognition and mood	re-experiencing	.03	.02	.151	.04
negative alterations in cognition and mood	avoidance	.10	.03	.002	.14
negative alterations in cognition and mood	negative alterations in cognition and mood	.31	.04	<.001	.22
negative alterations in cognition and mood	arousal	.03	.02	.163	.09
negative alterations in cognition and mood	body dissatisfaction	.03	.03	.329	.15
negative alterations in cognition and mood	fear of weight gain	.03	.03	.250	.10
negative alterations in cognition and mood	concentration impairment related to shape and weight	.07	.03	.025	.14
negative alterations in cognition and mood	restriction	.00	.03	.881	.12

Table 3.2: *Summary of temporal network of eating disorder and posttraumatic stress disorder symptoms, continued*

Path		Fixed Effect Estimate	SE	p	Random Effects SD
From	To				
negative alterations in cognition and mood	loss of control	.00	.03	.877	.07
negative alterations in cognition and mood	purging	.00	.04	.925	.12
arousal	re-experiencing	.07	.02	.001	.08
arousal	avoidance	.03	.03	.242	.11
arousal	negative alterations in cognition and mood	.07	.02	.003	.09
arousal	arousal	.52	.04	<.001	.21
arousal	body dissatisfaction	.00	.03	.985	.09
arousal	fear of weight gain	.03	.03	.273	.10
arousal	concentration impairment related to shape and weight	.03	.03	.286	.10
arousal	restriction	.04	.03	.112	.12
arousal	loss of control	.03	.03	.273	.10
arousal	purging	.01	.03	.658	.05
body dissatisfaction	re-experiencing	-.02	.02	.248	.06
body dissatisfaction	avoidance	.01	.03	.814	.14
body dissatisfaction	negative alterations in cognition and mood	.01	.02	.718	.10
body dissatisfaction	arousal	-.02	.02	.260	.07
body dissatisfaction	body dissatisfaction	.21	.03	<.001	.18
body dissatisfaction	fear of weight gain	.03	.03	.253	.13
body dissatisfaction	concentration impairment related to shape and weight	.03	.02	.217	.08
body dissatisfaction	restriction	.00	.02	.873	.06
body dissatisfaction	loss of control	-.02	.02	.461	.05
body dissatisfaction	purging	.02	.04	.643	.18
fear of weight gain	re-experiencing	.00	.02	.971	.09
fear of weight gain	avoidance	-.06	.03	.056	.14
fear of weight gain	negative alterations in cognition and mood	.00	.03	.914	.10
fear of weight gain	arousal	-.01	.03	.811	.11
fear of weight gain	body dissatisfaction	.07	.03	.027	.13
fear of weight gain	fear of weight gain	.21	.04	<.001	.23
fear of weight gain	concentration impairment related to shape and weight	.07	.04	.080	.20
fear of weight gain	restriction	.03	.03	.293	.14
fear of weight gain	loss of control	.05	.04	.168	.19
fear of weight gain	purging	-.03	.04	.371	.12

Table 3.2: Summary of temporal network of eating disorder and posttraumatic stress disorder symptoms, continued

Path		Fixed	SE	p	Random
From	To	Effect			Effects
		Estimate			SD
concentration impairment related to shape and weight	re-experiencing	.02	.03	.466	.15
concentration impairment related to shape and weight	avoidance	.08	.03	.012	.17
concentration impairment related to shape and weight	negative alterations in cognition and mood	-.01	.02	.743	.08
concentration impairment related to shape and weight	arousal	.03	.02	.193	.09
concentration impairment related to shape and weight	body dissatisfaction	.02	.03	.475	.11
concentration impairment related to shape and weight	fear of weight gain	.06	.02	.015	.08
concentration impairment related to shape and weight	concentration impairment related to shape and weight	.22	.04	<.001	.22
concentration impairment related to shape and weight	restriction	-.01	.02	.565	.09
concentration impairment related to shape and weight	loss of control	.01	.03	.661	.08
concentration impairment related to shape and weight	purging	-.01	.03	.679	.10
restriction	re-experiencing	.00	.02	.981	.08
restriction	avoidance	.01	.02	.607	.09
restriction	negative alterations in cognition and mood	.04	.02	.045	.06
restriction	arousal	.02	.02	.325	.06
restriction	body dissatisfaction	-.03	.02	.205	.09
restriction	fear of weight gain	.04	.02	.132	.10
restriction	concentration impairment related to shape and weight	.02	.02	.456	.09
restriction	restriction	.20	.05	<.001	.28
restriction	loss of control	.07	.03	.008	.11
restriction	purging	-.02	.03	.570	.05
loss of control	re-experiencing	.03	.02	.030	.06
loss of control	avoidance	.03	.02	.141	.10
loss of control	negative alterations in cognition and mood	.05	.01	<.001	.04
loss of control	arousal	.02	.02	.158	.05
loss of control	body dissatisfaction	.06	.02	.003	.07
loss of control	fear of weight gain	.06	.02	<.001	.04

