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**pro hac vice*

UNITED STATES DISTRICT COURT

NORTHERN DISTRICT OF CALIFORNIA

SAN JOSE DIVISION

JOHN D. HALTIGAN,

Plaintiff,

v.

MICHAEL V. DRAKE, in his official capacity as President of the University of California; CYNTHIA K. LARIVE, in her official capacity as Chancellor of UC Santa Cruz; BENJAMIN C. STORM, in his official capacity as Chair of the UC Santa Cruz Psychology Department; and KATHARYNE MITCHELL, in her official capacity as Dean of the UC Santa Cruz Division of Social Sciences,

Defendants.

No. 5:23-cv-2437-NC

SECOND AMENDED COMPLAINT

INTRODUCTION

1. The University of California (University or UC) has adopted a modern-day loyalty oath for professors who seek to join the faculty. Today's loyalty oath does not demand a pledge that professors are not members of the Communist Party, but professed agreement with "Diversity, Equity, and Inclusion" (DEI) policies and ideology. The DEI Statements demanded by the University are a thinly veiled attempt to ensure dogmatic conformity throughout the university system.

2. This requirement is imposed on every applicant to a faculty position in the University by means of a DEI Statement Requirement which applicants must clear in order to even get a foot in the door. The University administration ensures conformity and compliance by promulgating detailed rubrics and guidelines that tell applicants exactly what to say and what not to say in their Statements.

3. Dr. John D. Haltigan challenges this functional loyalty oath as a violation of his rights under the First Amendment. He has a PhD in Developmental Psychology and is ready and able to apply to a position at UC Santa Cruz, but the stringent ideological requirements of the DEI Statement make his application futile.

4. Dr. Haltigan is challenging the University of California's DEI Statement Requirement because what was true for the anti-communist loyalty oaths of the Cold War era is still true today: The First Amendment does not tolerate laws that cast a pall of orthodoxy over the classroom. *Keyishian v. Bd. of Regents of Univ. of State of N.Y.*, 385 U.S. 589, 603 (1967). Academic freedom and freedom of expression demand that mandatory DEI Statements meet the same fate as the loyalty oaths of previous generations.

JURISDICTION AND VENUE

5. This action arises under the First and Fourteenth Amendments to the United States Constitution and 42 U.S.C. § 1983. This Court has jurisdiction over this federal claim under 28 U.S.C. §§ 1331 (federal question) and 1343(a)(3) (redress for

deprivation of civil rights). Declaratory relief is authorized by the Declaratory Judgment Act, 28 U.S.C. § 2201.

6. Venue is proper in this Court under 28 U.S.C. § 1391(b)(2) because a substantial part of the events or omissions giving rise to the claim occurred and continue to occur in this district.

PARTIES

7. Plaintiff John D. Haltigan is a U.S. citizen and resident of Pennsylvania. He has a PhD in Developmental Psychology from the University of Miami, and until early in 2023 served as an Assistant Professor in the Department of Psychiatry at the University of Toronto.

8. Defendant Michael V. Drake is the President of the University of California and is sued in his official capacity.

9. Defendant Cynthia K. Larive is the Chancellor of UC Santa Cruz and is sued in her official capacity.

10. Defendant Benjamin C. Storm is a professor of psychology and the Chair of the UC Santa Cruz Psychology Department. He is sued in his official capacity.

11. Defendant Katharyne Mitchell is a professor of sociology and the Dean of the UC Santa Cruz Division of Social Sciences. She is sued in her official capacity.

FACTUAL BACKGROUND

The Evolution of the DEI Statement in the University of California

12. The University of California has long considered diversity to be an important value in faculty hiring.

13. Accordingly, in 2005, the University of California published a new section of its Academic Personnel Manual (APM) encouraging "diversity and equal opportunity." This section was designed to ensure that faculty which put effort into promoting equal opportunity and diversity receive some credit, but not to displace or substitute for scholarly rigor, objectivity, and originality.

14. Under the 2005 version of the APM, applicants were asked for DEI statements, but they were rarely decisive; DEI statements were weighed alongside more traditional measures of aptitude, including academic success, publications, research plans, and teaching ability.

15. Nor did the University provide prescriptive DEI statement guidelines and rubrics; the prevailing understanding of academic freedom prohibited the administration from dictating to faculty search committees about the beliefs of prospective academics.

16. Gradually, however, the University of California began to come under pressure to use DEI statements more aggressively to pursue ideological conformity and a vision of diversity focused on racial, ethnic, and gender balancing.

17. In 2015, the APM provision was revised, to add language that emphasized the importance of DEI achievement as compared to other traditional academic criteria.

18. In 2016, the California Budget Act allocated $2 million to promote racial and gender diversity, requiring a report from the University on fund usage and the racial/ethnic and gender composition of the University.

19. As a result, UC established the Advancing Faculty Diversity (AFD) program, which supports projects to increase racial and gender balance on UC campuses.

20. In November 2017, the UC Office of the President (UCOP) issued a detailed report on its use of the state funds.

21. The UCOP explained that the UC system was "particularly focused" on increasing diversity along racial and ethnic lines.

22. The UCOP Report highlighted a number of tools that particular departments or campuses could use to achieve the goal of enhanced racial and ethnic balance, including DEI statements.

23. As explained in the UCOP Report, AFD had allocated the state's funds to pilot programs that aimed to advance faculty racial and ethnic balancing within the constraint of Prop 209.

24. Among these programs was $600,000 for a UC Riverside program in the College of Engineering, which involved a unique approach to diversity statements.

25. UCOP highlighted UC Riverside as particularly successful because it resulted in a ten-fold increase in underrepresented minority finalists and a doubling of female representation.

26. According to UCOP, UC Riverside's success derived from their use of a simple rubric measuring research and diversity statements and particularly from their evaluation of DEI statements from the beginning of the candidate evaluation process and as part of the initial candidate screening.

27. In the following years, AFD received more state funding and has continued to build on its program to pursue racial balancing and ideological conformity and apply the lessons from the original effort. In 2018–19, AFD launched a grant program supporting campus efforts to increase diversity. This grant program is ongoing.

28. AFD has since launched five recruitment projects aiming to increase racial balance, at a total cost of about $2.5 million, including a pilot program at UC Santa Cruz.

29. The AFD-funded pilot program at UC Santa Cruz focused on several elements. Most importantly, it emphasized that DEI statements should be an "important part" of the selection process, which must be considered in the first round of review. The program also encouraged search committees to engage in more in-depth discussions about the value of these statements.

30. However, some search committees at UC Santa Cruz disregarded the emphasis on screening based on DEI statements, fearing they might lose top candidates.

31. This led the University and the administration on the Santa Cruz campus to refocus search committees on the importance of using DEI Statements aggressively.

32. Collectively, these initiatives and pressures have utterly transformed the DEI Statement's purpose and use in the University of California system.

33. Importantly, this transformation involved the widespread adoption of the UC Riverside experiment to perform an initial screening of candidates based only on the diversity statements (the Initial Screening Requirement).

34. The other major change has been the widespread adoption of detailed rubrics and guidelines to ensure uniformity.

35. For example, around the same time that the California State legislature was giving money to the University to adjust the racial and gender balance in its faculty, the University's Academic Personnel and Programs Office (APP) issued more detailed guidelines for evaluating DEI statements.

DEI Statements as Ideological Litmus Tests at UC Santa Cruz

36. Following these developments, UC Santa Cruz now provides prospective applicants with detailed guidelines on what to say and what not to say on their DEI statements.

37. On the main "Diversity" page for the UC Santa Cruz Office of Academic Personnel (APO), UC Santa Cruz makes clear that the University's understanding of diversity is about hiring and promoting individuals from specific racial and ethnic groups.

38. APO defines the terms "diversity," "equity," and "inclusion" in a specific manner that ensures successful applicants adhere to a particular ideology and worldview.

39. APO goes on to explain that DEI statements are evaluated in three categories: awareness, experience, and future plans at UC Santa Cruz.

40. Ideas and beliefs that applicants are supposed to convey are embedded throughout APO's expectations but particularly captured under the "awareness" heading.

41. Experience and future plans are evaluated based on an applicant's past or planned contributions to diversity, equity, and inclusion in teaching, research and professional work, and service and professional activities. The activities and contributions applicants are asked to discuss are thinly veiled proxies for particular beliefs that the administration favors.

42. The main diversity page also links to a "starting rubric," to further drive home to applicants exactly what they must say to pass through the DEI filter.[1]

43. To receive a high score under the terms set by the rubric, an applicant must express agreement with specific sociopolitical ideas, including the view that treating individuals differently based on their race or sex is desirable.

44. The rubric evaluates DEI statements based on the three criteria mentioned above: awareness (or "knowledge," as the rubric describes it), experience, and future plans, with a scoring range of 1–5 for each. 1–2 represents a low score, 3 represents a mixed score, and 4–5 represents a high score.

45. For each criterion, high scores are reserved for those who promise to adhere to a specific world view that requires treating individuals differently according to race.

46. Under the rubric, low scores are specifically promised for applicants that believe race and sex should not be used to judge individuals.

47. Further orthodoxy for applicants to recite is provided on a list on APO's website of "common myths" about DEI in faculty recruitment and hiring under its "Academic Recruitment Resources" page.[2]

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[1] *See* UCSC Starting Rubric to Assess Candidate Contributions to Diversity, Equity, and Inclusion, https://apo.ucsc.edu/docs/ucsc-rubrics-c2deistatements.pdf.

[2] *See* UC Merced Academic Personnel Office, *Addressing Common Myths About*

48. In the common myths document, among other things, the University makes clear its commitment to race-centric hiring and its focus on silencing dissent on these issues.

49. This document sends a clear message to applicants: those who reject DEI orthodoxy will demonstrate a low "understanding" or "awareness" of DEI and will not be considered for a position at UC Santa Cruz.

50. Finally, UC Santa Cruz's Psychology Department has a page for Resources on Antiracism under the heading of "DEI Resources."[3]

51. This page embraces without reservation numerous controversial political and ideological perspectives, including the ideas of controversial author Ibram Kendi, linking to and endorsing multiple speeches and works.

52. The documents on this page are not presented as academic research, or as the individual perspectives of particular professors, but as the official view of UC Santa Cruz's Psychology Department.

53. Individually and collectively, the guidelines, rubrics, and reference materials are intended to require applicants to repeatedly attest to particular beliefs to be considered for a position.

54. The mandatory beliefs have nothing to do with the University's mission, the qualifications for any given tenure-track position, or professional standards for academics. They are about propagating the ephemeral political ideology of the Administration.

55. Both the DEI Statement Requirement and the Initial Screening Requirement are applicable to every faculty job opening at UC Santa Cruz.

56. The combined result of this DEI Statement Requirement and the Initial Screening Requirement has created a situation where applicants who fail to

Diversity and Equity in Faculty Recruitment and Hiring, https://academicpersonnel.ucmerced.edu/common-myths-about-diversity-and-equity.

[3] *See* UC Santa Cruz Psychology Department, *Resources on Antiracism*, https://psychology.ucsc.edu/about/dei/dei-resources.html.

demonstrate conformity with the beliefs and ideology represented on the APO website know that their application is futile.

57. This process has the intent and the effect of driving contrary ideas and viewpoints out of the marketplace of academic hiring.

Dr. Haltigan's Job Search

58. Dr. Haltigan first learned about a position at UC Santa Cruz which fit his background and interests in early 2023.

59. At the time, Dr. Haltigan was (and remains) in a nationwide search for a tenure track position at a university.

60. Dr. Haltigan's job search is directed at places he would want to live and work, and to departmental openings that fit with his background and interests.

61. Academic jobs generally require the same materials for each application. Every academic job application requires at least a cover letter, a research statement, a teaching statement, and a curriculum vitae.

62. Job applicants for academic jobs seldom tailor these materials for the individual position at issue. Applicants often submit the same materials to every position they apply to, only changing a few words in the cover letter.

63. Many academic jobs also require some sort of statement on diversity. For these statements, candidates often reuse or slightly rework existing statements.

64. Dr. Haltigan applied to more than ten academic jobs in 2023, including multiple openings in California.

65. Dr. Haltigan's CV, attached as Exhibit A,[4] states that Dr. Haltigan obtained his PhD in Developmental Psychology from the University of Miami in 2009.

66. After obtaining his doctorate, Dr. Haltigan served as a postdoctoral fellow first at the University of Illinois at Urbana-Champaign (until 2011), then at the

[4] Although the attached application documents are from September of 2023, they reflect only minor changes in the time since the complaint was filed and Dr. Haltigan keeps all these materials ready to go at all times.

University of North Carolina at Greensboro (until 2013), then at the University of Ottawa (until 2016). *Id.*

67. From 2016 until earlier this year, Dr. Haltigan was an Assistant Professor in the Department of Psychiatry at the University of Toronto. *Id.*

68. His research interests include "the legacy of early caregiving experiences on child and adolescent environment"; "life history"; "[m]easurement and classification of child and adolescent psychopathology"; and "[l]ongitudinal data modeling." *Id.*

69. Dr. Haltigan has been a coinvestigator on several research programs operating under federal and other grants, has over 60 publications to his name, and has several additional manuscripts under review. *Id.*

70. Dr. Haltigan's Research Statement, attached as Exhibit B, further states that his "research program investigates the structure, determinants, course, and co-occurrence of child and adolescent mental and physical illness" and highlights quantitative approaches to address both basic and applied questions in developmental science. *Id.*

71. Dr. Haltigan's teaching statement, attached as Exhibit C, explains the importance to him of "passionate and personalized subject matter," and discusses his long history teaching and mentoring a diverse array of students.

72. A sample cover letter that Dr. Haltigan used for a university application in 2023 is attached as Exhibit D.

73. A sample of significant article reprint or preprints that Dr. Haltigan included in a university application in 2023 is attached as Exhibit E.

Dr. Haltigan's DEI Statement

74. In the midst of his job search, in February 2023, Dr. Haltigan posted a DEI statement he submitted with a recent job application to a university opening on his Substack newsletter. *See* Exhibit F.

75. As the introductory paragraph explains, Dr. Haltigan submitted the DEI statement to a job because he "strongly believe[s] taking a principled stand against the use of the DEI rubric in the Academy is crucial for the continued survival of our institutions of higher learning as they were intended: bastions of the unfettered pursuit of knowledge and truth and the immersion of its students into the principles of liberal discourse and the development of critical thought." *Id.*

76. Dr. Haltigan's posted DEI Statement states "I am committed to colorblind inclusivity, viewpoint diversity, merit-based evaluation, and value outreach to underrepresented groups in higher education. Across all of my teaching and mentorship, I have endeavored to treat students and mentees equally, without regard to identity-based characteristics." *Id.*

77. Dr. Haltigan's DEI statement expressed his fear of being discriminated against due to his viewpoint, observing that "[s]everal recent investigative journalism efforts have documented how DEI statements have been used to screen and penalize applicants for not possessing 'correct' political ideas or endorsing activist ideologies, such as the 'anti-racist' strand of 'scholarship' developed and promoted by Ibram Kendi as well as concepts such as 'intersectionality.'" *Id.*

78. Dr. Haltigan also writes that he has "provided mentorship to several students from underrepresented minority groups. Many of these students explicitly sought out my mentorship due to my clear position, communicated on social media, that I reject activist 'scholarship' that is neither conceptually coherent nor methodologically sound." *Id.*

UC Santa Cruz's Psychology Department's Job Opening

79. On July 21, 2022, UC Santa Cruz posted an open hiring announcement for a tenure-track position in Developmental Psychology. *See* Exhibit G.

80. The position stated that the University was "particularly interested in developmental psychology scholars whose research addresses diversity in human development. In addition, we seek a scholar whose research addresses the intersection

of developmental psychology and global and/or community health. Health here is broadly construed to include psychological, mental or physical health with a focus on the well-being of children and youth in their families, peer relations, schools, and/or cultural communities." *Id.*

81. As with every position at UC Santa Cruz in the Division of Social Sciences, the Psychology Department requires a DEI statement in order to apply, and "urges" candidates to review the scoring rubric explained above. *Id.*

82. It also makes clear that, consistent with University policy, an initial screening of candidates will be performed using only the DEI statement and a research statement.

83. In addition to the DEI Statement, the applicant must submit a cover letter, a curriculum vitae, a research statement, a teaching statement, and significant article reprint or preprints.

84. Dr. Haltigan's application materials at Exhibits A–E satisfy these requirements and could have been submitted to UC Santa Cruz with only minor edits to the cover letter.

85. UC Santa Cruz posts similar openings in the psychology department on an annual basis. On September 1, 2023, the University posted a hiring announcement for an assistant or associate professor in "quantitative psychology." *See* Exhibit H.

86. This hiring announcement seeks a candidate with a "PhD in Quantitative Psychology or in another field of Psychology" as long as they have expertise in quantitative methods and demonstrated excellence in teaching statistics. Dr. Haltigan meets these requirements and is currently teaching statistics at the undergraduate level at University of Miami.

87. This posting, like the other, is conditional on the University's ideologically demanding DEI Statement requirement.

88. Dr. Haltigan expects the University to continue posting job opportunities that he is interested in going forward.

Dr. Haltigan's Application Is Futile

89. Although Dr. Haltigan has over a decade of experience teaching and mentoring students from all backgrounds, his views on DEI rendered an application to UC Santa Cruz futile.

90. Dr. Haltigan is aware of research and investigative reports showing that candidates are often eliminated based on DEI statements alone.

91. For example, a self-survey conducted by UC Santa Cruz's sister school UC Berkeley found that, in one particular faculty search, 76% of applicants were eliminated solely on the basis of their diversity statements.[5]

92. This is consistent with UC Santa Cruz's approach to using DEI Statements as part of the initial screening process—the University seeks to eliminate candidates with DEI Statements below certain benchmarks without having to consider the application in more detail.

93. The University's public statements—on its Department of Psychology webpage, on the various hiring resources pages through the APO website, and through its DEI Statement scoring rubric—make it clear that Dr. Haltigan's application would have ended up discarded without consideration of his qualifications entirely on the basis of his views as expressed in the DEI Statement.

94. A cursory examination of the DEI rubric and other materials used by the University shows that Dr. Haltigan's views on DEI would have led to the dismissal of his application without due consideration. Dr. Haltigan's statements on colorblind meritocracy, his commitment to viewpoint diversity, his skepticism of scholars like Ibram Kendi, his intention to treat all scholars the same regardless of identity-based characteristics, and his rejection of the concepts of intersectionality and "anti-racism",

[5] *See, e.g.*, Greg Lukianoff and Rikii Schlott, *Universities Use DEI Statements To Enforce Groupthink*, Reason Magazine (Jan. 6, 2024) (discussing UC self-surveys and other evidence on the importance of DEI Statements and their value in enforcing viewpoint uniformity).

just to start, all are statements that run directly contrary to the materials posted by the University for applicants.