Table 3.2: *Summary of temporal network of eating disorder and posttraumatic stress disorder symptoms, continued*

Path		Fixed Effect Estimate	SE	<i>p</i>	Random Effects SD
From	To				
loss of control	concentration impairment related to shape and weight	.07	.02	.001	.08
loss of control	restriction	-.02	.02	.169	.05
loss of control	loss of control	.25	.05	<.001	.32
loss of control	purging	.02	.03	.456	.11
purging	re-experiencing	.02	.01	.056	.04
purging	avoidance	.05	.02	.006	.07
purging	negative alterations in cognition and mood	.00	.01	.867	.05
purging	arousal	.00	.01	.832	.02
purging	body dissatisfaction	-.02	.02	.381	.08
purging	fear of weight gain	.00	.01	.999	.02
purging	concentration impairment related to shape and weight	.02	.01	.095	.02
purging	restriction	.03	.01	.069	.04
purging	loss of control	-.01	.01	.542	.02
purging	purging	.28	.06	<.001	.26

Abbreviations: SD, standard deviation; SE, standard error.

Table 3.3: *Summary of contemporaneous network of eating disorder and posttraumatic stress disorder symptoms*

Path		$p\ 1 \rightarrow 2$	$p\ 1 \leftarrow 2$	Partial Correlation	Correlation
Node 1	Node 2				
avoidance	re-experiencing	<.001	<.001	.53	.59
negative alterations in cognition and mood	re-experiencing				
negative alterations in cognition and mood	avoidance	.105	.060	.05	.30
arousal	re-experiencing	<.001	<.001	.21	.37
arousal	avoidance	<.001	<.001	.11	.27
arousal	negative alterations in cognition and mood	.044	.056	.04	.26
body dissatisfaction	re-experiencing	<.001	<.001	.33	.43
body dissatisfaction	avoidance	<.001	<.001	.03	.13
body dissatisfaction	negative alterations in cognition and mood	.206	.202	-.01	.12
body dissatisfaction	arousal	.794	.765	.08	.21
fear of weight gain	re-experiencing	<.001	<.001	.08	.21
fear of weight gain	avoidance	.001	<.001	.08	.21
fear of weight gain	negative alterations in cognition and mood	.003	.005	.07	.16
fear of weight gain	arousal	.845	.901	.00	.14
fear of weight gain	body dissatisfaction	.110	.188	.04	.21
concentration	re-experiencing	.193	.524	.02	.19
impairment related to shape and weight concentration	re-experiencing	<.001	<.001	.25	.41
impairment related to shape and weight concentration	avoidance	.041	.145	-.04	.10
impairment related to shape and weight concentration	negative alterations in cognition and mood	.291	.586	.02	.11
impairment related to shape and weight concentration	arousal	.314	.045	.04	.19
impairment related to shape and weight concentration	body dissatisfaction	.001	.001	.06	.19
impairment related to shape and weight concentration	fear of weight gain	<.001	<.001	.13	.35
restriction	re-experiencing	<.001	<.001	.50	.59
restriction	avoidance	.569	.158	-.02	.02
		.193	.049	.03	.04

Table 3.3: *Summary of contemporaneous network of eating disorder and posttraumatic stress disorder symptoms, continued*