95. It is unlikely that the DEI Statement posted on Dr. Haltigan's Substack would receive better than a 1–2 on any category of the DEI Rubric. Many of the statements Dr. Haltigan makes in his DEI Statement are expressly rejected or treated scornfully in the materials linked on UC Santa Cruz's Academic Personnel Office website.

96. His views on these issues are all protected by the First Amendment and have no bearing on his ability to perform the job in question.

97. If Dr. Haltigan were to have applied for this position, or any others at UC Santa Cruz, he would be compelled to alter his behavior and either remain silent about the many important social issues addressed by the DEI Statement Requirement or recant his views to conform to the dictates of the University administration.

98. The University's policy is that those with views like Dr. Haltigan on issues related to DEI will not be considered beyond an initial screening. This is unconstitutional.

First Claim for Relief:

Violation of the First Amendment of the United States Constitution

Unconstitutional Conditions

99. Plaintiff hereby realleges and incorporates by reference the allegations contained in the previous paragraphs.

100. Defendants are acting under the "color of state law" within the meaning of 42 U.S.C. § 1983 in imposing and enforcing a DEI Statement Requirement on all applicants for faculty positions.

101. Defendants are denying a benefit to Plaintiff in a manner that infringes his First Amendment rights.

102. Defendants are requiring Dr. Haltigan to express ideas with which he disagrees in order to be eligible for employment. This is an unconstitutional form of

compelled speech and is unconstitutional even when that requirement is tied to a government benefit to which the speaker is not entitled.

103. The DEI Statement Requirement forces applicants to UC Santa Cruz to express agreement with the University's views on racism and social justice, and ultimately seeks to regulate speech outside the contours of the program.

104. The DEI Statement Requirement unconstitutionally leverages the availability of a position at the University to force applicants to express agreement with the University's ideology.

105. The DEI Statement Requirement places anyone with Dr. Haltigan's views who wants to work at the University of California in an untenable position. One can either file an honest, but doomed, application, or one can lie and recant his or her honest views. Silence and dissent are not options if he or she wants to progress past the initial screening.

106. Because the DEI Statement Requirement requires Dr. Haltigan to affirm particular beliefs that are inherently separate from the qualifications for a position on the University's faculty or the purpose of the University as a whole, it imposes a condition on employment that would be unconstitutional if done outright.

Second Claim for Relief:

Violation of the First Amendment of the United States Constitution

Viewpoint Discrimination

107. Plaintiff hereby realleges and incorporates by reference the allegations contained in the previous paragraphs.

108. Defendants are acting under the "color of state law" within the meaning of 42 U.S.C. § 1983 in imposing and enforcing a DEI Statement Requirement on all applicants for faculty positions.

109. The DEI Statement Requirement represents invidious viewpoint discrimination against any applicant holding views contrary to the detailed ideological standards set out in the DEI rubric and other guidance documents.

110. The purpose of the DEI Statement Requirement is to penalize certain viewpoints and drive those viewpoints from the marketplace of academic hiring.

111. Dr. Haltigan's views on colorblind inclusivity, viewpoint diversity, and merit-based promotion and hiring are all anathema to the University's express requirements in the DEI Statement.

112. The DEI Statement Requirement has no relationship to established professional standards, the University's mission, or the qualifications for a position on the University's faculty.

113. Because the DEI Statement Requirement is not tailored to any compelling interest, it is unconstitutional.

Request for Relief

Plaintiff respectfully requests the following relief:

A. A declaration that the DEI Statement Requirement employed by UC Santa Cruz violates the First Amendment to the United States Constitution;

B. A permanent injunction forbidding UC Santa Cruz and University of California officials from enforcing, or attempting to enforce, the DEI Statement Requirement against Dr. Haltigan;

C. An award of attorneys' fees, costs, and expenses in this action pursuant to 42 U.S.C. § 1988;

D. Such other relief as this Court deems proper.

DATED: February 2, 2024.

Respectfully submitted,

JOSHUA P. THOMPSON
WILSON C. FREEMAN*
JACK E. BROWN*

By /s/ Wilson C. Freeman
WILSON C. FREEMAN*
Attorney for Plaintiff
**pro hac vice*

CERTIFICATE OF SERVICE

I hereby certify that on February 2, 2024, Opposing Counsel received the foregoing Plaintiff's Second Amended Complaint via CM/ECF service.

By /s/ Wilson C. Freeman
WILSON C. FREEMAN*

Attorney for Plaintiff
**pro hac vice*

John D. Haltigan, Ph.D.

Home: ([redacted])[redacted]
[redacted]
[redacted]
[redacted]

Citizenship: United States; Canadian Permanent Residency

ACADEMIC & SCIENTIFIC APPOINTMENTS:

2016 – 2023	Assistant Professor, University of Toronto, Department of Psychiatry Graduate Faculty, University of Toronto Graduate Department of Applied Psychology and Human Development
2016 – 2023	Independent Scientist and Cundill Scholar The Centre for Addiction and Mental Health
2013 – 2016	Postdoctoral Research Fellow, University of Ottawa, Faculty of Education and School of Psychology
2011 – 2013	Postdoctoral Fellow, University of North Carolina at Greensboro, Department of Human Development and Family Studies
2010 – 2011	Postdoctoral Research Associate, University of Illinois at Urbana-Champaign, Department of Psychology

EDUCATION:

2004 – 2009	M.S., Ph.D., Developmental Psychology University of Miami, Coral Gables, FL Dissertation: *Emotional Communication and Attachment Security in Infants at Risk for Autism Spectrum Disorders*.
2001	M.A., Forensic Psychology Vermont State University at Castleton, Castleton, VT Thesis: *Attachment, Fantasy, and Antisocial Behavior: Implications for a Developmental Trajectory Model*.
1999	B.A., Psychology and Criminal Justice Mercyhurst University, Erie, PA (Summa Cum Laude, Departmental Honors)

RESEARCH INTERESTS:
The legacy of early caregiving experiences for child and adolescent development
Brain and bodily systems that mediate the effects of early social experiences on development
Evolutionary Developmental Psychopathology; life history
Measurement and classification of child and adolescent psychopathology; psychiatric nosology
Longitudinal data modeling; Generalizability Theory; mixed modeling; Item Response Theory

AWARDS & HONORS:

2015	Autism Research Training Program Awardee Canadian Institutes of Health Research (CIHR) *Measurement Science and Longitudinal Data Modeling in Autism Research*. University of Ottawa
1999	Social and Behavioral Sciences Academic Excellence Award. Mercyhurst University
1997 - 1998	Undergraduate Honor Societies: Kappa Gamma Pi, Psi Chi, Alpha Phi Sigma. Mercyhurst University

TEACHING INTERESTS:

Developmental Psychology; Developmental Psychopathology; Evolutionary Psychology; Introduction to Psychobiology; Introductory Statistics; Structural Equation Modeling (SEM); Hierarchical Data Modeling

RESEARCH SUPPPORT:

ACTIVE

Centre for Addiction and Mental Health Discovery Fund
The Toronto Adolescent and Youth (TAY) Cohort Study (https://www.taycohort.ca/)
PI: Aristotle Voineskos
Role: Co-Principle Investigator
5/2021 – Current

Canadian Institutes of Health Research SPOR Operating Grant $3,000,000
Enhancing Evidence-Based Practice for Youth and Emerging Adults with Early Psychosis: Implementation and Evaluation in Diverse Service Settings
(Nominated PI: Aristotle Voineskos)
Role: Co-Investigator
4/2018 – 3/2022

National Institute of Mental Health (NIMH; 1R01 MH114879-01A1) $1,120,131
Social Processes Initiative in Neurobiology of Autism-Spectrum and Schizophrenia-Spectrum Disorders (SPIN-ASD)
(PI: Stephanie Ameis)
Role: Co-Investigator
5/2018 – 5/2022

Social Sciences and Humanities Research Council (SSHRC) $199,351
Validation and Dissemination of an Observed Screening Tool of Maladaptive Parenting for use in Community Agencies Working with Vulnerable Children and Families
(PI: Sheri Madigan)
Role: Co-Investigator
3/2017 – 3/2020

Miner's Lamp Innovation Fund in Prevention and Early Detection of Severe Mental Illness.
University of Toronto, Department of Psychiatry. $150,000
Early Identification of Psychosis Spectrum Symptoms: A Novel Ascertainment Approach Through Tertiary Care Child and Youth Clinics
(PIs: Aristotle Voineskos, Joanna Henderson)
Role: Co-Investigator
4/2016 – 3/2020

Canadian Institutes of Health Research (CIHR) Project Grant $749,211
The Enduring Reach of Childhood Bullying: Longitudinal Links to Adult Mental Health, Academic Achievement, and Functional Outcomes
(PI: Tracy Vaillancourt)
Role: Co-Investigator
9/2016 – 8/2021

Social Sciences and Humanities Research Council (SSHRC) $286,650
The Long Reach of Childhood Bullying: Longitudinal Links to Academic Achievement and Adult Functional Outcomes
(PI: Tracy Vaillancourt)
Role: Collaborator
3/2016 – 3/2021

FUNDING AS A TRAINEE

Early Career Travel Award, Society for Research in Child Development (SRCD).
University of North Carolina at Greensboro (2013)

Fred C. and Helen Donn Flipse Dissertation Research Award
Provost Travel Award
Max and Peggy Kriloff Graduate Student Travel Scholarship
University of Miami (2009)

National Institute of Health: Institutional Training Grant, NICHD T32-HD007473-10
Training in Mental Retardation & Developmental Disabilities
University of Miami (2004-2009)

PUBLICATIONS

A. Peer Reviewed Journal Articles (bold font denotes research mentee).

1. Haltigan, J. D. (2023). Introduction to special section. The influence of COVID-19 pandemic policy on child and adolescent mental health: Strong signal or mostly noise? *Journal of the Canadian Academy of Child and Adolescent Psychiatry, 32,* 69-70.

2. Tiego, J., Martin, E., DeYoung, C.G., Hagan, K., Cooper, S. E.,…and members of the HiTOP Neurobiological Foundations Workgroup (in press). Precision behavioral phenotyping as a strategy for uncovering the biological correlates of psychopathology. *Nature Mental Health*. Preprint available: https://osf.io/geh6q/

3. Aiken, M., Perquier, F., Haltigan, J. D., Wang, L., Andrade, B. F., Battaglia, M., Szatmari, P., & Georgiades, K. (2023). Broad and specific: Associations between child psychopathology and parenting at the individual and family level. *Development & Psychopathology.*

4. Haltigan, J. D., Pringsheim, T., & **Rajkumar, G.** (2023). Social media as an incubator of personality & behavioral Psychopathology: Symptom and disorder authenticity or psychosomatic social contagion? *Comprehensive Psychiatry, 121,* 152362.

5. Del Giudice, M., & Haltigan, J. D. (2023). A new look at the relations between attachment and intelligence. *Developmental Review, 67,* 101054.

6. Waldman, I., King, C. D., Poore, H. E.,…Haltigan, J.D.,…and contributing members of HiTOP Quantitative Methods Workgroup. (2023). Best practices for adjudicating among alternative structural models of psychopathology. *Clinical Psychological Science, 11,* 616-640. https://doi.org/10.1177/21677026221144256

7. Laskar, J., Haltigan, J. D., & Richardson, G. (2021). Measurement Issues in Tests of the Socioecological Complexity Hypothesis. *Evolutionary Psychological Science*. doi: https://doi.org/10.1007/s40806-021-00301-0.

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B. Manuscripts Under Review and in Preparation (bold font denotes research mentee).

67. Michelini, G., Carlisi, C. O., Eaton, N. R., Haltigan, J. D., Krueger, R. F., Kotov, R.,…et al. (under review). Where are neurodevelopmental symptoms in transdiagnostic frameworks of psychiatric conditions? Incorporating a new neurodevelopmental spectrum.

68. Stewart, L. C., Asadi, S., Rodriguez-Seijas, C., Wilson, S., Kotov, R., Cicero, D., Haltigan, J. D. & Olino., T. M. (under review). Measurement invariance of the child behavior checklist (CBCL) across race/ethnicity and sex in the ABCD Study.

69. Haltigan, J. D., Duriseti, R., & Colyvas, K. (in prep). Generalizability Theory and the COVID-19 mask dialectic: Why mask mandates lack empirical justification.

70. Haltigan, J. D., **Gheorghiu, D.**, and members of the HiTOP consortium (in prep). Conceptual and empirical review of the multidimensional structure of the Obsessive-Compulsive Disorder (OCD) clinical phenotype: Implications for an OCD spectrum.

71. DeYoung, C., Latzman, R. D., Grazioplene, Haltigan, J. D…et al. (revised & resubmitted). The Hierarchical Taxonomy of Psychopathology (HiTOP) and the search for neurobiological substrates of mental illness. *The American Journal of Psychiatry*.

72. Haltigan, J.D., **Bi, M., Proulx, S., Anjali, & Gheorghiu, D.** (in prep). Differences in Sensitivity to Environmental Context Depending on Parasympathetic Vagal Functioning: Conceptual Review and Meta-Analysis.

73. Jacobs, G. A., Haltigan, J. D., Ameis, S. A., & Voineskos, A. V. (under review). Bifactor models of psychopathology using multi-informant and multi-instrument dimensional measures in a population-based sample of children from the ABCD study.

74. Haltigan, J. D., Del Giudice, M., et al. (in prep). Identifying functional subtypes within ASD, ADHD, and OCD diagnostic categories: A preliminary evaluation of the FSD life history model of psychopathology.

75. Haltigan, J. D., **Khorsand, S.**, Mills-Koonce, R., and Lyons-Ruth, K. (in prep). Infant attachment behavior in maternal context: A dyadic mixture modeling approach to the patterning of attachment behavior.

C. Book Chapter.

1. Messinger, D.S., Mahoor, M.H., Chow, S-M., Haltigan, J.D., Cadavid, S., & Cohn, J.F. (2010). Early emotional communication: Novel approaches to interaction. In J. Gratch & S. Marsella (Eds.), *Social emotions in nature and artifact: Emotions in human and human-computer interaction (Vol. 14)*. New York: Oxford University Press.

D. Training Manuals.

1. Madigan, S., Bronfman, E., Haltigan, J. D., & Lyons-Ruth, K. (2018). The Atypical Behavior Instrument for Assessment and Classification - Brief (AMBIANCE-Brief) University of Calgary, Calgary Alberta Canada.

CONFERENCE PRESENTATIONS (bold font denotes mentee).

1. Haltigan, J. D. (2021). Symposium discussant. Attaining greater insight into determinants of parent-child attachment: One (conceptual) model does not fit all. Paper symposium to be presented at the 2021 virtual biennial conference of the Society for Research in Child Development (SRCD).

2. Haltigan, J. D., Henderson, J., Ameis, S. H., Skilling, T., Andrade, B., Cleverley, K., Courtney, D. B., Foussias, G., Szatmari, P., & Voineskos, A. N. Associations of childhood adversity with psychiatric symptoms in adolescence: Towards a fully dimensional framework. In N. Kozloff (chair), Thinking Outside the Box: Broadening Perspectives to Improve Research, Diagnosis and Treatment of Youth with Mental Illness. Paper to be presented at the American Academy of Child & Adolescent Psychiatry 66th Annual Meeting, Chicago, October 14-19, 2019.

3. Haltigan, J. D., **Khorsand, S.**, & Lyons-Ruth, K. L. R. (2019). Patterning of maternal and infant attachment behavior among unclassifiable infants: An exploratory investigation. Paper presented at the Society for Emotion and Attachment Studies (SEAS) preconference at the 2019 Society for Research in Child Development (SRCD) biennial meeting. Baltimore, MD.

4. Madigan, S. M., Haltigan, J. D., Cooke, J., Eirich, R., Bronfman, E., Racine, N., Bailey, H., Borland-Kerr, C., Plamondon, A., Tarabulsy, G., Cyr, C., Mills-Koonce, R., & Lyons-Ruth, K. (2018). Validation of the AMBIANCE parent-child observational assessment tool for use in applied settings with families at high social risk. In S. Madigan (chair), Bridging the knowledge to practice gap: Implementing and validating attachment measures for use in community agencies. Paper presented at the 16th World Association for Infant Mental Health (WAIMH) World Congress, Rome, Italy.

5. Martin, K.B., Prince, E.B., Haltigan, J.D., Ekas, N., & Messinger, D.S. Insecure-resistant attachment classification (and behaviors) in infants later diagnosed with autism spectrum disorder. Poster presented at the 2017 International Meeting for Autism Research, San Francisco, California, USA.

6. Haltigan, J. D., Bronfman, E., Madigan, S., Bailey, H., Kerr, C., & Lyons-Ruth, K. (2017). Refining the assessment of disrupted maternal communication: Using item response models to identify central maternal behaviors. Individual poster presentation presented at the 2017 Society for Research in Child Development (SRCD) biennial meeting. Austin, TX.

7. Haltigan, J. D., & Vaillancourt, T. (2017). The influence of intrapersonal factors on peer victimization through emerging adulthood: A latent transition analysis. In M. Brendgen (chair), Moderating factors of the link between childhood peer victimization and adjustment in young adulthood. Paper presented at the 2017 Society for Research in Child Development (SRCD) biennial meeting. Austin, TX.

8. Martin, K., Haltigan, J. D., Ekas, N., Prince, E., & Messinger, D. M. (2017). Attachment security differs by autism spectrum disorder diagnosis: A prospective study. Poster presented at the 2017 Society for Research in Child Development (SRCD) biennial meeting. Austin, TX.

9. Vaillancourt, T. V., & Haltigan, J. D. (2016). Joint trajectories of depression and perfectionism across adolescence and childhood risk factors. Paper presented at the 2016 International Association for Child and Adolescent Psychiatry and Allied Professions World Congress and Canadian Academy of Child and Adolescent Psychiatry (IACAPAP), Calgary, Alberta, Canada.

10. Haltigan, J. D., & Vaillancourt, T. V. (2015). Identifying trajectories of borderline personality features in early adolescence: Antecedent and interactive risk factors. Poster presented at The Canadian Psychological Association's 76th Annual Convention. Ottawa, Ontario, Canada.

11. Haltigan, J. D., & Vaillancourt, T. V. (2015). Peer victimization: Subtypes or severity? In T. Daniels (chair), Longitudinal approaches to examining peer victimization: What are the risk and protective factors and where do we go from here? Paper presented at the 2015 Society for Research in Child Development (SRCD) biennial meeting. Philadelphia, PA.

12. Martin, K., Zambrana, K., Haltigan, J. D., Rongfang, J., Beebe, B., Messinger, D. M. (2015). More is not always better: Gaze patterns and later attachment security. Poster presented at the 2015 Society for Research in Child Development (SRCD) biennial meeting. Philadelphia, PA.

13. Haltigan, J. D., & Vaillancourt, T. V. (2014). Childhood developmental and mental health antecedents of trajectories of borderline personality disorder. New Research Poster selected for presentation at the American Academy of Child and Adolescent Psychiatry's 61st Annual Meeting, San Diego, CA.

14. Haltigan, J. D., & Vaillancourt, T. V. (2014). Developmental trajectories of bullying and peer victimization across elementary and middle school and associations with symptoms of psychopathology. In G. Steffgen (chair), Bullying and aggression. Paper presented at the 21st World Meeting of the International Society for Research on Aggression, Atlanta, GA.

15. Wang, W., Brittain, H. L., Haltigan, J. D., & Vaillancourt, T. (2014). The influence of school transition experiences on peer victimization and bullying perpetration. Poster presented at the 21st World Meeting of the International Society for Research on Aggression, Atlanta, GA.

16. Wang, W., Vaillancourt, T., Brittain, H. L., McDougall, P., Krygsman, A., Smith, D., Cunningham, C. E., Haltigan, J. D., & Hymel, S. (2014). School climate, peer victimization, and academic achievement: Results from a multi-informant study. In W. Wang and T. Vaillancourt (chairs), School climate and bullying. Paper presented at the 21st World Meeting of the International Society for Research on Aggression, Atlanta, GA.

17. Leerkes, E. M., Haltigan, J. D., Wong, M. S., Fortuna, K. S., O'Brien, M., Calkins, S., & Supple, A. (2013). A psychobiological model of the origins of maternal sensitivity to distress. In J. Mesman (chair), Maternal sensitivity: New insights on its antecedents, outcomes, and cross-cultural relevance. Paper presented to the Society for Research in Child Development (SRCD) Biennial Meeting, Seattle, WA.

18. Haltigan, J. D., Leerkes, E. M., et al. (2013). The latent structure of adult attachment: Confirmatory replication, measurement invariance, and relations to maternal sensitivity. In J. D. Haltigan (chair), Associations between parental representations of past attachment experiences and caregiver sensitivity: New analytic approaches and new insights. Paper presented to the Society for Research in Child Development (SRCD) Biennial Meeting, Seattle, WA.

19. Haltigan, J. D., Leerkes, E. M., et al. (2013). The three-factor structure of adult attachment: Measurement invariance and evidence for ethnic differences in passive, loss-related phenomenon. Poster presented to the Society for Research in Child Development (SRCD) Biennial Meeting, Seattle, WA.

20. Haltigan, J. D., & Leerkes, E. M. (2012). Self-reports of adult attachment and mothers' cognitive and emotional responses to distress. In J. D. Haltigan (chair), The multiple determinants of parenting revisited: Adult attachment, the Big 5, and child temperament. Paper presented to the International Society on Infant Studies (ISIS) Biennial Meeting, Minneapolis, MN.

21. Ekas, N. V., Haltigan, J. D., & Messinger, D. M. (2012). The dynamic still-face effect: Do infants decrease bidding over time when parents are not responsive? In D. Messinger (chair), Early temporal dynamics of self-organization and interaction: New research approaches. Paper presented to the International Society on Infant Studies (ISIS) Biennial Meeting, Minneapolis, MN.

22. Leerkes, E. M., Haltigan, J. D., Wong, M., & Fortuna, K. (2012). Pregnant women's physiological, emotional, and cognitive responses to infant crying vary based on their adult attachment status. In A. M. Groh (chair), Mediators and moderators of maternal sensitivity: The role of physiological, neurobiological, and behavioral responding to distress. Paper presented to the International Society on Infant Studies (ISIS) Biennial Meeting, Minneapolis, MN.

23. Behrens, K., Parker, A. C., & Haltigan, J. D. (2012). Maternal behavior during the Strange Situation Procedure predicts children's reunion behaviors and quality of attachment. Poster presented to the International Society on Infant Studies (ISIS) Biennial Meeting, Minneapolis, MN.

24. Haltigan, J. D., Roisman, G. I., & Fraley, R. C. (2011). The predictive significance of early caregiving experiences for externalizing and internalizing symptomatology through age 15. Poster presented to the Society for Research in Child Development (SRCD) Biennial Meeting, Montreal, Quebec, Canada.

25. Haltigan, J. D., Roisman, G. I., Susman, E. J., Barnett-Walker, K., Monahan, K., & The National Institute of Child Health and Human Development Early Child Care Research Network. (2011). Elevated trajectories of externalizing problems are associated with lower awakening cortisol levels in mid-adolescence. Poster presented to the Society for Research in Child Development (SRCD) Biennial Meeting, Montreal, Quebec, Canada.

26. Haltigan, J. D., Roisman, G. I., & Fraley, R. C. (2011). The predictive significance of early caregiving experiences for symptoms of psychopathology through mid-adolescence: Enduring or transient effects. Poster presented to the inaugural University of Illinois at Urbana-Champaign postdoctoral research symposium at the Beckman Institute.

27. Belt, R. V., Kouvo, A., Flykt, M., Punamäki, R., Haltigan, J. D., & Tamminen, T. (2010). Does an early intervention prevent disturbance in mother-infant relationship after maternal experiences of traumatic loss? Integrating the AAI, Strange Situation, and Emotional Availability methods in a case of mother-infant psychotherapy. Poster presented to the 12th World Congress of the World Association for Infant Mental Health, Leipzig, Germany.

28. Ekas, N. V., Haltigan, J. D., Gealy, W., & Messinger, D. (2009). Early interaction between infants at-risk for autism spectrum disorder and their mothers. Poster presented to the International Conference for Infant Studies (ICIS), Baltimore, MD.

29. Baker, J. K., Haltigan, J. D., Brewster, R., Jaccard, J., & Messinger, D. (2009). Non-expert ratings of parent and infant emotion: Concordance with expert ratings and relevance to early autism risk. Hot Topic paper presented to the International Society for Research in Emotions (ISRE), Leuven, Belgium.

30. Messinger, D. S., Mahoor, M., Cadavid, S., Kimijima, M., Haltigan, J.D., & Cohn, J. (2009). The role of eye constriction in positive and negative infant emotional expressions. Hot Topic paper presented to the International Society for Research in Emotion (ISRE), Leuven, Belgium.

31. Haltigan, J. D., Seifer, R., Chan, S., Gealy, W., & Messinger, D. (2009). Parental sensitivity and attachment security in infants at risk for autism spectrum disorders (ASDs). Poster presented to the Society for Research in Child Development (SRCD) Biennial Meeting, Denver, CO.

32. Kelley, K. M., Ibanez, L., Haltigan, J. D., Acosta, S., McDonald, N., Grantz, C., Brewster, R., & Messinger, D. (2008). Sibling studies measuring infant learning and emotion: A longitudinal study of infants at-risk for autism spectrum disorder. Poster presented to the annual meeting of the Marino Autism Research Institute (MARI), Nashville, TN.

33. Mc Donald, N., Haltigan, J.D., Kelley, K., & Messinger, D. (2008). Empathic responding and attachment security in young children at-risk for an autism spectrum disorder (ASD). Poster presented to the International Meeting for Autism Research (IMFAR), London, England.

34. Messinger, D., Cassel, T., Ibanez, L. Haltigan, J. D., Acosta, S. & Kelley, K. (2008). Emotion, attention, and joint attention in infants at-risk for autism. Paper presented to the International Meeting for Autism Research (IMFAR), London, England.

35. Messinger, D., Cassel, T., Ibanez, L., Haltigan, J. D., Acosta, S. & Kelley, K. (2008) Early attention shifting and joint attention deficits in infants at-risk for autism. Poster presented to the International Society for Infant Studies (ISIS), Vancouver, British Columbia, Canada.

36. Haltigan, J.D., Messinger, D.S., Chow, Sy-Miin, Jaccard, J., and Wang, T. (2007). Continuous emotion ratings of infants at risk for autism and their parents. Poster presented to the Association for Psychological Science (APS), Washington, D.C.

37. Haltigan, J. D., Messinger, D. S., Sy-Miin, C., Jaccard, J., and Wang, T. (2007). Continuous measurement of infant and parent emotional valence in the face-to-face still-face paradigm: Infants at-risk for autism. Poster presented to the International Meeting for Autism Research (IMFAR), Seattle, WA.

38. Haltigan, J. D., Messinger, D. S., Chow, Sy-Miin, & and Jaccard, J. (2007). Exploring interactive infant-mother communication using continuous rating software (CRS). Poster presented to the Society for Research in Child Development (SRCD), Boston, MA.

39. Messinger, D. S., Cassell, T., Ibanez, L., Haltigan, J. D., and Acosta, S. (2007). Early social and emotional communication in infant siblings of children with autism spectrum disorders. Paper presented to the Society for Research in Child Development (SRCD), Boston, MA.

40. Messinger, D., Cassel, T. D., Ibanez, L. V., Haltigan, J. D., Acosta, S., and Buchman, A. (2006). Early social and emotional communication in the infant siblings of children with autism spectrum disorder: An examination of the broad phenotype. Poster presented to the International Conference on Infant Studies (ICIS), Kyoto, Japan.

41. Haltigan, J. D., Messinger, D., Chow, S., Linick, J., Wang, T., & Jaccard, J. (2006). Time-based measurement of interaction emotion using non-expert observers. Poster presented to the International Society for Research on Emotions (ISRE), Atlanta, GA.

42. Haltigan, J. D., Messinger, D., Chow, S., Linick, J., Wang, T., & Jaccard, J. (2006). Emotion ratings from continuous rating software: Reliability, validity, and applicability. Poster presented to the Association for Psychological Science (APS), New York, NY.

43. Cassel, T., Messinger, D., Acosta, S., Haltigan, J. D., & Linick, J. (2005). Emotional deficits in infants with siblings with autism spectrum disorders. Poster presented to the American Psychological Association (APA), Washington, D.C.

44. Messinger, D. S., Haltigan, J. D., Hu, Changbo, Venezia, M., Hamilton, L., Cohn, J. F. (2005). Interactive influence in mutual smiling. Paper presented to the Society for Research in Child Development (SRCD), Atlanta, GA.

45. Messinger, D. S., Chow, S. M., Koterba, S.Hu, C., Haltigan, J. D., Wang, T., & Cohn, J. F. (2005). Smile and emotion dynamics in infant-mother interaction. Paper presented to the International Society for Research in Emotion (ISRE), Bari, Italy.

46. Schober, J., Lipman, R., Haltigan, J. D., & Kuhn, P. J. (2003). The impact of monosymptomatic enuresis on attachment parameters. Paper presented to the European Society of Pediatric Urology, Madrid, Spain.

EDITORIAL ACTIVITIES:

Attachment and Human Development: International Advisory Board (1/2014-current)
Journal of the Canadian Academy of Child and Adolescent Psychiatry: Associate Editor

AD HOC REVIEWS (SELECTED):

Aggressive Behavior, Assessment, Attachment and Human Development, Autism, Biological Psychiatry, British Journal of Developmental Psychology, Canadian Journal of Behavioural Science, Child Development, Child Development Perspectives, Clinical Child Psychology & Psychiatry, Clinical Psychological Science, Criminal Justice and Behavior, Developmental Psychology, Development and Psychopathology, Developmental Science, Evolutionary Behavioral Sciences, Journal of Autism and Developmental Disorders, Journal of Child Psychology and Psychiatry, Infancy, Infant Behavior & Development, Infant Mental Health Journal, Molecular Autism, OpenPsych, Personal Relationships, Psychological Bulletin, Psychoneuroendocrinology, Research in Autism Spectrum Disorders, SAGE Open, Society for Research in Child Development Biennial Meeting, The Journal of the American Academy of Child and Adolescent Psychiatry, The Canadian Journal of Psychiatry

TEACHING:

University of Ottawa

2016	Instructor: PSY 3102C: Interpersonal Relationships Designed and lead all classes Student Evaluation of Teaching (range 1-5): *Mean* = 4.1, *SD* = .3
2014	Instructor: PSY 3173A: Forensic Psychology Designed and led all classes Student Evaluation of Teaching (range 1-5): *Mean* = 4.2, *SD* = .3

University of Miami

2006 Instructor: PSY 202K: Introduction to Psychobiology (Fall)
Designed and led all classes
Student Evaluation of Teaching (range 1-5): *Mean* = 4.5, *SD* = .2

Vermont State University at Castleton

2001 Teaching Assistant: PSY 208: *Abnormal Psychology* (Spring)
Graded all written assignments and exams

Teaching Assistant: PSY 305: *Child Psychopathology* (Spring)
Graded all written assignments and exams

2000 Instructor: MAT 117: *Introduction to Computers* (Spring)
Designed and led all classes

Co-Instructor: PSY 1010: *Introduction to Psychology* (Fall)
Designed and led classes; graded written assignments and exams

MENTORSHIP:

University of Toronto/Centre for Addiction and Mental Health

2018-2022 Soha Khorsand, B.MSc., University of Western Ontario
Niloofar Izadivahedi, Psychiatry Resident, University of Toronto
Dan Gheorghiu, B.Sc. Hons. Psychology, M.Sc. Biology, York University
Spencer Proulx, B.MSc., University of Western Ontario
Michelle Bi, B.Sc., University of Western Ontario
Anjali, B. A. Psychology, University of British Columbia, Vancouver
Gayathiri Rajkumar, Medical Sciences Stream, Honors Specialization in Biology, University of Western Ontario
Lauren Saurette, George Brown College; Ryerson University, Disability Studies
Ali Dhalla, B.Sc., University of Western Ontario
Mahima Tirunelveli Santhakumar, B.Sc Hons. (Psych). University of Toronto
Sanjana Raja Rao Nagaraj, B.Sc Hons. Toronto Metropolitan University
Ana Paula Mendes Silva, Ph.D., Post-doctoral fellow, CAMH (CAMH Research Training Mentorship Program)
Ayesha Rashidi, MSc., University of Toronto (Ph.D. advisory committee member)
Connor Burke, B.S., Northeastern University

University of Illinois at Urbana-Champaign

2010-2011 Supervisor: PSYCH 290: Adult Attachment Transcription Team. Coordinated large team of undergraduate assistants; facilitated weekly seminar on relevant literature and research scholarship. (Fall and Spring)

University of Miami

2007 Supervised Psychology Honors Student:
"Continuous Measurement of Perceived Emotion in Infants At-Risk for Autism."
Stephanie Beckel

2007 Co-supervised Psychology Honors Student:
"Temperament in the Infant Siblings of Children with an Autism Spectrum Disorder"
Cristina Fernandez

2006 Supervised Psychology Honors Student:
"*Continuous Ratings of Infant and Parent Emotional Valence"*
Jessica Linick

2005 Supervised Psychology Honors Student:
"Computer Vision and Undergraduate Ratings of Infant and Mother Smiles."
Jessica Gaby

LECTURES AND INVITED TALKS:

University of Toronto, Department of Psychiatry

2021-2023 *Attachment Theory in Psychiatry and Medicine*
PGY1 Core Lecture Series

Centre for Addiction and Mental Health and The Hospital for Sick Children

2021-2022 *Differences in Sensitivity to Environmental Quality Depending on Parasympathetic Vagal Functioning: Conceptual Review and Meta-Analysis*
CAMH Research Trainee Seminar Series

2019 *A Common Quantitative Framework for Child & Adolescent Mental Illness.*
University of Toronto, Department of Psychiatry, Harvey Stancer Research Day

2019 *The Promise of Empirical Assessment of Psychopathology and Mental Illness.*
CAMH Child and Youth Grand Rounds

2018 *The General Psychopathology Factor: Identification and Significance*.
McCain Centre Rounds

2016 *Refining the Assessment of Disrupted Maternal Communication: Using Item Response Models to Identify Central Maternal Behaviors.*
McCain Centre Rounds

University of North Carolina at Greensboro

2011 *Heterogeneity in Infant Attachment Disorganization: In Search of Developmental Antecedents and Correlates.*
Graduate Colloquium Series (Fall)

University of Illinois at Urbana-Champaign

2011 *Early Experience, Intraindividual Risk, and Developmental Psychopathology.* Brown-Bag Colloquium (Spring)

MEDIA EXPOSURE

Social media as an incubator of personality & behavioral Psychopathology: Symptom and disorder authenticity or psychosomatic social contagion?
-March 2, 2023
The Ben Domenech Podcast (FOX News Radio), interviewed by Ben Domenech regarding the above-titled published manuscript in *Comprehensive Psychiatry* and my work in child & adolescent mental health.
-January 23, 2023
Newsmax, appeared on Rob Schmitt Tonight to discuss published manuscript in *Comprehensive Psychiatry*
-January 19, 2023
Published manuscript in *Comprehensive Psychiatry* was covered by The Daily Wire
-January 8, 2023
Published manuscript in *Comprehensive Psychiatry* was covered by The Guardian, Australia.
https://www.theguardian.com/australia-news/2023/jan/09/urgent-need-to-understand-link-between-teens-self-diagnosing-disorders-and-social-media-use-experts-say
December 29, 2022
-Nautilus Magazine, interviewed by Brian Gallagher regarding the above-titled published manuscript in *Comprehensive Psychiatry* and my work in child & adolescent mental health.
Right Ideas: Philip Rieff
-December 12, 2022
The National Association of Scholars (NAS), participated as a panelist in this live-streamed webinar discussing the work of sociologist and cultural critic Philip Rieff and his relevance to contemporary conservative thought.

ADDITIONAL QUANTITATIVE AND METHODOLOGICAL TRAINING:

Carolina Consortium on Human Development (2012)
Causal Inference in Developmental Science Proseminar
Chair: Patrick Curran, Ph.D.
University of North Carolina at Chapel Hill

Adult Attachment Interview Coding Training (2008)
Instructors: June Sroufe, Ph.D., & Sonia Gojman de Millan, Ph.D. (Certified, 2009)
University of Minnesota, Institute of Child Development

Introduction to Hierarchical Linear Modeling (2007)
Instructor: Nicholas D. Myers, Ph.D.
University of Miami

Structural Equation Modeling (2006)
Ancillary Coursework: Generalizability Theory & Measurement Reliability
Instructor: Maria Llabre, Ph.D.
University of Miami

Facial Action Coding System (2004)
Instructor: Erika Rosenberg, Ph.D. (Certified, 2005)
University of California, San Francisco

Early Social and Communication Scales Coding Training (2005)
Instructor: Peter Mundy, Ph.D. (Certified, 2005)
University of Miami

Strange Situation Infant Attachment Paradigm (2004)
Instructors: L. Alan Sroufe Ph.D. and E. B. Carlson Ph.D. (Certified ABC, 2004)
University of Minnesota, Institute of Child Development

CLINICAL RESEARCH TRAINING:

University of Miami

Infant-Sibs Project (2004-2009)
Trained in and conducted developmental and cognitive assessments, including the Mullen Scales of Early Learning, Bayley Scales of Infant and Toddler Development, and Wechsler Scales of Intelligence (WISC-IV, WPPSI-III)
Supervisor: Dr. Kara Kelley, Psy. D.