Path		$p\ 1 \rightarrow 2$	$p\ 1 \leftarrow 2$	Partial Correlation	Correlation
Node 1	Node 2				
restriction	negative alterations in cognition and mood	.093	.037	.03	.05
restriction	arousal	.267	.603	.02	.04
restriction	body dissatisfaction	.352	.339	-.02	.01
restriction	fear of weight gain	.045	.212	.04	.05
restriction	concentration impairment related to shape and weight	.409	.244	.02	.04
loss of control	re-experiencing	.967	.744	.00	.05
loss of control	avoidance	.398	.813	.01	.05
loss of control	negative alterations in cognition and mood	.057	.032	.04	.08
loss of control	arousal	.106	.058	-.04	.02
loss of control	body dissatisfaction	.398	.388	.02	.10
loss of control	fear of weight gain	<.001	<.001	.12	.18
loss of control	concentration impairment related to shape and weight	.158	.075	.04	.14
loss of control	restriction	.035	.007	-.12	-.11
purging	re-experiencing	.884	.751	.00	.01
purging	avoidance	.814	.907	.00	.02
purging	negative alterations in cognition and mood	.036	.250	.03	.04
purging	arousal	.829	.738	-.01	.01
purging	body dissatisfaction	.583	.501	.01	.02
purging	fear of weight gain	.823	.806	.00	.02
purging	concentration impairment related to shape and weight	.611	.901	.00	.01
purging	restriction	.858	.537	-.01	-.01
purging	loss of control	.010	.012	.06	.06

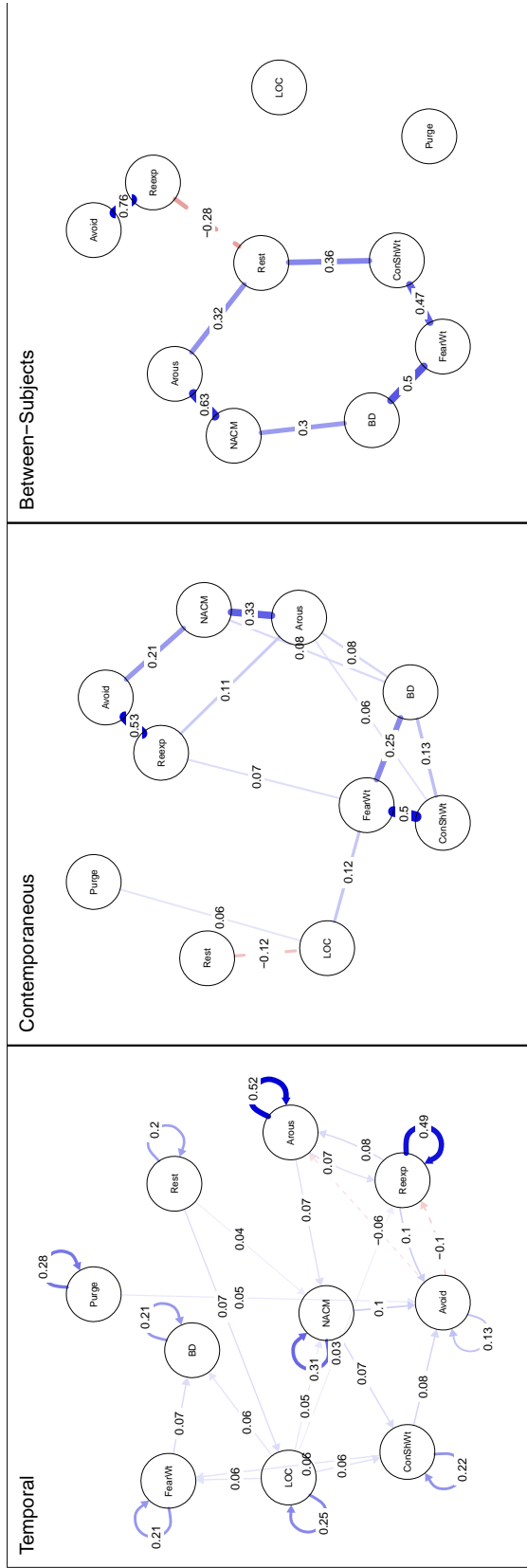
Note: Correlations and paths are estimated post-hoc.

Table 3.4: Summary of between-subjects network of eating disorder and posttraumatic stress disorder symptoms

Path		$p\ 1 \rightarrow 2$	$p\ 1 \leftarrow 2$	Partial Correlation	Correlation
Node 1	Node 2				
avoidance	re-experiencing	<.001	<.001	.76	.97
negative alterations in cognition and mood	re-experiencing				
negative alterations in cognition and mood	avoidance	.533	.640	.01	.91
arousal	re-experiencing	.229	.063	.17	.93
arousal	avoidance	.156	.188	.16	.93
arousal	negative alterations in cognition and mood	.527	.208	.11	.94
body dissatisfaction	re-experiencing	<.001	<.001	.63	.94
body dissatisfaction	avoidance	.145	.276	.16	.61
body dissatisfaction	negative alterations in cognition and mood	.036	.235	-.20	.62
body dissatisfaction	arousal	.016	.007	.30	.70
fear of weight gain	re-experiencing	.397	.003	-.22	.58
fear of weight gain	avoidance	.015	.190	-.23	.66
fear of weight gain	negative alterations in cognition and mood	.138	.060	.21	.69
fear of weight gain	arousal	.092	.036	.24	.75
fear of weight gain	body dissatisfaction	.046	.070	-.24	.65
concentration	re-experiencing	<.001	<.001	.50	.85
impairment related to shape and weight					
concentration	avoidance	.100	.369	.15	.85
impairment related to shape and weight					
concentration	negative alterations in cognition and mood	.869	.985	.01	.87
impairment related to shape and weight					
concentration	arousal	.435	.199	-.13	.87
impairment related to shape and weight					
concentration	body dissatisfaction	.067	.131	.21	.87
impairment related to shape and weight					
concentration	fear of weight gain	.527	.221	.12	.75
impairment related to shape and weight					
restriction	re-experiencing	<.001	<.001	.47	.83
restriction	avoidance	.013	.011	-.28	.77
		.147	.021	.21	.81

Table 3.4: Summary of between-subjects network of eating disorder and posttraumatic stress disorder symptoms, continued

Path		$p\ 1 \rightarrow 2$	$p\ 1 \leftarrow 2$	Partial Correlation	Correlation Node 1
Node 1	Node 2				
restriction	negative alterations in cognition and mood	.196	.244	-.14	.77
restriction	arousal	.003	.006	.32	.83
restriction	body dissatisfaction	.908	.354	.06	.56
restriction	fear of weight gain	.661	.235	-.11	.62
restriction	concentration impairment related to shape and weight	.005	.002	.36	.83
loss of control	re-experiencing	.664	.491	.01	.63
loss of control	avoidance	.884	.698	-.02	.64
loss of control	negative alterations in cognition and mood	.008	.661	-.12	.63
loss of control	arousal	.023	.104	.23	.66
loss of control	body dissatisfaction	.437	.939	.05	.51
loss of control	fear of weight gain	.504	.539	.08	.56
loss of control	concentration impairment related to shape and weight	.300	.344	.13	.67
loss of control	restriction	.786	.265	-.08	.56
purging	re-experiencing	.469	.528	.08	.65
purging	avoidance	.067	.997	.10	.67
purging	negative alterations in cognition and mood	.273	.657	-.09	.61
purging	arousal	.413	.495	.00	.63
purging	body dissatisfaction	.047	.613	.14	.50
purging	fear of weight gain	.655	.496	.02	.51
purging	concentration impairment related to shape and weight	.475	.397	-.10	.61
purging	restriction	.008	.051	.28	.66
purging	loss of control	.461	.322	-.03	.42



Figures

Figure 3.1: Networks of eating disorder and posttraumatic stress disorder symptoms

Note. Orange nodes represent posttraumatic stress disorder symptoms and blue nodes represent eating disorder symptoms. Edge weights are located in the middle of the path between nodes. Blue (solid) lines are positive associations and red (dashed) lines are negative associations.

Abbreviations: Arous, arousal; Avoid, avoidance; BD, body dissatisfaction; ConShWt, concentration impairment related to shape and weight; FearWt, fear of weight gain; LOC, loss of control; NACM, negative alterations in cognition and mood; Purge, purging; Reexp, re-experiencing; Rest, restriction