MEMBERSHIP HISTORY IN SCIENTIFIC AND PROFESSIONAL ORGANIZATIONS/CONSORTIUMS:

American Psychological Association (APA)
Association for Psychological Science (APS)
Canadian Psychological Association (CPA)
Heterodox Academy (Academic Member)
International Society for Psychoneuroendocrinology (ISPNE)
International Society for Research on Aggression (ISRA)
Society for Open Inquiry in Behavioral Science (SOIBS)
Society for Research in Child Development (SRCD)
Society for Research in Psychopathology (SRP)
The Hierarchical Taxonomy of Psychopathology (HiTOP) Consortium

UNIVERSITY SERVICE AND VOLUNTEER ACTIVITIES:

Centre for Addiction and Mental Health

2019	Member: Psychosis Early Intervention and Prevention spectrum (PEIPs) Working Group
2018-Present	Member: YouthCan Impact Study Data and Safety Monitoring Board

University of Toronto

2022	Mentor, CAMH Research Training Mentorship Program
2021	Invited Panelist, University of Toronto, Youth Support Network (YSN), Youth Mental Disorder Presentation Series
2019	Facilitator: Global Medical Student Partnership (GMSP) Child Health Discussion Session, University of Toronto Faculty of Medicine.
2018-Present	Ad-hoc research advisor, Department of Psychiatry
2018	Reserve member, Admissions and Evaluation Subcommittee, Child and Adolescent Psychiatry (CAP) Subspecialty Program, Department of Psychiatry

University of Miami

2021	Graduate alumni panel member, Psychology Career Panel
2008	Graduate student committee, Developmental Faculty Search Team
2006	Counselor, Mini Canes recreational summer camp

Research Statement

Overview

My research program investigates the structure, determinants, course, and co-occurrence of child and adolescent mental and physical illness from a developmental psychopathology perspective. I have a core substantive interest in the legacy of early caregiving and social experiences and the mechanisms and processes that bring them to bear on child and adolescent functioning and health. My research is informed by a life history, evolutionary perspective, and cuts across developmental, evolutionary, and personality psychology, as well as epidemiology and psychiatric medicine. This work draws on both primary data collection efforts and secondary analyses of large, multi-site longitudinal investigations. A distinctive feature of my work is the novel and creative use of measurement science and longitudinal methods to address classic and emerging questions in human development and psychopathology (e.g., Haltigan & DelGiudice, 2021; Haltigan, Aitken et al., 2018; Haltigan & Vaillancourt, 2016; Haltigan, Leerkes, et al., 2014) and to improve early identification of incipient psychopathology. I seek to apply cutting-edge variable and person-centered quantitative approaches that enable more precise and rigorous tests of developmental hypotheses in social science and health research (Haltigan, Roisman, & Fraley, 2013). In my work, I strive to utilize multi-informant, multi-method, and multi-level analytic approaches to address both basic and applied questions in developmental science.

Consistent with the developmental psychopathology principles of equifinality and multifinality, my work is informed by the idea that there are multiple and coactive pathways to competence and maladaptation, and seeks to better understand variation in the onset, course, and correlates of psychopathology (e.g., Haltigan & Vaillancourt, 2014; Haltigan, Roisman, et al., 2011). I have conducted work both in normative and high-risk populations in order to understand the processes that contribute to normal development as well as those that contribute to abnormal development. *Common to each strand of my research described below is work exploring the development of both children at-risk for and those with a clinical diagnosis of Autism Spectrum Disorder (ASD; Haltigan et al., 2011; Vaillancourt, Haltigan, et al., 2017) as the social, emotional, and behavioral deficits in ASD bridge both streams of my work.*

The Measurement, Classification, and Developmental Course of Child and Adolescent Psychopathology

My work investigating the structure and developmental course of child and adolescent psychopathology has been characterized by the identification and validation of distinct trajectories of psychopathology from childhood through adolescence and, more recently, the transdiagnostic empirical structure of psychopathology. By determining when and how children embark upon and exit from pathways toward elevated, chronic, and co-occurring mental health problems, it is possible to contribute to applied science and inform prevention and intervention strategies for children and adolescents at risk for psychopathology. Similarly, quantitative evidence bearing upon the phenotypic behavioral organization of the psychopathology hierarchy is increasingly organizing efforts in understanding individual differences in mental health, and has become an influential paradigm in psychiatric nosology known as "The Quantitative Classification Movement."

Contributing evidence relevant to testing Moffitt's (1993) seminal account of early-onset persistent (EOP) versus adolescence-onset (AO) antisocial trajectories, along with my colleagues, I have shown that lower basal awakening cortisol is reliably associated with higher levels of externalizing behavior across childhood and adolescence (Haltigan, Roisman, Susman et al., 2011). Counter to the prediction based on Moffitt's taxonomy of antisocial behavior, we found that the negative relationship between awakening cortisol and externalizing behavior across time was observed for all groups of individuals with elevated levels of externalizing behavior, regardless of their timing of onset. I have also recently examined questions concerning the developmental course of internalizing symptomatology through early adulthood (Haltigan, Roisman et al., 2017). Findings from this work suggest that prepubertal and postpubertal internalizing symptomatology are unlikely to be distinct developmental phenomena, but rather share biological, family psychosocial, and peer relationship common influences. Importantly, peer relationship influences were found to be the strongest correlate of internalizing symptomatology, regardless of timing of onset.

Using data from a large school-based sample of Canadian youth, along with my colleague, I have presented evidence for the longitudinal and gender measurement invariance of a two-factor model of the Borderline Personality Features Scale for Children (BPFS-C), a self-report measure of borderline personality features in adolescence (Haltigan & Vaillancourt, 2016). A key aspect of this work was the development of a revised BPFS-C factor structure after failing to confirm an originally proposed four-factor model. In step with this work, I also presented evidence for distinct developmental trajectories of borderline personality features across childhood and early adolescence including a low/stable group, an intermediate/stable, and an elevated/rising group (Haltigan & Vaillancourt, 2016). In addition to showing that antecedent mental health symptomatology, intraindividual risk factors, and peer relations were able to distinguish the groups, we found evidence for an interaction between children's self-reported reactive temperament and the experience of peer victimization in predicting borderline personality features trajectory group. This finding suggests the importance of

considering peer relationships when examining the harsh interpersonal experiences component of the diathesis-stress model of borderline personality disturbance.

Along with my colleagues, I (Haltigan, Aitken et al., 2018) have advanced my work on the factor structure of psychopathology by extending evidence for a general factor of psychopathology in clinically referred adolescents using large samples of both parent-report and youth self-report behavioral problem rating data. This work is the first to model the general factor of psychopathology using item-level data from the widely used, gold-standard Achenbach System of Empirically Based Assessment (ASEBA). In addition to demonstrating that general factor scores were robust predictors of self-harm and suicidality, we demonstrated theoretically expectable relations between child sex and age, and lower-order, syndrome-specific, internalizing, externalizing, and thought problem symptom dimensions. In this research, we also demonstrated—consistent with psychopathology and clinical diagnostic data obtained from adults—that the latent structure of the different dimensions of child and adolescent psychopathology as measured by both reporters was best characterized by a continuous distribution of problem behavior symptoms, suggesting that they are better understood as reflecting a continuous liability gradient rather than as composted of discrete classes of psychopathology symptoms.

In a conceptually and methodologically related series of papers, my colleagues and I have examined the dynamic co-occurrence of different forms of child and adolescent psychopathology in both normal and atypical samples. This work has utilized group-based modeling techniques to explore the unfolding of psychopathology across adolescence and has addressed questions relevant to the onset and temporal ordering of co-occurring psychopathology. In the first paper, along with my colleague, I modeled joint trajectories of involvement in bullying and peer victimization across middle school and provided evidence that peer victimization may be more likely to precede increased involvement in bullying rather than vice versa (Haltigan & Vaillancourt, 2014). In the second paper, I collaborated on work (Vaillancourt, Haltigan, et al., 2017) which examined heterogeneity in the developmental co-occurrence of internalizing and externalizing symptomatology in children with ASD. Our findings suggested that elevated mental health symptomatology in children with ASD is associated with similar social determinants as in non-ASD children, and may not necessarily be an epiphenomenon of the underlying neuropsychological atypicalities that characterize the ASD phenotype. Finally, along with my colleague (Vaillancourt & Haltigan, 2018) I examined the joint development of depression and perfectionism across late childhood and adolescence. This work contributed to the debate concerning links between perfectionism and depression and suggested that elevated levels of depressive symptoms may be more likely to precede the development of elevated levels of perfectionism rather than vice versa.

The Legacy of Early Relationship Experiences for Child and Adolescent Development

A primary substantive locus of my research is organized around a fundamental, long-standing question in developmental science—the predictive significance of early relationship experiences for child and adolescent development. This work has leveraged both original data collection efforts and secondary analyses of existing large-sample datasets (e.g., the National Institute of Child Health and Human Development Study of Early Child Care and Youth Development; NICHD SECCYD) and has involved the assessment of parent-child relationships, including those at increased levels of social and neurobiological risk. In keeping with a developmental psychopathology perspective, this work is genetically and physiologically informed and considers the dynamic and coactive influence of culture and biosocial risk in shaping child and adolescent development. To capture the complexity of developmental processes, this work draws on advanced analytic techniques and on the use of a multi-informant approach. This work heavily relies on gold-standard methods in this field, including observational assessments of parent-child interactive behavior (e.g., the Face-to-Face Still/Face Procedure and the Strange Situation Procedure) as well as interview-based measures of adult attachment (e.g., the Adult Attachment Interview).

Formalizing Models of the Predictive Significance of Early Experience

A key question in developmental science concerning the legacy of early experience is whether early caregiver-child experiences exert transient or long-term, enduring effects on social and emotional adaptation. Using existing longitudinal data from the NICHD SECCYD, my colleagues and I addressed this issue (Fraley, Roisman, & Haltigan, 2013; Haltigan, Roisman, & Fraley, 2013). To adequately capture the complexity of the developmental processes under investigation, we developed and implemented a novel modeling approach which examines whether the association between early maternal sensitivity and later adaptation is best accounted for by an enduring account of early interpersonal experiences (in which early experiences continue to exert effects on later adaptation), or a revisionist account of early experiences (which suggests that the effects of early interpersonal experiences on adaptation are only proximal to that point in development and decay to little or no influence over time). Findings from this work suggested that the influence of early interpersonal experiences on later social/academic competence and problem behavior symptomatology is often better explained by an enduring model of the role of early experiences rather than a revisionist perspective. I have recently conceptually

extended this work in an examination of the potential lasting effects of grade 5 peer victimization on grade 10 peer victimization (Haltigan & Vaillancourt, 2017).

Developmental Origins and Correlates of Infant and Adult Attachment

The formation of a secure attachment relationship with a primary caregiver is an important organizational-developmental task that contributes to setting the foundation for social and emotional development throughout the life course. Two notions central to attachment theory and research are its clear emphasis on the quality of the parent-child attachment relationship, which is argued to be distinct from constitutionally based adaptation, such as temperament, for infant attachment security and, more recently, that infant attachment disorganization serves as a diathesis for the subsequent development of later dissociative symptomatology. Along with my colleagues, I have completed work addressing these propositions. Using molecular genetic methods, my collaborators and I found little evidence for either main effects of genes or G x E interactions in explaining infant attachment security and disorganization (Luijk, Roisman, Haltigan, et al., 2012). Second, along with my colleague, I have demonstrated that it may not be infant attachment disorganization, but rather infant attachment avoidance, that may hold especial predictive significance for later dissociative symptomatology (Haltigan & Roisman, 2014). Notably, our failure to detect a reliable association between infant attachment disorganization and dissociative symptomatology questions the prevailing orthodoxy in attachment scholarship that infant disorganization represents a specific vulnerability factor for the development of dissociative symptomatology.

The Adult Attachment Interview (AAI; George, Kaplan, & Main, 1985) is a semi-structured interview used to assess adults' access to a secure attachment-relevant 'schema,' or a mental understanding that effective attachment relationships serve the complimentary functions of secure base and safe haven provision. Since its inception, an axiomatic assumption has been that security in adulthood is a unitary construct. In contrast, adult attachment insecurity has been conceptualized as taking one of two mutually incompatible forms; dismissing states of mind reflect emotional deactivation and preoccupied states of mind reflect emotional hyperactivation during the recounting of early life attachment experiences of individuals administered the AAI. In a programmatic set of studies anchored in measurement science that addressed these fundamental assumptions of the AAI, my colleagues and I have also examined the latent structure, measurement invariance, and predictive significance of adult attachment as measured by the AAI (Haltigan, Leerkes et al., 2014; Haltigan, Roisman, & Haydon, 2014). In the first paper from this work, my colleagues and I provided confirmatory factor analytic evidence that the latent structure of individual differences in adult state of mind with respect to attachment is distributed along two dimensions that are only weakly correlated: dismissing and preoccupied states of mind. The importance of this work was that it that demonstrated that adult attachment security is not a monolithic construct, but rather is empirically defined by low levels of dismissing and preoccupied states of mind.

As African American populations are often disproportionally represented in high-risk and disadvantaged samples with high mental health burden, it is important to better understand how cultural and contextual factors influence attachment states of mind, parenting behavior, and children's development. In the second paper from this work, I examined cultural differences in adult attachment states of mind measured prenatally and their associations with individual differences in parenting using an independent sample (Haltigan Leerkes et al., 2014). This study provided the first demonstration that the constructs measured by the AAI have similar empirical meanings across African American and European American subsamples of women and that the predictive significance of dismissing states of mind for parental sensitivity across the two ethnic groups of mothers is equivalent. Additionally, the AAI narratives of African Americans demonstrated elevations in attachment preoccupation and loss relative to European Americans. I have interpreted these differences as stemming in part from meaningful adaptations to contextual adversities and reflective of the legacy of sociocultural-specific influences that are brought to bear on African Americans as a function of their ethnic and ecological niche (Wakschlag et al., 1996). One hypothesis that flows from this later finding is that the negative parenting behaviors typically associated with insecure attachment states of mind may not show the same degree of association with behavioral and other developmental outcomes in African American children as they do for European Americans. I have recently published collaborative, multi-sample work (Haltigan et al., in press) which has examined this hypothesis while programmatically extending the above AAI cross-ethnic measurement work.

Early Caregiving and Social Influences on Biobehavioral and Emotion Regulation

Although a large body of theoretical work suggests that the quality of the caregiving environment, and more specifically the infant-caregiver attachment relationship, is a core calibrating agent of the infant and child's emotional regulatory capacities and stress response systems, there is a relative lack of empirical research convincingly demonstrating such associations. As such, there remains an important need for hypothesis-driven, mechanistic research, evaluating the contribution of early caregiving and social experiences to the development and functional integration of behavioral and physiological stress regulation in children. My colleagues and I have examined process mechanisms linking adult attachment security and stylistic parenting behaviors with distinct affective-behavioral

signatures (Haltigan, Leerkes, et al., 2014). This research was informed in part by theoretical work suggesting that the minimization and maximization of behavioral and emotional affect, which are characteristic of distinct insecure attachment patterns, may be conditional behavioral strategies that serve to maintain caregiver-child attachment relationships in different ecological contexts. As such, adult attachment states of mind can be viewed as a set of cognitively encoded rules for caregivers to process and behaviorally respond to attachment-relevant cues and information, especially negative affect (Main et al., 1985).

Results of this work were consistent with the notion of emotional display rule acquisition (Malatesta & Haviland, 1982) and suggested that mothers' attachment representations *moderated* the relationship between infant negative affect and maternal interactive behavior within the FFSF paradigm. In response to infant negative affect, mothers with elevated levels of attachment preoccupation engaged in more intrusive and withdrawn behaviors with their children. Intrusive and withdrawn behavior may facilitate the maintenance of these mothers' (habitual) preoccupied state of mind by stimulating and prolonging their infant's negative affect, and can also be seen as adaptive in 'risky' ecological contexts where very high levels of parent-infant vigilance regarding the availability of one another confers survival or protective (i.e., safe haven) value. However, they also have the potential to foster affective dysregulation and attachment resistance in their children, and may forecast long-term deficits in children's ability to effectively regulate emotions and behavior.

This work, along with related biobehavioral work with colleagues using the same sample (Leerkes, Supple, O'Brien, Calkins, Haltigan et al., 2014), which found that mothers' physiological regulation in response to infant crying indirectly effected their sensitivity to infant distress via its influence on mothers' cognitive processing of their infants crying, further stimulated me to consider how the quality of the early caregiving environment is physiologically reflected in parents and children as well as how attachment relationships and biobehavioral emotional regulation strategies may be transmitted across generations. Porge's polyvagal theory (1995, 2007), which emphasizes the neuroperception of safety as a necessary condition for attachment, provides a strong empirical framework from which to evaluate claims that caregivers with secure attachment representations will show clearer signs of parasympathetic withdraw, and other endocrine indicators of adaptive coping, when confronted with attachment-related stressors such as infant negative affect. It has been suggested that such adaptive physiological response patterns may serve to scaffold the instantiation of adaptive stress system responses, emotion regulation capacities, and the development of attachment security in caregivers' own children (Schore, 1994). In addition to current collaborative work investigating this issue (Groh, Haltigan, et al., in prep), I am currently developing several grant proposals to initiate collaborative neurobiologically informed, research examining the impact of environmental adversity on psychopathology in both perinatal and youth cohorts.

This line of my research is also being developed through current work with my colleagues (Haltigan, Lyons-Ruth et al., in prep) examining the latent structure and predictive significance of high-risk parenting behaviors, characterized by intrusiveness and withdraw, for atypical infant attachment patterns and early childhood psychopathology. This work is conceptually positioned as a downward extension of recent work emphasizing the heuristic and explanatory value of conceptualizing early environmental adversity as a multidimensional construct, with a core distinction between adversity characterized by threat versus that characterized by deprivation (Sheridan & McLaughlin, 2014), for understanding child and adolescent psychopathology. Central to this effort has been the use of Item Response Theory (IRT) and item response mixture modeling in the development of a clinical short-form of the Atypical Maternal Behavior Instrument for Assessment and Classification (AMBIANCE) for use in family and child protection settings (Haltigan, Madigan, et al., 2018).

Social Media & The Covid Pandemic: Implications for Child and Adolescent Mental Health

Most recently, in the wake of the COVID-19 pandemic and associated public health response, my work has strongly focused on the influence of social media and public health messaging on child and adolescent mental health. This work was strongly facilitated by several collaborations with new colleagues which organically formed during the course of the pandemic via networking on social media and web-based platforms. These collaborations resulted in a widely publicized commentary in *Comprehensive Psychiatry* (Haltigan, Pringsheim, & Rajkumar, 2022) addressing the influence of immersive social media on child and adolescent mental health, especially that of adolescent girls, as well as the shepherding of a special section in *The Canadian Journal of Child and Adolescent Psychiatry* (Haltigan, 2023) that brought together several researchers from medicine and psychology to openly debate the evidence concerning whether and how much pandemic non-pharmaceutical interventions (NPIs), especially school closures, had on child and adolescent mental health functioning. Finally, along with colleagues from Stanford University and the University of Newcastle, Australia, I am completing a rigorous re-analysis of the widely publicized Bangladesh Mask Study (Abaluck et al., 2021) which served as a principal source of empirical support for public health messaging concerning the efficacy of mask-wearing in preventing Sars-CoV-2 community spread and the imposition of regional mask mandates. This re-analysis (Haltigan et al., in prep) leverages mixed-modeling and

Generalizability Theory methods to demonstrate that the original study findings were problematic and ultimately counter-productive for enhancing public trust in official federal, state, and university health institutions and authorities.