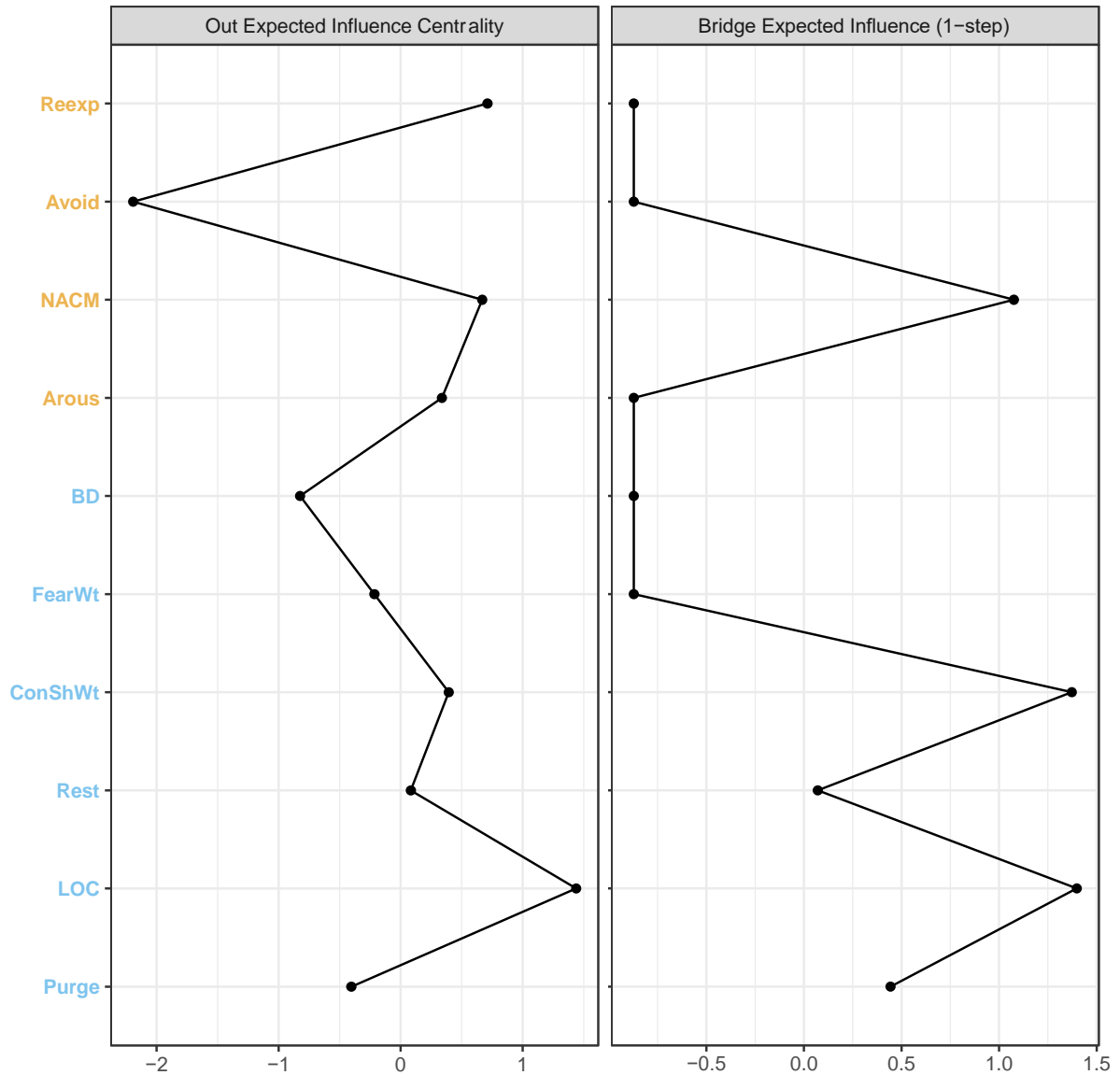


Figure 3.2: Plot of centrality and bridge coefficients for nodes in the temporal network

Note. Posttraumatic stress disorder symptoms are in orange text and eating disorder symptoms and in blue text. Coefficients are standardized.

Abbreviations: Arous, arousal; Avoid, avoidance; BD, body dissatisfaction; ConShWt, concentration impairment related to shape and weight; FearWt, fear of weight gain; LOC, loss of control; NACM, negative alterations in cognition and mood; Purge, purging; Reexp, re-experiencing; Rest, restriction.

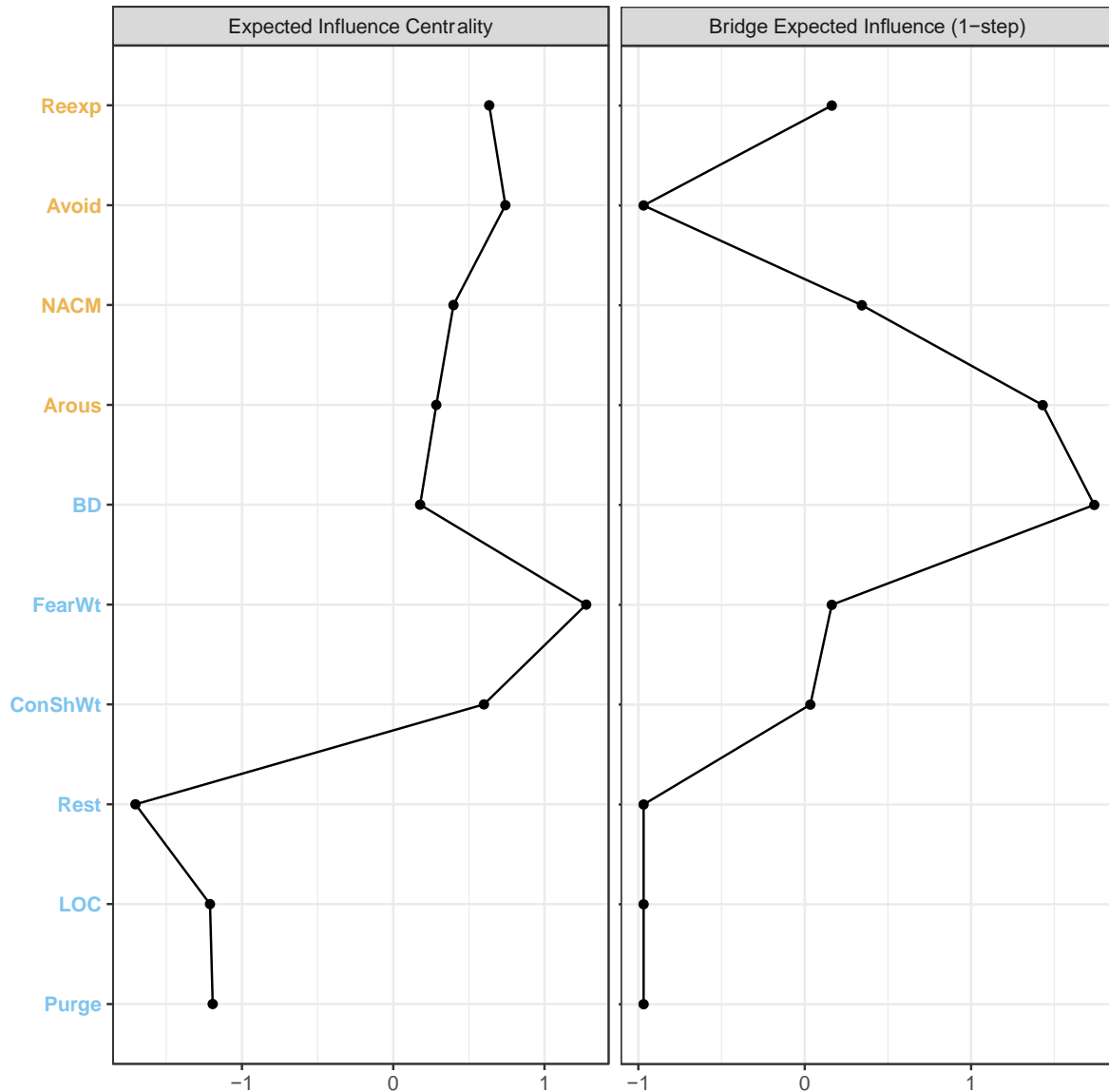


Figure 3.3: Plot of centrality and bridge coefficients for nodes in the contemporaneous network

Note. Posttraumatic stress disorder symptoms are in orange text and eating disorder symptoms and in blue text. Coefficients are standardized.

Abbreviations: Arous, arousal; Avoid, avoidance; BD, body dissatisfaction; ConShWt, concentration impairment related to shape and weight; FearWt, fear of weight gain; LOC, loss of control; NACM, negative alterations in cognition and mood; Purge, purging; Reexp, re-experiencing; Rest, restriction.