Summary

I plan to programmatically advance my interdisciplinary work in developmental psychopathology, with both new, independent work as well as continued collaborations. I plan to use findings from my prior research to serve as pilot work for research funding that will allow me to pursue my future research and help develop my independent research program. *Collectively, this work will be organized by: (1) a focus on understanding how early childhood adversity, life stress, and social experiences impact developing brain and bioregulatory systems and shape the course of mental and physical health in childhood and adolescence; and (2) a top-down, and theoretically functional, empirical approach to understanding the transdiagnostic phenotypic architecture of psychopathology that complements current bottom-up efforts to identify transdiagnostic neurobiological mediators of mental illness, such as the NIH RDoC initiative.* By developing a clearer understanding of the history-dependent, multi-leveled, multi-causal nature of psychopathology and adaptation, I hope to contribute to basic science in developmental psychology and to provide translational contributions that inform clear targets for prevention and intervention science.

Teaching Statement

Overview

I have had a number of teaching experiences across my graduate and post-graduate training that have shaped my philosophy and practice of teaching. I believe that the attitude, conviction, and authenticity with which one engages in teaching is a crucial factor influencing how well course material is received, retained, and applied by the student. A passionate and personalized attitude regarding the subject matter conveys the importance and personal meaning of the information from the speaker's perspective, and in my experience, readily engages students' attention and critical thinking potential. From this standpoint, I believe that the choice of what one teaches should strongly be influenced by areas of personal interest.

Teaching Philosophy & Pedagogical Approach

My philosophical approach to teaching has been influenced by the thoughts and ideas of Dr. Margaret McFarland (1905-1988). McFarland, a professor of child psychology at the University of Pittsburgh believed that in teaching, attitude mattered above all. "Attitudes aren't taught, they're caught" she was known to say. Inherent in this view of attitudes being 'caught' is the notion that an individual's intellectual passion can be contagious and that teachers and mentors play an important role in the formation of students' personal and professional identities. To this end, I strive to demonstrate my passion and enthusiasm for course subject matter when engaging with students. I will often share my own research findings with them as well as why I was interested in a given research question. I strive to encourage each student to develop his or her own passions, set personal goals, and formulate plans that will help them accomplish their short and long-term goals as they progress in their education.

Identify, Engage, & Inspire

I believe it is important to develop a strong rapport with students allowing them an opportunity to identify with me. At the beginning of a course, I often share my academic story with students and spend time getting to know their interests and future aspirations through open dialog and by asking them to complete a short questionnaire that prompts students to provide relevant information about themselves. I also believe it is essential to engage students in the course material in ways that are interesting, relevant, and that stimulate critical thinking skills. As a way of accomplishing this, I design class activities that help students learn through discovery. For example, in providing lectures on child development and attachment I have shown students actual Strange Situation Procedures and have had them rate and classify infants; similarly, I have provided de-identified Adult Attachment Interview transcripts which I have had students read and classify. Following these exercises, students' ratings and classifications are compared to experts' ratings and classifications and class discussion surrounding coding and classification issues has been stimulated. Most recently, in my Forensic Psychology course at the University of Ottawa, *New York* magazine contributing editor Robert Kolker participated in an online Blackboard discussion forum and conducted a Skype interview with my class regarding his book *Lost Girls,* a *Publishers Weekly* top ten book of 2013 and *New York Times* book review 100 notable books of 2013, which was required reading for my course. By relating course material in a more direct, hands-on, and personalized manner I believe it is possible to facilitate retention of concepts, encourage classroom engagement, and motivate students to autonomously pursue knowledge acquisition outside of the classroom.

Finally, I believe it is highly important to foster critical thinking skills and an open-mindedness in my students. I make efforts to prepare assignments and lectures that reflect these skills rather than merely testing students' ability to memorize facts. I supplement objective exams with papers, projects, and small-group assignments that teach students to critically evaluate the literature, understand the challenges facing researchers, summarize knowledge in meaningful ways, and to engage in verbal dialog that provides an opportunity to marshal a coherent view on a given topic and to reasonably defend it against alternative viewpoints. I strive also to prepare students to be critical consumers of knowledge and to develop skills to find, evaluate, and apply current

subject knowledge and research findings. As one example of how I encourage students to develop critical thinking skills, I discuss with them my research examining the question of whether early experiences have an enduring or transient influence on human development. In so doing, I am able to provide a personal example of how students can begin to think more critically and from multiple perspectives about ideas or theories that interest them.

Teaching from a Developmental Psychopathology Perspective

In view of my research program in developmental psychopathology, a unique aspect of my teaching philosophy is that I strive to teach with an eye towards a developmental psychopathology perspective in the classroom. In my instruction, I strive to highlight that development is influenced by a dynamic interplay among biological, behavioral, and sociocultural factors and that it is a probabilistic rather than a deterministic process. Using the rail yard metaphor of developmental pathways in adaptive and maladaptive development, I encourage students to consider aspects of their own developmental histories that may have served as deflections away from or onto the 'mainline' of developmental adaptation. In this manner, classroom discussion is organized by a focus on an organizational-developmental approach to the study of human development where the child is considered in transaction with its environment in an ever-increasing array of developmental issues in which prior adaptation is brought forward and transformed by current experiences.

With this approach as a guiding framework, I encourage students to make connections across multiple domains of development and the diverse range of biological, ecological, and sociological determinants of mental health. As an example of this approach, in my Forensic Psychology course, I have students trace the developmental pathways of individuals who became homicide victims. Using a reputable published book on the case, we trace each individuals probable adaptation considered from the perspective of developmental psychopathology and discuss ways in which failures of adaptation at earlier stages of development may have served as risk factors for the later development of maladaptation (e.g., drug use, sex trade work) which placed these individuals at risk for victimization.

Teaching and Mentorship Experience

I have had a variety of teaching experiences at the undergraduate level which have exposed me to a diverse array of students. I have formally taught Introduction to Psychology (PSY 1010; Castleton State College) and Introduction to Computers (MAT 117; Castleton State College, VT) and Introduction to Psychobiology (PSY202K; University of Miami, FL). I have also taught fairly large-sized 3rd year courses (~100 students) during my post-doctoral fellowship at the University of Ottawa. In the fall 2014 session, I was the instructor for Forensic Psychology (PSY3173A). This course had as a focus the developmental origins and course of antisocial behavior. Most recently, during the winter 2016 session, I was the instructor for Interpersonal Relationships (PSY3102C). This course examined in detail the role of environmental and biological contributions to close interpersonal relationships across the lifespan. In addition to these formal primary instructor experiences, as a graduate student at the University of Miami, I frequently provided lectures on attachment for child development or developmental psychology introductory courses. Further, I have served as a TA for both abnormal psychology and child psychopathology (Castleton State College), and served as the post-doctoral facilitator of the *Adult Attachment Interview (AAI) Transcription Group* (PSYCH 290) at the University of Illinois at Urbana-Champaign. This latter experience with PSYCH 290 involved overseeing a large team of undergraduate assistants in AAI interview transcription and facilitating a weekly seminar on relevant developmental psychology research literature and scholarship. Each of these experiences has been rewarding and has continually allowed me to evolve and refine my course materials and presentation strategies.

My experience teaching Introduction to Psychobiology at the University of Miami was particularly rewarding as I was able to convey my strong personal appreciation of the study of psychobiological processes within the context of didactic course presentations and lectures that reflected my positive personal attitude and enthusiasm

toward the material. My written and item-based teaching evaluations for this class were positive, with the majority of students reporting that course material was presented effectively, and that their interest in the course was stimulated. Most rewarding to me, however, were evaluations indicating that I challenged students to think. My view is that effective teaching lies more in stimulating creative and critical thinking skills rather than in memorizing facts or precepts in a given content domain.

Besides the teaching experiences noted above, I have also served as both faculty supervisor and graduate student co-supervisor to a number of undergraduate research assistants and scholars both at the University of Toronto and the University of Miami. Most recently I was the faculty mentor of an advanced undergraduate student from the University of Western Ontario who completed a summer research immersion experience with me at the University of Toronto. This experience was very enriching for me, and resulted in the student developing a final working conceptual paper that we aim to submit for publication. I was also the primary graduate supervisor and mentor of two undergraduate honors students who completed honors theses research projects in our laboratory. This experience was particularly rewarding for me as I felt that I was able to help these students critically evaluate and develop their research projects while also effectively communicating the theoretical and practical motivations behind specific data collection efforts in our laboratory. This experience also highlighted to me the importance and value of involving undergraduates in the research process and stimulated me to think about novel ways in which undergraduates could be involved in my own research program.

Courses of Interest

Given my training in developmental psychology and my focused interest in developmental psychopathology, as well as my methodological and quantitative background, I am particularly interested in teaching a variety of courses including, but not limited to:

- Developmental Psychology
- Developmental Psychopathology
- Developmental Psychobiology
- Research Design and Methods
- Introductory Statistics Courses
- Advanced Statistics Courses (e.g., SEM, latent mixture modeling)

I am also interested in developing and teaching more focused seminars on special topics such as attachment and parenting which my research background makes me strongly equipped to do.

September 2nd, 2023

Search Committee
[redacted]
Department of Psychology
[redacted]

Dear Search Committee,

It is with great enthusiasm that I submit my application materials for the posted faculty position in developmental psychology within the Department of Psychology at the University of Southern California. My research program spans developmental psychology, developmental psychopathology, and adolescent health. I believe my methodological training and background in developmental processes that occur across infancy, childhood, and adolescence fit extremely well with the expertise you are seeking for this faculty position.

My research program is theoretically informed by both attachment and evolutionary, life history theory perspectives and examines normative and atypical development to understand basic processes that influence pathways of development toward or away from psychopathology and mental illness. It is both multi-method and multi-level in its approach. My current work includes research contributing to the development of a common quantitative framework for psychopathology and an examination of the interplay between early social experience and neurobiological functioning in forecasting adolescent and young adult psychopathology. A core feature of my work is the application of cutting-edge variable and person-centered quantitative approaches that enable more precise and rigorous tests of developmental hypotheses in psychiatric science.

I possess expertise in a wide array of cross-sectional and longitudinal data analytic methods relevant to research in developmental psychopathology and child and adolescent psychiatry, including structural equation modeling (SEM), cross-sectional latent class and longitudinal growth mixture modeling (e.g., growth mixture modeling), and generalizability theory (GT). Additionally, I have highly specialized expertise in the dimensional modeling of psychiatric symptomatology, including item response theory (IRT), as well as experience using innovative predictive analytic tools, such as random forest regression, as well as Monte Carlo simulation methods, for both hypothesis testing and power analyses. I possess a strong background in the development of programming language in various analytic platforms including SPSS, M*plus*, and R.

My experience working with longitudinal datasets, including the National Institute of Child Health and Human Development Study of Early Child Care, has provided me with insight into the complexities of conducting and analyzing data from research projects of various sizes. I am also a member of the Hierarchical Taxonomy of Psychopathology (HiTOP) Consortium. The Consortium's chief objective is the advancement of the classification of psychopathology and the maximization of its usefulness for research and clinical practice. In this capacity, the consortium provides me with a valuable network of experienced and well-known psychiatric nosologists, psychologists, and psychometrists. I am

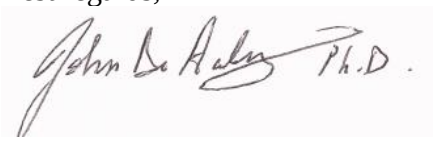
currently actively involved with the Neurobiological Foundations, Genetics, and Quantitative workgroups within the Consortium. My work with these workgroups is reflected in published work in outlets including *World Psychiatry* and the *Journal of Consulting and Clinical Psychology*.

I plan to programmatically build upon my recent quantitative work in psychiatric nosology that examined the metastructure of child and adolescent psychopathology and provided large sample evidence for a general factor of psychopathology in clinically referred children and adolescents assessed at CAMH (Haltigan et al., 2019; *Journal of the American Academy of Child and Adolescent Psychiatry*). This work has already been highly cited and was a key source for the developmental methodology of the Toronto Adolescent and Youth (TAY) cohort study (https://www.taycohort.ca) on which I am a principal investigator. I also plan to advance my research with both continued social and public health collaborations as well as new, independent work. This work includes the biological instantiation of emotion regulation in infancy and its relationship to the developmental of affective psychopathology and mood disorders in adolescence, as well as the mechanistic investigation of neurobiological pathways and endophenotypes that may serve to transmit the latent vulnerability of early social adversity to maladaptation. I am also current leading a comprehensive effort within the HiTOP consortium to further clarify the empirical structure of obsessive-compulsive (spectrum) disorder symptomatology and its relationship to emotional dysfunction, including both depressive and bipolar mental illness.

I am strongly committed to open and reproducible science, the teaching and mentorship of undergraduate and graduate students from diverse backgrounds, and actively promoting an academic climate of rigor, inclusivity, and liberal discourse. To this end, I have pre-registered research projects, shared teaching materials on the Open Science Framework (OSF), and mentored students from diverse backgrounds. My teaching evaluations have been highly positive, and I strive to engage students in novel ways as part of the classroom experience. I possess the capacity to teach both introductory and advanced (e.g., SEM, latent growth modeling) research methods and statistics courses.

I believe my research background and interests align well with faculty in the Department of Psychology at the [redacted] and look forward to hearing from you.

Best regards,



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Refining the assessment of disrupted maternal communication: Using item response models to identify central indicators of disrupted behavior

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Abstract

The Atypical Maternal Behavior Instrument for Assessment and Classification (AMBIANCE; Bronfman, Madigan, & Lyons-Ruth, 2009–2014; Bronfman, Parsons, & Lyons-Ruth, 1992–2004) is a widely used and well-validated measure for assessing disrupted forms of caregiver responsiveness within parent–child interactions. However, it requires evaluating approximately 150 behavioral items from videotape and extensive training to code, thus making its use impractical in most clinical contexts. Accordingly, the primary aim of the current study was to identify a reduced set of behavioral indicators most central to the AMBIANCE coding system using latent-trait item response theory (IRT) models. Observed mother–infant interaction data previously coded with the AMBIANCE was pooled from laboratories in both North America and Europe ($N = 343$). Using 2-parameter logistic IRT models, a reduced set of 45 AMBIANCE items was identified. Preliminary convergent and discriminant validity was evaluated in relation to classifications of maternal disrupted communication assigned using the full set of AMBIANCE indicators, to infant attachment disorganization, and to maternal sensitivity. The results supported the construct validity of the refined item set, opening the way for development of a brief screening measure for disrupted maternal communication. IRT models in clinical scale refinement and their potential for bridging clinical and research objectives in developmental psychopathology are discussed.

A large body of empirical work has established the importance of early disturbed care as a correlate of infant disorganized attachment (Cyr, Euser, Bakermans-Kranenburg, & van IJzendoorn, 2010). Disorganized attachment, in turn, is a reliable predictor of later maladaptation (e.g., Fearon, Bakermans-Kranenburg, van IJzendoorn, Lapsley, & Roisman, 2010; Madigan, Brumariu, Villani, Atkinson, & Lyons-Ruth, 2016; van IJzendoorn, Schuengel, & Bakermans-Kranenburg, 1999). However, the forms of disturbed care accompanying infant disorganization have proved more difficult to identify. Initial scales for parental sensitivity, while reliable predictors of organized forms of insecure attachment (i.e., avoidance and resistance) failed to provide good discrimination of disorganized attachment relationships (NICHD Early Child Care Research Network, 1997; van IJzendoorn et al., 1999; although see Bernier & Meins, 2008). To overcome the limitations of more global sensitivity rating scales, Lyons-Ruth and colleagues developed the Atypical Maternal Behavior Instrument for Assessment and Classification (AMBIANCE), which codes for the disrupted interactions more strongly associated with infant disorganization (Bronfman, Madigan, & Lyons-Ruth, 2009–2014; Bronfman, Parsons, & Lyons-Ruth, 1992–2008; Lyons-Ruth, Bronfman, & Parsons, 1999). The AMBIANCE is based on the premise that the parental response to infant distress must be predictable and responsive enough to allow the infant to develop a minimally effective attachment strategy for eliciting protection and care (Lyons-Ruth, Bronfman, & Atwood, 1999; Lyons-Ruth, Bronfman, & Parsons, 1999). The AMBIANCE measure includes indices of the frightening or frightened parental behavior discussed by Main and Hesse (1990), as well as additional indices of the caregiver's failure to help the infant regulate fearful or stressful arousal.

The resulting AMBIANCE coding system includes five higher order conceptual dimensions of disrupted caregiver behaviors: affective communication errors, role/boundary confusion, fearful/disorientation, intrusiveness/negativity, and withdrawing behavior. Within each of these five broadband dimensions, disrupted behaviors are further grouped according to subdimensions that reflect particular stylistic features and contexts within that dimension. In total, there are 15 subdimensions (see Table 1) that are thought to reflect relatively homogenous or unidimensional constructs. Trained coders record the number of disrupted behaviors displayed by a caregiver on each

We are grateful to our collaborators and colleagues who contributed data for this project and to the families who participated in the studies. Further work with the refined Atypical Maternal Behavior Instrument for Assessment and Classification item set presented in this manuscript is being supported by the Social Sciences and Humanities Research Council of Canada.

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Table 1. *AMBIANCE indicator descriptives and trimmed bootstrapped AMBIANCE item parameter estimates for fitted AMBIANCE maternal behavior subdimensions*

AMBIANCE Subdimension and Item Description	Proportion Occurrence (Raw Event Count)	Discrimination (a) (Mad)	Severity (b) (Mad)
Dimension 1: Affective Communication Errors			
Subdimension 1A: Contradictory signaling to child			
Sweet voice with derogatory message	.11 (38)	−7.40 (1.39)	−2.69 (1.59)
Invites approach verbally then distances	.02 (8)	0.32 (1.54)	0.89 (6.54)
Uses friendly tone, threatening posture	.02 (6)	−0.95 (2.63)	0.27 (6.19)
Directs infant to do, then not do something	.02 (6)	2.20 (1.50)	2.97 (1.67)
Offers then withdraws toy	.04 (13)	4.53 (1.51)	2.92 (1.28)
Holds affectionately, simultaneously withdraws/threatens infant	.01 (3)	−1.44 (1.28)	−1.48 (4.49)
Subdimension 1B: Failure to initiate responsive behavior to infant's cues			
Does not soothe infant when distressed	.24 (83)	13.08 (15.72)	0.78 (0.16)
Does not offer comfort when infant falls	.09 (32)	0.37 (0.23)	6.94 (3.16)
Fails to set appropriate safety limits	.02 (5)	0.56 (0.69)	4.23 (6.00)
Ignores cues for pickup	.38 (129)	6.72 (0.55)	0.48 (0.11)
Does not intervene when infant engages in dangerous behavior	.02 (6)	0.99 (0.78)	4.57 (1.93)
Does not respond to infant vocalization directed at caregiver	.30 (91)	0.06 (0.19)	2.05 (8.86)
Does not respond to infant cue	.26 (81)	0.14 (0.30)	1.26 (4.46)
Subdimension 1C: Inappropriate responding to infant's cues			
Laughs while infant crying/distressed	.17 (58)	2.36 (0.87)	1.25 (0.22)
Directs inauthentic affect toward infant	.04 (13)	−0.15 (0.63)	−0.15 (12.88)
Ignores infant cue for distance	.10 (31)	−0.15 (0.41)	−2.59 (12.14)
Ignores infant's "no"	.14 (49)	0.23 (0.28)	4.06 (3.94)
Mother smiles when infant angry, upset, afraid, or sad	.26 (67)	1.71 (0.46)	0.99 (0.19)
Minimize/discount infant's display of distress	.32 (111)	1.24 (0.34)	0.79 (0.18)
Dimension 2: Role/Boundary Confusion			
Subdimension 2A: Role confusion			
Elicits reassurance from infant	.09 (32)	1.00 (0.34)	2.80 (0.76)
Defers to infant	.01 (4)	0.29 (0.80)	3.30 (11.04)
Asks infant's permission to do something	.06 (19)	0.61 (0.34)	5.26 (2.18)
Demands affection from infant	.18 (60)	1.94 (0.62)	1.31 (0.25)
Seeks physical attention from infant while infant engaged in activity	.03 (10)	0.79 (0.58)	4.99 (2.08)
Prioritizes own needs over infant needs	.08 (28)	1.95 (0.69)	1.96 (0.38)
Repeats self-references	.35 (120)	0.42 (0.20)	1.67 (0.70)
Behaves as a child rather than parent	.09 (31)	0.88 (0.31)	3.11 (0.88)
Speaks in baby talk (not in response to infant)	.11 (38)	0.77 (0.28)	3.15 (0.96)
Uses "we" to describe self or infant	.06 (20)	0.37 (0.33)	6.76 (4.04)
Encourages infant to engage in negative behaviors	.02 (8)	0.17 (0.42)	3.79 (19.96)
Fake cries in response to infant–fake sadness	.02 (5)	1.40 (0.71)	4.00 (1.21)
Directs infant to self	.50 (153)	1.20 (0.40)	−0.02 (0.12)
Pleads with infant for attention	.04 (13)	0.73 (0.52)	4.60 (2.09)
Asks infant for reassurance around separation	.03 (9)	0.79 (0.49)	5.08 (2.09)
Threatens to cry	.01 (2)	1.95 (0.73)	4.39 (1.18)
Escalates infant's distress	.17 (45)	0.87 (0.31)	2.18 (0.61)
Subdimension 2B: Treats child as sexual/spousal partner			
Speaks in hushed intimate tones to infant	.09 (29)	17.45 (10.50)	1.43 (0.15)
Touches inappropriate body parts of infant	.01 (4)	−0.49 (1.01)	2.73 (6.41)
Behaves/speaks in manner more appropriate for spouse	.01 (4)	25.17 (4.38)	2.39 (0.33)
Kisses infant in sexualized manner	.03 (9)	1.55 (0.65)	3.15 (0.78)
Strokes in a sexualized manner	.01 (2)	20.55 (18.81)	2.60 (0.28)
Cups infant's face in hands with extended eye gaze	.01 (3)	0.00 (1.11)	2.16 (4.64)
Dimension 3: Fearful/Disorientation			
Subdimension 3A: Fearful behavior: appears frightened, apprehensive, or deferential in relation to the infant			
Exhibits frightened expression	.05 (17)	1.17 (0.65)	3.53 (1.13)
Handles infant in timid or helpless manner	.03 (11)	1.11 (1.25)	2.03 (1.09)
Exhibits smile with fear elements	.01 (4)	1.23 (1.29)	2.41 (1.19)

Table 1 (*cont.*)

AMBIANCE Subdimension and Item Description	Proportion Occurrence (Raw Event Count)	Discrimination (a) (Mad)	Severity (b) (Mad)
Exhibits highly vigilant posture in presence of infant	.01 (3)	1.52 (2.52)	1.35 (1.56)
Exhibits irrational fear regarding environment	.01 (4)	1.16 (1.35)	3.15 (2.21)
Startles to infant behavior without clear cause	.01 (3)	25.52 (24.47)	2.02 (0.42)
Treats infant as more powerful than self	.04 (12)	1.45 (1.07)	2.17 (0.83)
Hesitant, apprehensive, or stop-start movement in relation to infant	.04 (10)	0.19 (1.42)	1.23 (5.97)
Unexpected hesitancy/pause at moment of infant's bid for closeness/contact	.01 (3)	0.76 (1.56)	2.25 (3.78)
Approaches or moves away from infant in circuitous manner	.10 (33)	0.79 (0.98)	1.47 (1.16)
Approaches infant then quickly moves away	.02 (6)	0.86 (1.65)	1.84 (1.93)
Actively recoils from infant	.01 (2)	2.04 (3.02)	1.60 (1.35)
Fearful posture or expression (e.g., raised eyebrow, open mouth)	.10 (9)	0.83 (1.00)	2.22 (1.96)
Subdimension 3B: Disorientation or dissociative behavior			
Exhibits sudden change in mood unrelated to environment	.02 (6)	1.10 (0.69)	6.33 (3.24)
Handles infant as though inanimate	.06 (20)	0.69 (1.09)	1.36 (1.66)
Assumes trancelike posture or expression	.06 (19)	0.49 (1.18)	1.24 (2.86)
Deadened or flattened affect leaving empty feel to interaction (interaction)	.05 (15)	0.06 (1.57)	0.97 (3.83)
Exhibits sudden loss of affect	.03 (10)	1.38 (3.28)	0.73 (1.25)
Exhibits rapid shifts in affect unrelated to environment	.02 (6)	1.00 (1.75)	1.87 (2.44)
Exhibits disoriented or odd facial expression	.03 (10)	0.34 (1.47)	1.17 (6.48)
Sudden movement unrelated to environment	.03 (8)	0.60 (1.66)	1.03 (2.65)
Treats inanimate objects as animate	.02 (6)	1.19 (1.37)	2.69 (2.03)
Shifts rapidly from topic to topic or activity to activity	.13 (44)	0.05 (0.94)	1.20 (4.69)
Fails to finish movements	.01 (2)	−0.23 (1.92)	2.64 (8.06)
Subdimension 3C: Fearful or disoriented voices			
Exhibits haunted voice	.03 (10)	1.03 (0.69)	6.82 (3.67)
Exhibits frightened voice	.03 (8)	0.53 (2.12)	0.45 (4.90)
Exhibits sudden rise in intonation	.08 (26)	0.09 (0.50)	1.85 (11.92)
Exhibits stammering voice quality	.07 (23)	−0.95 (1.70)	0.54 (4.66)
Exhibits "ghost-like" whispering, stilted voice affectively disconnected	.19 (58)	−0.26 (0.37)	−2.57 (5.24)
Exhibits tense, high-pitched, squeaky voice tone such as at entry to room	.06 (18)	1.07 (2.10)	0.60 (2.60)
Exhibits sudden drop in pitch	.05 (18)	0.42 (1.43)	1.24 (5.53)
Exhibits sudden voice change, almost as if different person	.17 (51)	0.16 (0.86)	0.86 (5.06)
Affect or voice tone seems odd/unvarying in relation to environment	.01 (2)	0.06 (7.65)	0.80 (6.67)
Dimension 4: Intrusiveness/Negativity			
Subdimension 4A: Physical communications			
Pulls infant by wrist	.11 (36)	1.14 (0.34)	2.37 (0.51)
Looms	.12 (42)	1.01 (0.30)	2.36 (0.53)
Wipes infant's nose vigorously	.07 (24)	1.01 (0.42)	3.13 (0.86)
Pushes infant	.13 (43)	1.26 (0.33)	2.01 (0.35)
Attempts to grab infant	.06 (22)	1.88 (0.57)	2.15 (0.34)
Restrains infant	.10 (35)	1.20 (0.41)	2.31 (0.51)
Picks up or continues holding despite infant resistance	.09 (29)	1.35 (0.37)	2.32 (0.44)
Pulls infant into standing position	.03 (10)	1.21 (0.64)	3.57 (1.12)
Turns infant's head	.01 (3)	1.94 (0.77)	3.47 (0.90)
Behaves aggressively toward infant	.08 (26)	0.44 (0.34)	5.99 (2.80)
Touches infant in manner appearing affectionate but is irritating to infant	.18 (61)	1.35 (0.38)	1.53 (0.31)
Engages in rough physical play without enjoyment	.02 (8)	0.11 (0.56)	2.47 (14.27)
Tickles infant when infant resists	.01 (2)	6.37 (1.54)	3.54 (0.89)
Tosses toy or object at infant	.02 (7)	1.04 (0.58)	4.50 (1.73)
Physically crowds or hovers closely over infant	.12 (35)	1.12 (0.39)	2.22 (0.51)
Provides physical contact which offers no comfort	.09 (29)	0.63 (0.37)	4.11 (1.80)
Subdimension 4B: Verbal communications			
Mocks/teases infant	.11 (39)	1.58 (0.48)	1.85 (0.33)
Hushes crying infant (distinct from comforting sounds)	.16 (55)	0.88 (0.29)	2.27 (0.59)
Uses loud, sharp, or angry voice	.10 (33)	1.60 (0.45)	1.99 (0.33)
Disapproves, criticizes, or threatens	.25 (87)	2.32 (0.81)	0.86 (0.15)

Table 1 (*cont.*)

AMBIANCE Subdimension and Item Description	Proportion Occurrence (Raw Event Count)	Discrimination (a) (Mad)	Severity (b) (Mad)
Plays frightening games such as chasing infant	.06 (22)	1.05 (0.41)	3.17 (0.92)
Makes negative comment about infant	.10 (35)	0.83 (0.27)	3.10 (0.81)
Laughs at infant	.03 (8)	0.67 (0.42)	5.72 (2.21)
Subdimension 4C: Inappropriately attributes negative feelings or motivation to infant			
Suggests negative motivation to innocuous behaviors	.02 (5)	−2.41 (1.07)	−5.51 (2.64)
Indicates infant's actions could have harmful consequences	.02 (7)	2.12 (3.72)	1.20 (1.47)
Personalizes infant behavior as negative	.06 (20)	−1.38 (3.74)	−0.58 (2.83)
Ascribes negative feelings to the infant	.02 (6)	1.34 (2.67)	0.76 (5.15)
Subdimension 4D: Exerts control using objects			
Removes toy from infant despite engagement	.15 (51)	1.74 (0.53)	1.52 (0.26)
Withholds toy from infant	.12 (42)	1.96 (0.63)	1.61 (0.27)
Directs infant to new activity while infant clearly immersed in playing with toy	.27 (91)	1.31 (0.43)	1.07 (0.25)
Deals with objects in an angry manner	.05 (12)	1.35 (0.57)	2.99 (0.75)
Ignores cue that activity is not liked, continued too long, or is too difficult for infant	.04 (13)	1.76 (0.60)	2.67 (0.51)
Dimension 5: Withdrawing Behavior			
Subdimension 5A: Creates a physical distance from infant			
Holds infant away from body with stiff arms	.06 (22)	0.98 (0.30)	3.27 (0.78)
Squats behind infant to play	.13 (43)	0.97 (0.28)	2.40 (0.53)
Backs away from infant	.08 (27)	1.77 (0.55)	2.06 (0.34)
Stands and looks down to interact with infant	.14 (47)	0.73 (0.26)	2.94 (0.83)
Turns infant away from body when holding	.18 (62)	1.06 (0.27)	1.77 (0.34)
Stands behind infant to lift	.02 (5)	0.85 (0.66)	5.17 (2.57)
Averts gaze	.09 (30)	0.54 (0.25)	4.93 (1.88)
Adopts posture designed to keep infant at a distance	.22 (75)	1.77 (0.39)	1.10 (0.15)
Maintains interaction at distance from infant	.16 (54)	0.46 (0.25)	4.18 (1.65)
Indicates touching infant uncomfortable/unpleasant	.02 (6)	1.17 (0.47)	4.26 (1.19)
Leaves area after infant approach	.04 (12)	0.86 (0.42)	4.63 (1.63)
Holds infant awkwardly	.07 (21)	0.89 (0.37)	3.52 (1.05)
Directs approaching infant away	.21 (62)	0.82 (0.24)	1.93 (0.48)
Distances when infant approaches	.05 (13)	0.95 (0.38)	3.79 (1.13)
Moves out of interaction to chair when infant clearly wants contact or interaction	.19 (56)	0.97 (0.27)	1.84 (0.39)
Puts infant down too soon before cue from infant	.36 (107)	1.70 (0.37)	0.54 (0.12)
Abrupt end to interaction	.02 (4)	1.37 (0.41)	3.90 (0.86)
Subdimension 5B: Use of verbal communication to maintain distance			
No interaction with infant	.01 (3)	1.81 (1.10)	10.01 (4.37)
Uses words to create distance	.03 (10)	0.14 (0.67)	1.93 (12.70)
Does not greet infant after separation	.45 (153)	2.14 (1.43)	−0.05 (0.41)
Interacts silently with infant	.36 (106)	−0.35 (1.07)	0.30 (1.42)
Leaves silently without speaking to infant	.09 (27)	−0.09 (0.71)	0.96 (7.38)
Subdimension 5C: Directs infant away from self via toys			
Steers infant toward toys from behind	.04 (15)	1.09 (0.48)	3.66 (1.25)
Redirects infant to toys not self as substitute for closer contact with parent	.47 (161)	1.19 (0.53)	0.14 (0.15)
Uses prop to keep infant at a distance	.09 (29)	3.72 (2.14)	1.84 (0.41)
Offers object to infant over unusual distance	.04 (12)	1.26 (0.76)	3.12 (0.89)

Note: The Atypical Maternal Behavior Instrument for Assessment and Classification (AMBIANCE) coding system descriptors taken from Bronfman et al. (2009–2014). Ordinary nonparametric bootstrap; 500 bootstrap replicates. Parameter estimates reflect the 10% trimmed mean across bootstrap replicates. Mad, median absolute deviation (from the median). For all subdimensions except 4C (see below), italicized items for each subdimension reflect the three strongest, positively discriminating items at the more severe end of the latent trait. Note that virtually all of these items also contain the most information (i.e., measurement precision) at the more severe end of the AMBIANCE latent trait, as item information/precision is related to the items discrimination value in the 2PLM model (see text). This set of items was then selected to constitute a potential screening version of the AMBIANCE for further analyses. Model solutions for Subdimensions 1A, 1B, 2B, and 4C were unstable across different random starts despite model convergence. As such, parameter estimates for these subdimensions should be considered as especially provisional.

subdimension during interactions with the infant, assign an overall rating (1–7) of the level of disrupted communication, and classify the caregiver behavior as either "disrupted" or "not disrupted" in communication with the infant (Bronfman et al., 1992–2008, 2009–2014). In the only major revision to the AMBIANCE, rating scales were added for each of the five dimensions of disrupted behavior, so that both continuous ratings as well as frequency data could be generated for each of the dimensions (Bronfman et al., 2009–2014). Prior to 2009, only very minor changes occurred in which a small number of behavioral items that were hard to define clearly and that coders then found difficult to code were deleted.

The AMBIANCE system has been used to code disrupted caregiver behaviors in both low- and high-risk samples among caregivers with children aged 4 months to 7 years (for review, see Lyons-Ruth & Jacobvitz, 2016). Meta-analytic work has confirmed an association of moderate effect size between disrupted maternal communication and disorganized attachment ($r = .35$; Madigan et al., 2006). Other work has provided evidence for the predictive and discriminative validity of disrupted caregiver behaviors in relation to disorganized, but not secure, infant attachment assessed 1 year later (e.g., Forbes, Evans, Moran, & Pederson, 2007). In addition, Madigan, Voci, and Benoit (2011) demonstrated that disrupted caregiver behaviors coded with the AMBIANCE were stable over a 6-year period. Finally, reduction in maternal disrupted communication was shown to be one mechanism mediating reduction in infant disorganized behavior in the context of a randomized intervention trial (Tereno et al., 2017).

Whereas the reliability and validity of the AMBIANCE coding system has been well documented, to date the measurement properties of the disrupted maternal behavior indicators that comprise the first-level of coding in the AMBIANCE system have not been investigated. The absence of a systematic investigation of these measurement properties is due in part to analytic and modeling challenges associated with frequency count variables (e.g., Madigan et al., 2006; Sterba et al., 2010). In addition, a reasonably large sample size is needed, given that most individual disrupted behaviors constitute low base-rate events. Nevertheless, as Madigan et al. (2006) noted almost a decade ago, more vigorous analyses of anomalous caregiving behaviors is needed to improve our understanding of the specific disrupted caregiving behaviors that best define atypical parenting. Moreover, given the high-fidelity nature of the AMBIANCE coding system, coding of particular caregiver behaviors is a laborious process, so that there is a significant demand from those working in clinical settings for a streamlined version that focuses on the most central indicators of disturbed interaction.

Latent Trait Models and Item Response Theory (IRT)

The latent trait model is the analogue of the factor analysis model for binary observed data (Muthén, 1989; Rizopoulos, 2006). Within the latent trait purview, IRT has emerged as a powerful set of modeling techniques for the analysis of item-level data obtained to measure interindividual variation (e.g., mental health status; Edelen & Reeve, 2007). However, the IRT methodological tradition originated in the measurement of latent traits of scholastic ability (e.g., reading and arithmetic; Baker, 2001), and thus, it has been used less commonly in clinical and developmental psychological science, where classical test theory approaches to instrument evaluation have been the standard. Nevertheless, the benefits and utility of IRT methods have been increasingly applied in clinical and developmental research (Cole et al., 2011; Edelen & Reeve, 2007; Fraley, Waller, & Brennan, 2000; Gordon, 2015; Reise & Waller, 2009). In particular, IRT methods can be used to provide highly detailed information on the properties of existing coding systems and their indicators, which can then be used to optimally shorten the instrument to effectively reduce coding or response burden. Accordingly, IRT methods have been applied to measurement instruments assessing mental health symptoms (e.g., depression; Cole et al., 2011), alcohol and drug symptomatology (Krueger et al., 2004; Langenbucher et al., 2004), and psychopathy (Cooke & Michie, 1997).