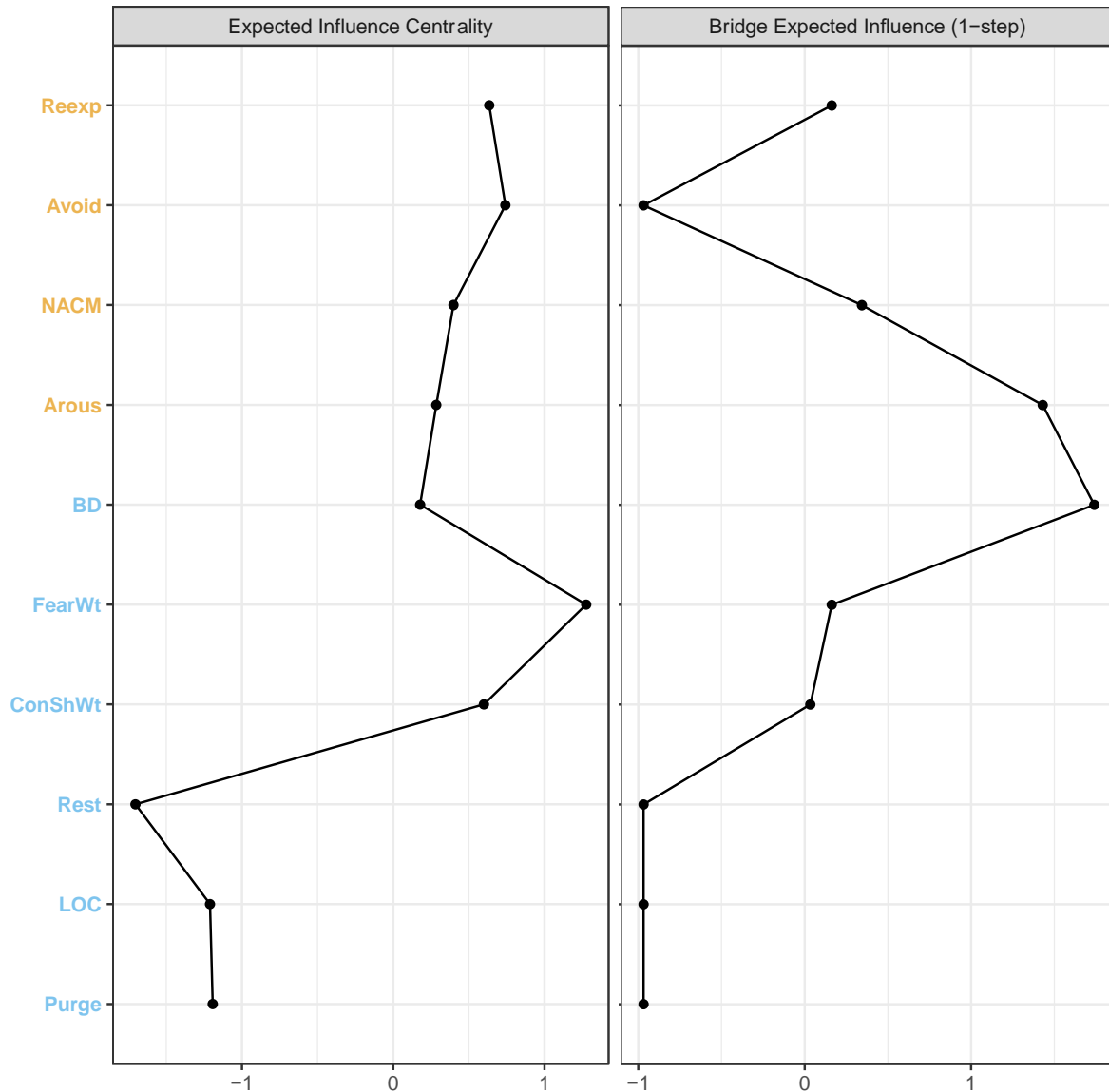


Figure 3.4: Plot of centrality and bridge coefficients for nodes in the between-subjects network

Note. Posttraumatic stress disorder symptoms are in orange text and eating disorder symptoms and in blue text. Coefficients are standardized.

Abbreviations: Arous, arousal; Avoid, avoidance; BD, body dissatisfaction; ConShWt, concentration impairment related to shape and weight; FearWt, fear of weight gain; LOC, loss of control; NACM, negative alterations in cognition and mood; Purge, purging; Reexp, re-experiencing; Rest, restriction.

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CHAPTER 4: INTEGRATED SUMMARY

Theoretical models in the field of eating disorders has emphasized how stress, broadly defined, can impact eating pathology (Brewerton, 2015), contributing to both onset (Pike et al., 2006; Rojo et al., 2006; Striegel-Moore et al., 2007) and maintenance (Harney et al., 2014; Naish et al., 2019; Smith et al., 2021). Specifically, theories such as the escape model of binge eating highlight how stressful experiences that induce self-critical thoughts may lead to eating pathology as a method of alleviating or avoiding these aversive thoughts (Heatherton & Baumeister, 1991). Though originally conceptualized as only encompassing binge eating behavior, research has also hypothesized that other eating disordered behaviors (e.g., restriction, purging) can also serve to escape negative affect (Pearson et al., 2015; Schmidt & Treasure, 2006).

Types of stressful experiences that have been extensively investigated include childhood trauma (Caslini et al., 2016; Pignatelli et al., 2017), sexual assault (Madowitz et al., 2015), and weight- and appearance-related bullying (Lie et al., 2019). However, many types of stressful experiences have yet to be investigated. For example, researchers have only recently examined how stressors associated with sexual minority identities may impact eating disordered behavior (Calzo et al., 2017). Most research in the area has focused on sociocultural (e.g., Tiggemann, 2011) or minority stress (Meyer, 2003) mechanisms for explaining disparities in eating pathology by sexual minority status, with only a few studies seeking to integrate the two theories (Calzo et al., 2017). Thus, **Study 1** sought to examine a comprehensive, integrated model to better explain eating disorder disparities among young sexual minority men and women. The integrated model tested explained more variance in disordered eating outcomes as compared to either model independently, thus emphasizing that incorporating stressors into eating disorder

sociocultural models may improve their explanatory power. Further, given that existing models for eating pathology have been used to inform interventions (Pennesi & Wade, 2016), improvements in theory can be translated into more effective interventions for certain subgroups of eating disorder patients.

Similarly, a recent systematic review concluded that exposure to traumatic events was associated with both premature dropout from and increased rate of relapse after eating disorder treatment (Convertino & Mendoza, 2023); thus, this patient population is in great need of treatment improvements. Trauma types that have been extensively explored are sexual assault (Madowitz et al., 2015), childhood abuse (Molendijk et al., 2017), and combat exposure (e.g., Arditte Hall et al., 2017, 2018), and a recent systematic review concluded that trauma and other severe adverse experiences are strong correlates of eating pathology. However, about 90% of the US population has experienced a trauma event (Kilpatrick et al., 2013), meaning that tailoring treatment to trauma-exposed populations may require a more targeted approach. **Study 2** then sought to both examine the association of eating disorders in a US-representative sample with relatively underexplored trauma types (i.e., non-sexual interpersonal trauma and non-interpersonal) and in a single model, such that the differential associations of each trauma type with each eating disorder can be observed. This study found that binge eating disorder was associated with all trauma types when placed in a single model, but only sexual interpersonal trauma was associated with anorexia nervosa in the multivariate model. Therefore, modifications to existing interventions should consider targeting specifically sexual trauma when treating anorexia nervosa, but may require a more universal intervention for trauma-exposed binge eating disorder patients.

Finally, individuals with posttraumatic stress disorder as a result of trauma exposure represent a unique class of patients in eating disorder treatment. The bidirectional associations between eating disorder and posttraumatic stress disorder symptoms (Mitchell et al., 2021; Trottier et al., 2016) mean that treatment for each disorder separately is unlikely to be successful. Indeed, posttraumatic stress disorder has been found to moderate treatment outcomes in eating disorder treatment (Convertino & Mendoza, 2023) and eating disordered behaviors were not reduced in posttraumatic stress disorder treatment (Mitchell et al., 2012). Therefore, new innovations in treatment are necessary to fully address this complex comorbidity. **Study 3** examined the longitudinal associations of posttraumatic stress disorder and eating disorder symptoms in a sample of US undergraduates with elevated symptoms with network analysis, an innovative framework for identifying the most important symptoms and associations to target during treatment. This study found that loss of control eating, concentration impairment related to shape and weight, purging, fear of weight gain, negative alterations in cognition and mood, and body dissatisfaction were the most important symptoms in the temporal and contemporaneous networks, indicating their potential salience for interventions.

Collectively, the findings from the three studies highlighted potential new avenues for both theoretical and clinical work. **Study 1** emphasizes the importance of including chronic stressors in existing models of eating pathology, and when considering sexual minority stress, the incremental improvement in variance explained for eating pathology. This finding suggests that traditional models of eating pathology may be incomplete for sexual minority individuals. Thus, minority stress may be an important target for tailoring both intervention and prevention efforts in this population, should findings from the current study be replicated in longitudinal studies. More immediately, practitioners that work with sexual minority individuals with eating

disorders should be aware that sexual minority stressors may influence eating disorder symptoms

Study 2 found that sexual assault might hold increased salience for individuals with anorexia nervosa as opposed to other trauma types; in contrast, all trauma types were associated with binge eating disorder. Researchers should consider examining potential longitudinal mediators in this relationship between types of traumatic exposure and specific eating disorder presentations in order to identify potential ways to either prevent or intervene for trauma-exposed populations.

Finally, **Study 3** highlighted the importance of key symptoms in the association between posttraumatic stress disorder and eating disorders and should be considered when creating treatment protocols for individuals with this comorbidity. These results may also guide practitioners in identifying important symptom relationships that must be disrupted. Collectively, the findings from these studies should inform future prevention and intervention tailoring for populations impacted by stressful experiences.

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