There are a variety of different IRT models that can be fit to binary response data (for a review, see Gordon, 2015). However, the two-parameter logistic IRT (2PLM) is often applied (e.g., Krueger et al., 2004; Langenbucher et al., 2004). A key assumption of this model is that the latent trait under investigation is a unidimensional (i.e., single-factor) construct. Item trace lines or item characteristic curves (ICCs) are produced, which are S-shaped logistic functions that graphically relate item endorsement probabilities across latent trait values (Edelen & Reeve, 2007; Martin et al., 2006). These lines are described by two parameters, the location (b) parameter, and the slope (a) parameter. The b, or location, parameter is the point along the ICC at which the probability of a positive response for a dichotomous item is 50%. The larger the location parameter, the more of the measured construct (often denoted as θ) a respondent must possess for a particular item to be endorsed. When the construct of interest (i.e., the latent trait) is relevant to mental health problems or physical disease, this parameter can be cast as the "severity" parameter. The a, or discrimination parameter, reflects how well a particular item discriminates respondents or "participants" at contiguous points around (i.e., above and below) the location parameter. In other words, it is the slope of the ICC at the value of the location parameter and indicates the extent to which the item is related to the underlying construct or latent trait. This parameter is analogous to the factor loading in traditional factor analysis.

After estimating the parameters of an IRT model, researchers can investigate the fidelity by which items measure a given latent trait by examining the item's information. In the 2PLM, an item's information value is inversely related to the item's discrimination parameter and reflects the standard error of the indicator at its location on the latent trait. As such, examining an item's information value provides crucial insight into how well (i.e., the precision with which) an indicator is measuring the latent construct under consideration. Exploring how an item's information changes as a function of the latent trait level is one of the most widely cited motivations for using IRT

in clinical measurement (Reise & Waller, 2009). Such information is especially useful in guiding efforts at reducing item measurement batteries so as to maximize their efficiency and precision. This contribution of IRT is especially useful in clinical and high-risk samples, where it is important to efficiently extract as much information as possible at the severe end of relevant traits in order to screen for mental and physical health concerns (Kim & Pilkonis, 1999). As Forero and Maydeu-Olivares (2009) note, there is significant demand from practitioners within medical settings for short assessment tools capable of gathering the maximum amount of information in the minimum possible time.

Study Overview

In the current study, we collected all known observations of disrupted maternal behaviors for which item-level data were available, in order to provide the first large-sample analysis of the item structure of the AMBIANCE using latent trait modeling under the IRT approach. As noted, the AMBIANCE is composed of 15 disrupted behavior subdimensions (Table 1). These subdimensions were originally conceptualized as unidimensional constructs reflecting particular stylistic patterns of disrupted maternal behavior. Thus, the latent trait IRT approach is well suited to address the chief objective of the current work, which is to assess the item properties for each AMBIANCE subdimension and identify the behavioral items most central to each subdimension. In so doing, we developed a preliminary and empirically informed, refined AMBIANCE item set and evaluated its convergent and discriminant validity with constructs in its nomological network (Cronbach & Meehl, 1955), including maternal sensitivity and infant attachment disorganization. Given the central importance of assessing disrupted parenting behaviors in clinical and child protective settings, identifying these empirically central items was seen as a critical first step toward the development of a more efficient clinical screening instrument for disrupted maternal behavior that maintained adequate conceptual and content coverage with maximum precision.

Method

Participants and procedure

Item-level AMBIANCE data, acquired from six subsamples (pooled $N = 343$) were used in the current project. Data were drawn from various parent studies conducted in the United States, Canada, and Great Britain. In the United States, AMBIANCE indicator-level data were obtained from the Harvard Longitudinal Study, a longitudinal investigation of the effects of social risk factors on child development ($n = 55$; Lyons-Ruth, Bronfman, & Parsons, 1999), and from a subset of participants in the NICHD Study of Early Child Care and Youth Development (SECCYD; $n = 219$; see NICHD Early Child Care Research Network, 2005, and the study website http://secc.rti.org). In Canada, AMBIANCE data were acquired from a larger study of preschool behavioral problems in healthy and pediatric medical conditions ($n = 39$; Goldberg, Gotowiec, & Simmons, 1995; Madigan et al., 2011), and in Great Britain data were acquired from a study investigating personal relatedness and attachment patterns in 12-month-old infants of mothers with and without borderline personality disorder ($n = 30$; Hobson et al., 2009).

AMBIANCE data were coded from mother–child interactions in a variety of standard interactive research paradigms, such as the Strange Situation Procedure, free play, and cleanup task, and included children from 12 months through 54 months of age (58% of children in the pooled sample were 15 months of age). Girls and boys were approximately equally distributed in the pooled sample. Individual study cohorts were heterogeneous with respect to demographic risk given the differing aims of the parent studies (for additional detail about the demographic characteristics of parent studies comprising the pooled sample, see parent study references cited above).

Measures

Disrupted Behavior Instrument for Assessment and Classification (AMBIANCE). The AMBIANCE coding system (Bronfman et al., 1992–2008, 2009–2014) is a detailed observational coding protocol that provides objective behavioral criteria for coding disrupted caregiver communication with the infant during videotaped caregiver–infant interactions. The coder first documents the frequency of approximately 150 behavioral items on 5 dimensions of disrupted maternal behavior. Frequency counts for individual items are then summed to yield total frequency scores for each of 15 subdimensions. A final overall level of disrupted communication (1–7) is assigned by the coder, based on both the frequency and the intensity of the disrupted communications displayed by the caregiver. The overall level of disrupted communication is scored as follows: 1 = *warm and sensitive communication*, 3 = *generally positive interaction with some evidence of disrupted communication*, 5 = *clear and repeated disruption in affective communication*, and 7 = *disrupted communication with few or no ameliorating behaviors*. Scores of 5 or above on the overall rating are classified as "disrupted" and scores of less than 5 are classified as "not disrupted" (Bronfman et al., 1992–2008, 2009–2014).

Reliability of AMBIANCE coding at the level of the overall rating and classification has been strong across all the studies whose item-level data are included here (see original parent study publications as follows: Hobson et al., 2009; Lyons-Ruth, Bronfman, & Parsons, 1999; Madigan et al., 2011). For the SECCYD subsample, reliability coefficients on $n = 62$ tapes (20%) between two coders was high, with ICCs for ratings on each of the five AMBIANCE dimensions all >0.80 (Mills-Koonce et al., 2017).

Although the exact number of AMBIANCE items has changed slightly over time (see Analytic Plan section), the total numbers of behavioral items for each AMBIANCE dimension available for inclusion in the current investigation were

as follows: affective communication errors = 23 indicators; role/boundary confusion = 24 indicators; fearful/disoriented behaviors = 37 indicators, intrusive/negativity = 34 indicators; and withdrawing behavior = 29 indicators. Thus, a total of 147 behavioral items were available for potential inclusion in the current analyses.

Infant attachment disorganization. In all studies, infant attachment was assessed during the standard Strange Situation Procedure (Ainsworth, Blehar, Waters, & Wall, 1978). The Strange Situation Procedure is an observational procedure that contains eight brief episodes of increasing stress for the infant, including two mother–infant separations and reunions. All video recordings were coded for infant attachment behaviors and for the three attachment classifications as described by Ainsworth et al. (1978) and for disorganized/disoriented behaviors as described by Main and Solomon (1990). Reliability of attachment classifications were satisfactory within each of the parent samples (Goldberg et al., 1995; Lyons-Ruth, Connell, Grunebaum, & Botein, 1990; Hobson, Patrick, Crandell, & Garcia-Pérez, 2005; NICHD Early Child Care Research Network, 1997).

Maternal sensitivity. The maternal sensitivity measure was available only in the SECCYD subsample (n = 197). Early maternal sensitivity was assessed in the context of mother–child interactions that were videotaped during 15-min semi-structured play procedures at 6, 15, 24, and 36 months. At 6 months, mothers and children were instructed to play together, first with toys available in the home (or none at all) and then with a standard set of toys. At 15, 24, and 36 months, mothers were asked to show their children age-appropriate toys in three containers in a set order. As in prior studies of this sample (e.g., NICHD Early Child Care Research Network, 2001), observations of maternal sensitivity from the first 3 years of life (6, 15, 24, and 36 months) were standardized and averaged to create a composite of the observed early sensitivity. At 6, 15, and 24 months, the a priori maternal sensitivity composites were constructed by summing ratings for sensitivity to nondistress, positive regard, and intrusiveness (reversed). At 36 months the supportive presence, respect for autonomy, and hostility (reversed) scales were composited (as reported in NICHD Early Child Care Research Network, 2001, internal consistencies of composites were 0.75, 0.70, 0.79, and 0.78 for the 6-, 15-, 24-, and 36-month composites, respectively, and intercoder reliabilities on scales >.80; for additional details on the sensitivity composite, see NICHD Early Child Care Research Network, 2001, 2004).

Analytic plan

We first transformed the 147 AMBIANCE indictors from their original count scales to a dichotomous (0 = *behavior did not occur*, 1 = *behavior did occur*) scale to ease modeling burden and permit IRT modeling under the 2PLM. This transformation resulted in relatively little loss of information because most indicators had extremely low base rates due to their atypical nature.

Over time, the exact number of items in the coding manual has changed slightly, because some items were found to be difficult to code and were deleted, while new behaviors of particular import were observed in subsequent samples and added to the item list. In the current analyses, we included all AMBIANCE items that were coded in *any* of the parent studies. Next, to improve model estimation tractability (i.e., the ability to generate stable model solutions and parameter estimates), from the total pool of 147 AMBIANCE indicators available for consideration in the current analyses, indicators with zero variance (i.e., no event occurrences) and/or minimal variability (i.e., only one event occurrence) were excluded. Percentages of items with zero or minimum variability were generally evenly distributed across the five primary dimensions of the AMBIANCE: affective communication errors (17%); role/boundary communication (19%); fearful/disorientation (17%); intrusiveness/negativity (6%); and withdrawing behavior (10%). In light of model estimation concerns, if greater than 90% of the data for a specific AMBIANCE indicator were missing (e.g., due to removal from subsequent versions of the manual), it was also removed from consideration in analyses.[1] This resulted in a total AMBIANCE indicator pool of 133 items for inclusion in the IRT analyses. Missing or unavailable data on remaining AMBIANCE indicators across study cohorts ranged from none to 29%.

Using the *ltm* package (Rizopoulos, 2006) in the R environment for statistical computing (R Core Team, 2016), we then fitted latent trait models for each of the 15 subdimensions comprising the AMBIANCE maternal behavior system. The ltm package uses marginal maximum likelihood estimation (Bock & Aitkin, 1981), which is a commonly used iterative estimation procedure that provides maximum likelihood estimates of severity and discrimination parameters. Under marginal maximum likelihood estimation, all missing data are treated as missing at random, and all available cases are used in model estimation taking into account the observed part of sample units with missing data. We also specified start.val = "random" to allow for inspection of local maxima issues in likelihood surfaces (i.e., replication of the best log-likelihood across different start values).

It is well known that maximum likelihood estimation of latent (trait) models with binary or ordered categorical data present modeling challenges (Albanese & Knott, 1994; Sterba et al., 2010). This problem is further magnified in rare-event behavioral data, where many indicators demonstrate a preponderance of nonevents (i.e., the disrupted maternal behavior does not occur). Sparse data response patterns may lead to extreme parameter and/or standard error estimates, which are unstable (de Menezes, 1999) or frequently drift into inadmissible regions (Swaminathan, Hambleton, Sireci, Xing, & Rizavi, 2003). Albanese and Knott (1994)

1. Full descriptions of the 14 items removed from analyses are available from the first author upon request.

showed that estimated asymptotic variances of the parameter estimates in a one-factor model for binary data are unreliable. They arrived at a better idea of the sampling distribution of the parameter estimates by bootstrapping. Given the model estimation characteristics of the current data set, we also performed 500 bootstraps of each latent trait model using the *boot* package (Canty & Ripley, 2016) to arrive at a better approximation of the *sampling behavior* of the estimators (i.e., more precise values of the estimators and their standard errors).

For each of the 15 disrupted maternal behavior latent traits, we decided a priori to select, whenever possible, the three items possessing the strongest positive discrimination parameters on the severe end of the trait. This approach was taken to maintain the empirical meaning of each latent trait while also maintaining adequate content coverage of the AMBIANCE as a whole. These three items from each subdimension were then used to construct a 45-item refined AMBIANCE summary measure. We then evaluated the construct validity of this refined measure in relation to the full AMBIANCE coding protocol, as well as in relation to infant attachment disorganization and maternal sensitivity.

Results

Descriptive data

The proportions of observed occurrence and the raw frequency counts for all binary AMBIANCE indicators included in the present analyses are presented in Table 1. Base rates ranged from 0.01 to 0.50. As can be seen, means (i.e., proportions of occurrence) of AMBIANCE binary indicators indicated that most are rare behaviors, with low base rates of occurrence. The relative rarity of these observed behaviors is consistent with the goal of the AMBIANCE coding system to detect atypical maternal behavior.

Latent trait models for AMBIANCE subdimensions

To assess the unidimensionality of each AMBIANCE subdimension, we performed a likelihood ratio test evaluating the fit of one- and two-factor models for each subdimension using the ANOVA function in the ltm package (Rizopoulos, 2006). With the exception of the physical communications (4A) and creates physical distance from infant (5A) subdimensions, a two-factor model did *not* provide a significantly better fit to the data than did a unidimensional model (for model comparisons, all $ps \geq .05$). This supports the notion that for 13 of 15 AMBIANCE subdimensions, indicators for each disrupted maternal behavior should be conceptualized as indicators of a single, dominant underlying dimension.

Although the likelihood ratio test above suggested that the physical communications (4A) and creates physical distance from the infant (5A) subdimensions may be better explained by two factors rather than a single factor, we examined them under the 2PLM IRT unidimensional assumption in this investigation for two reasons. First, these subdimensions were two of the three subdimensions in the current analyses with 16 or more indicators (see Table 1). With relatively large numbers of items, there are many opportunities for subsets of items to have shared method variance reflecting inconsequential multidimensionality not accounted for by the dominant trait (Cook, Kallen, & Amtmann, 2009; see Floyd & Widaman, 1995). Second, confirmatory model fit indices for the physical communications subdimension (4A) were adequate (i.e., confirmatory fit index ≥ 0.93, root mean square error of approximation < 0.05, weighted root mean square residual < 1.0), suggesting the tenability of a unidimensional latent trait.[2]

The interdecile (i.e., 0.10) trimmed mean bootstrap IRT discrimination and location parameter estimates and their median absolute deviation from the median for each latent trait model are also listed in Table 1. Recall that, within the context of the current investigation, an item's location refers to the point on each latent disrupted maternal behavior trait (i.e., each subdimension) at which there is a .5 probability of that item's being observed (vs. not). An item's discrimination reflects its ability to discriminate individuals around the item's location. Exemplar item characteristic and item information curves for selected subdimensions and items are presented in Figures 1–5.[3]

For 11 of the 15 subdimensions (excluded subdimensions are discussed below), the trimmed mean discrimination (slope) estimate across the AMBIANCE subdimensions was 1.23 (range = 0.24–2.97), which corresponds to a correlation of ~.59 between a particular disrupted maternal behavior item and the underlying disrupted maternal behavior latent trait continuum. This suggests that the AMBIANCE items were generally moderately to strongly associated with their underlying latent trait abstractions. The trimmed mean severity parameter estimate across these same AMBIANCE subdimensions was 2.27 (range = 0.73–3.43). Recall that the severity parameter is scaled on a standard (z-score) metric and therefore can be directly referenced to the underlying latent trait continuum. In the present investigation, zero indicates the average level of the particular disrupted maternal behavior in the sample on the underlying latent disrupted maternal behavior trait. Thus, the AMBIANCE maternal behavior indicators are, in general, measuring the higher or more severe end of their associated disrupted maternal behavior latent traits. For example, the strongest average within-subdimension severity was observed for the role confusion subdimension (2A), suggesting that the disrupted maternal behavior indicators for this construct were measuring its

2. Categorical data factor analyses were performed in Mplus V. 7.11 (Muthén & Muthén, 1998–2012) using a robust weighted least squares estimator. Note that categorical data factor analyses and IRT are equivalent parameterizations of the same underlying model. We return to the issue of the unidimensionality of AMBIANCE subdimensions in the Discussion section.
3. Additional item characteristic and information curves can be requested from the first author.

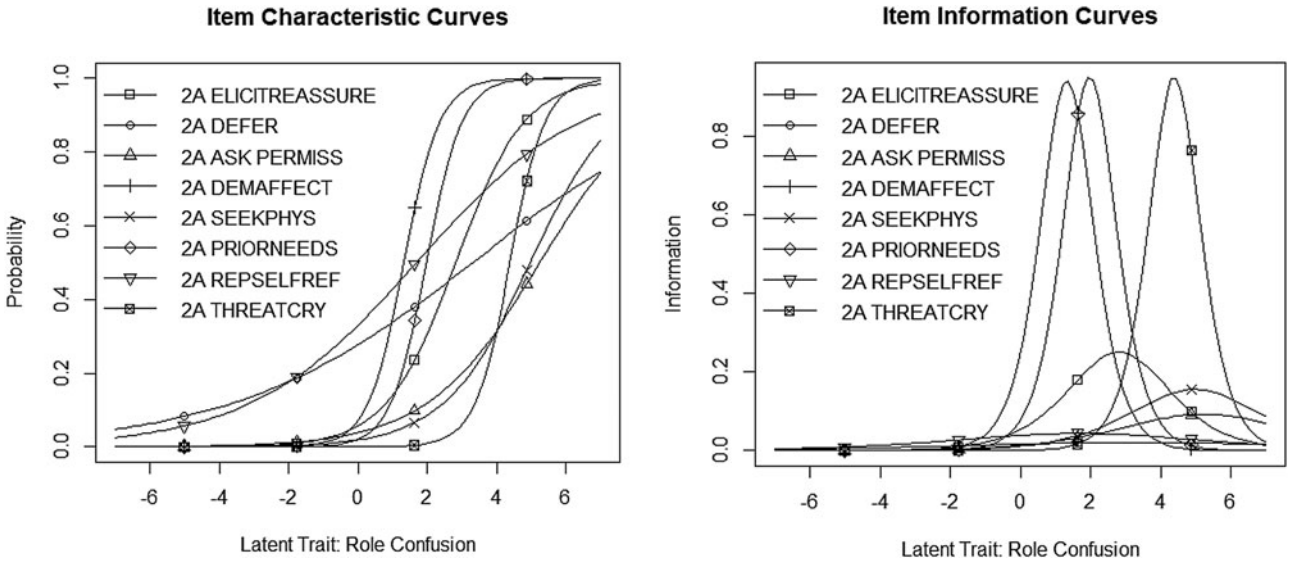

Figure 1. Item characteristic and item information curves for eight indicators of AMBIANCE subdimension 2A: "role confusion." Full item code descriptors are provided in Table 1. Chosen indicators were selected to facilitate visual interpretation.

more severe end particularly well. This is visually reflected in the right shift of the item characteristic and information curves on the latent trait (see Figure 1), as well as in the test information function for this subdimension (see Figure 6), where the largest portion of the area under the test information curve is at the more extreme end of the role confusion latent trait.

For the remaining 4 of the 15 AMBIANCE subdimensions (1A, 1B, 2B, and 4C; Table 1), model estimation was tentative. For each of these latent trait models, we were unable to replicate the best log-likelihood value three times in initial (nonbootstrapped) runs. In addition, some random starting values produced unstable individual model solutions in bootstrapped models as indicated by the absence of positive definite Hessian matrix at convergence. Finally, for some items bootstrapped standard error estimates were excessively large, indicating the extreme degree of instability for these particular parameter estimates (implications for model interpretation are discussed more fully below).

Another indicator of how well AMBIANCE items are indexing the higher or more severe end of disrupted maternal behavior latent traits is their precision (i.e., reliability) in doing so. An item's precision in IRT is reflected via its information. Item information may be thought of as the reliability of the items with respect to their ability to distinguish between respondents at *a given level of the latent trait.* AMBIANCE

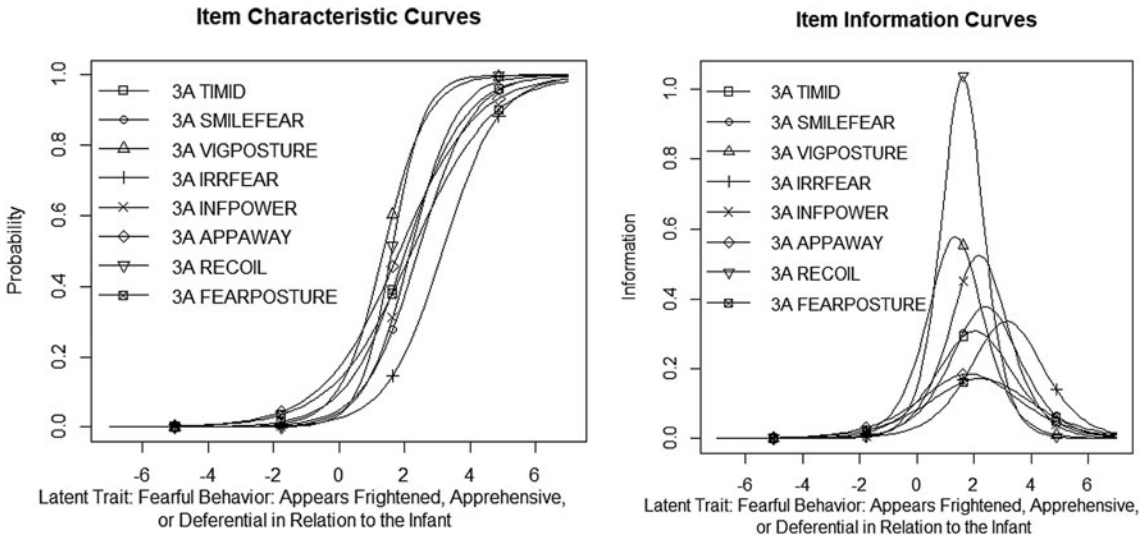

Figure 2. Item characteristic and item information curves for eight indicators of AMBIANCE subdimension 3A: "fearful behavior." Full item code descriptors are provided in Table 1. Chosen indicators were selected to facilitate visual interpretation.

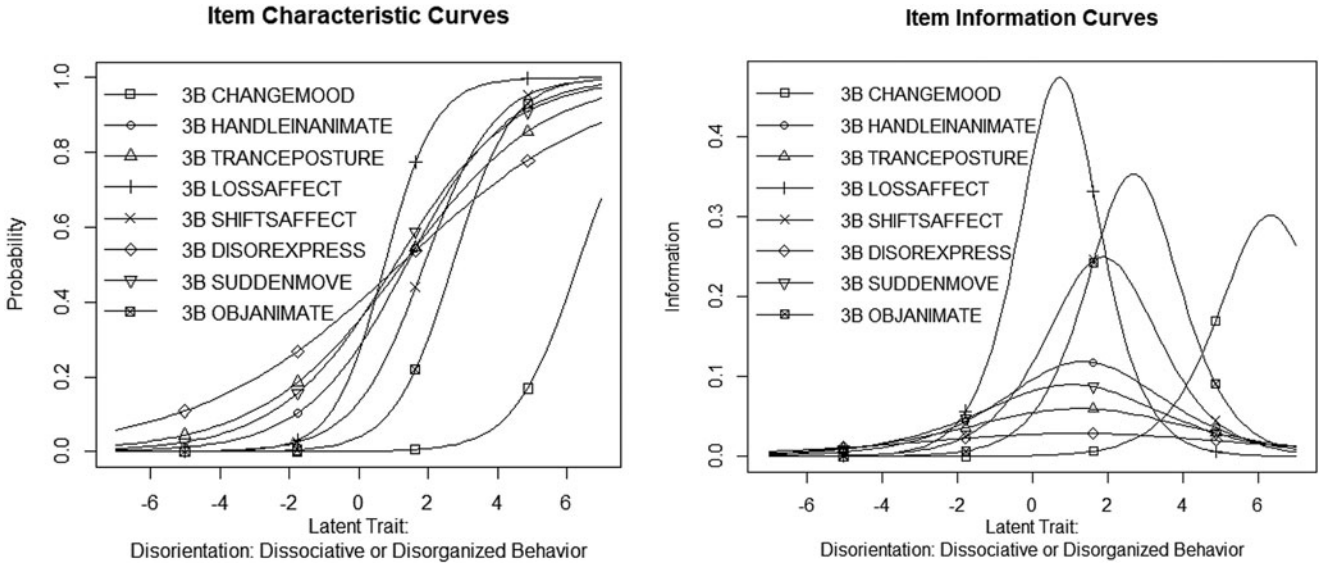

Figure 3. Item characteristic and item information curves for AMBIANCE subdimension 3B: "disorientation: disorganized or dissociative behavior." Full item code descriptors are provided in Table 1. Chosen indicators were selected to facilitate visual interpretation.

maternal behavior indicators generally demonstrated satisfactory reliability within the selected range at the more severe end of the disrupted maternal behavior latent trait (see Figure 6 for the role confusion test information function). Approximate total information values for each AMBIANCE subdimension (excluding the four subdimensions noted previously) on the latent trait continuum from 0 and +5 (i.e., the more severe end) were as follows: 1C (4.29), 2A (11.51), 3A (28.34), 3B (12.43), 3C (9.68), 4A (16.53), 4B (7.2), 4D (7.2), 5A (13.23), 5B (1.36), and 5C (5.1). These total information values correspond to IRT reliability (i.e., internal consistency) approximations of (0.77, 0.91, 0.97, 0.92, 0.90, 0.94, 0.86, 0.86, 0.92, 0.27 and 0.80, respectively), where reliability is calculated as 1 minus the squared reciprocal of the square root of the information for scores in that severity range of the disrupted latent trait (i.e., their error variance).[4] Thus, total information value provides a metric of how precisely the AMBIANCE items are measuring the more severe end of disrupted maternal behavior latent traits. The relatively smaller value of 0.27 corresponded to the subdimension use of verbal communication to maintain distance (5B). Visual inspection of the total information curve for this subdimension (not shown) revealed that the lower reliability value for this subdimension was because total item precision was most concentrated (i.e., the bulk of the item information) between –2 and +3 on the latent trait continuum (i.e., across the midpoint of the latent trait), rather than disproportionately at the severe end. This suggests that for this subdimension, items are more precisely measuring less severe and more benign aspects of this trait, rather than those mostly at the severe end.

Selecting a reduced indicator set for further evaluation as a screening measure

IRT parameter estimates were used to inform our selection of a reduced set of AMBIANCE indicators. As noted above, each of the 15 disrupted maternal behavior latent traits, we decided a priori to select, whenever possible the three items that possessed the strongest positive discrimination parameters on the severe end of the trait (recall that these items also possess the most information or precision). This approach was taken to maintain adequate coverage of the empirical meaning of each latent trait (i.e., item discrimination parameters are analogous to factor loadings), as well as to maintain adequate content coverage of the full AMBIANCE protocol (Edelen & Reeve, 2007), while also ensuring a clinically practical reduced set of indicators.

Note that we also selected items from the four AMBIANCE subdimensions (1A, 1B, 2B, 4C) with tentative model solutions per the a priori selection rule described above. As further discussed below, we selected items from these models despite tentative model solutions because omitting items from AMBIANCE subdimensions considered clinically and conceptually important would have substantively altered the meaning and conceptual content of the reduced item set in relation to the full AMBIANCE system.

Construct validity

Following identification of the 45-item set, a unit-weighted sum was computed ($M = 4.59$, $SD = 3.33$) and used as a re-

4. The reciprocal of the square root of the information value provides an estimate of the standard error of latent trait ability measurement in the specified latent trait range, which, when squared, provides an index of error variance in the same latent trait range.

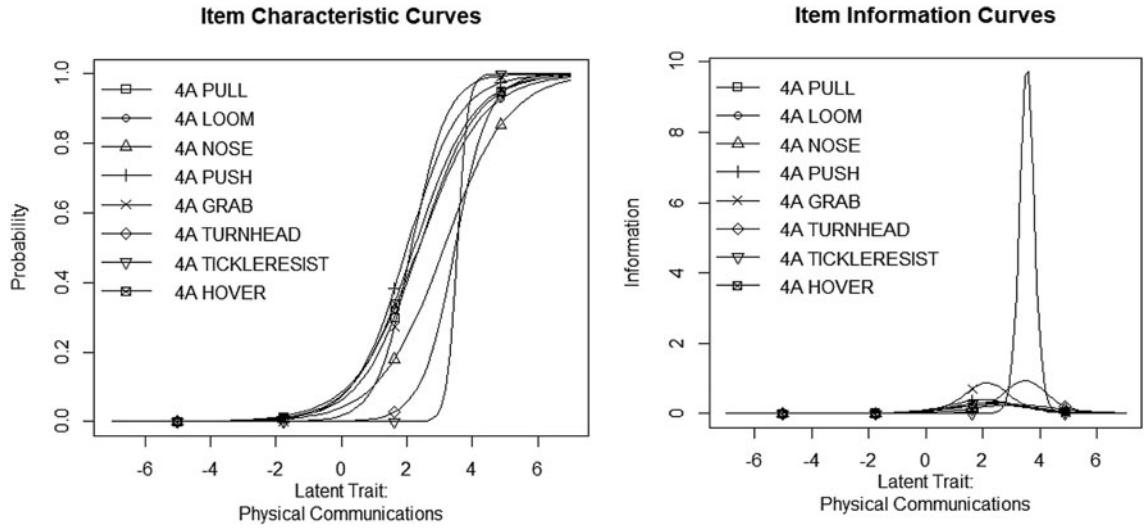

Figure 4. Item characteristic and item information curves for AMBIANCE subdimension 4A: "physical communications." Full item code descriptors are provided in Table 1. Chosen indicators were selected to facilitate visual interpretation.

fined index of disrupted maternal behavior. The unit-weighted summary score for the 45-item refined AMBIANCE was strongly convergent with a unit-weighted sum of the full AMBIANCE item pool (133 items) available in the present study ($r = .89$, $p < .001$). We evaluated the construct validity of the refined AMBIANCE 45-item set in several ways. First, we used receiver operating characteristic (ROC) analyses to explore the clinical utility of the AMBIANCE 45-item set with respect to the final overall maternal disrupted classification status that is assigned using the full AMBIANCE. To make a final classification, trained coders use the full AMBIANCE protocol to take into consideration the frequency and severity of all disrupted maternal behaviors and rate the overall level of disrupted communication observed in the parent–child interaction. Ratings of 5 or above on the overall level result in a classification as disrupted. Thus, the disrupted classification is based on a broader coder judgment than frequency counts alone.

ROC analyses are similar to logistic regression in that one can use the strength with which a set of explanatory variables predicts a given binary outcome to calibrate the precision of a measure by plotting the range of classification accuracy at different thresholds of the predictor variables. The resultant area under the curve (AUC) can then be quantified by a value representing the likelihood that a random chosen positive case (i.e., a mother classified as disrupted) will exceed the result for a randomly chosen negative case (thus ranging from

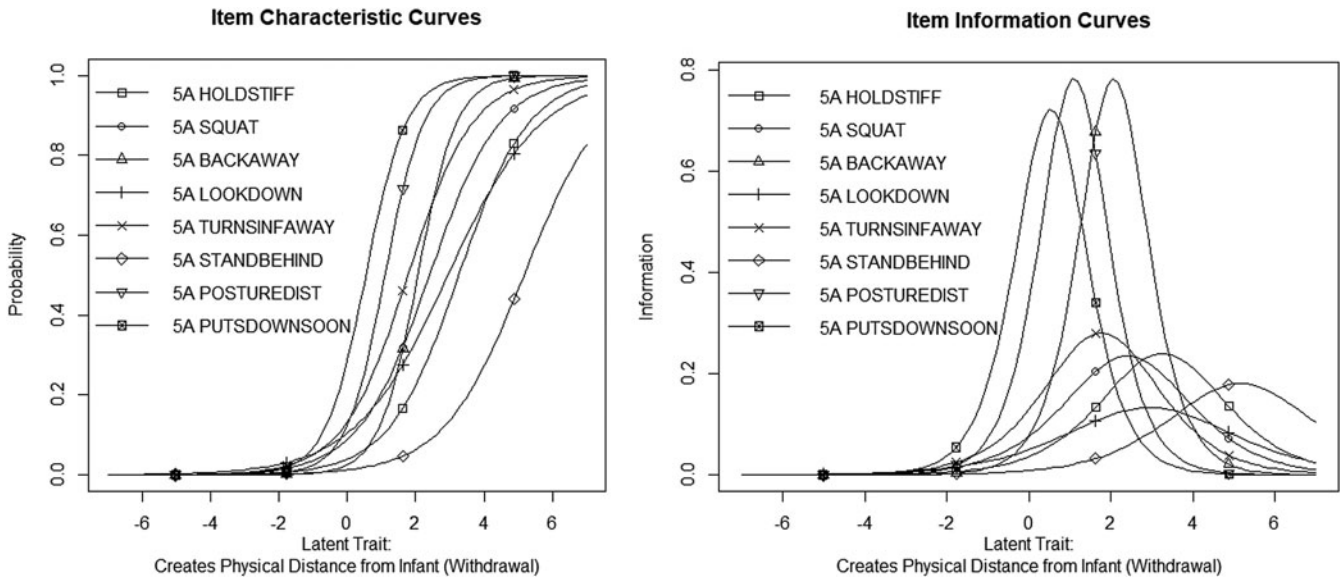

Figure 5. Item characteristic and item information curves for eight indicators of AMBIANCE subdimension 5A: "creates physical distance from infant." Full item code descriptors are provided in Table 1. Chosen indicators were selected to facilitate visual interpretation.

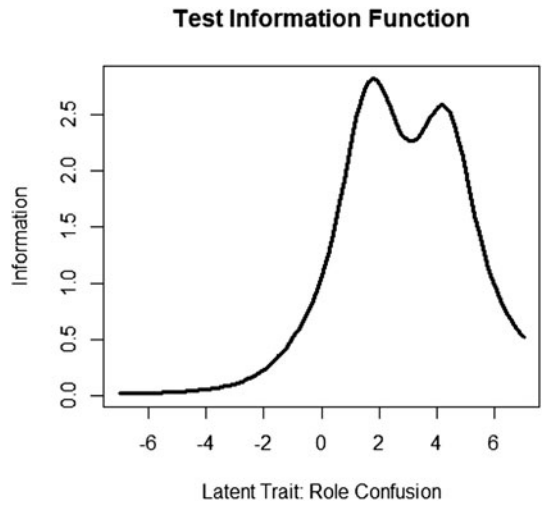

Figure 6. Test information function for AMBIANCE subdimension 2A: "role confusion." Note that the test information function is reflective of the total set of AMBIANCE indicators for a given latent trait.

0.50 to 1.00; Ondersma, Chaffin, Mullins, & Lebreton, 2005).

ROC analyses for the AMBIANCE IRT-based 45-item set (see Figure 7) showed that the AUC value and standard error was significant ($p < .01$), with an AUC of 0.85. These results indicate that the 45-item set showed good diagnostic accuracy with respect to coder classifications of maternal disrupted communication status using the complete AMBIANCE item set and coding protocol.

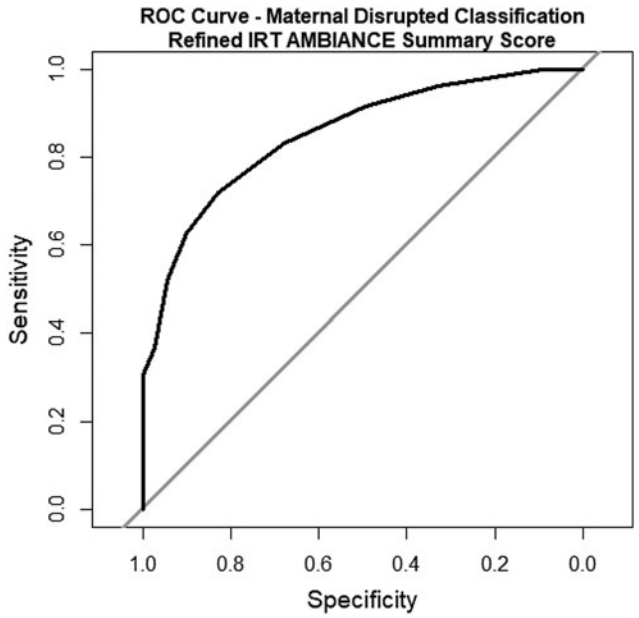

Figure 7. Receiver operating characteristic (ROC) analysis curve for the item response theory (IRT) based refined AMBIANCE summary score in detecting maternal disrupted classification status as defined using the AMBIANCE protocol. Area under the curve = 0.85, $p < .01$.

Second, we examined whether the full and the reduced AMBIANCE item sets demonstrated similar positive associations with infant attachment disorganization. Mothers of disorganized infants demonstrated significantly higher scores on the refined 45-item AMBIANCE measure ($M = 5.67$, $SD = 3.42$) compared to mothers of nondisorganized infants ($M = 4.45$, $SD = 3.40$), $t\ (254) = -2.50$, $p < .02$, $d = \sim 0.36$. These results were quite similar to those using the full AMBIANCE item set (disorganized $M = 14.11$, $SD = 7.27$; nondisorganized $M = 11.48$, $SD = 7.28$), $t\ (254) = -2.52$, $p < .02$, $d = \sim 0.36$; see Figure 8 for raw data, descriptive statistics, and inferential statistics plot of refined 45-item summary scores by attachment disorganization. Thus, scores obtained with the IRT-based 45-item set demonstrated virtually identical associations with infant attachment disorganization as did the full AMBIANCE summary score, with effect size magnitudes (i.e., strength of associations) intermediate in degree between small and medium-sized effects (Cohen, 1992).

To assess discriminative validity, we also compared the magnitude of the association between the AMBIANCE 45-item set and infant attachment disorganization to the magnitude of the association between maternal sensitivity (assessed at infant ages 6, 15, 24, and 36 months) and infant disorganization at 15 months of age in the SECCYD subsample ($n = 197$). We tested this comparison to assess whether the refined 45-item summary measure of disrupted communication would show a stronger relation to infant disorganization

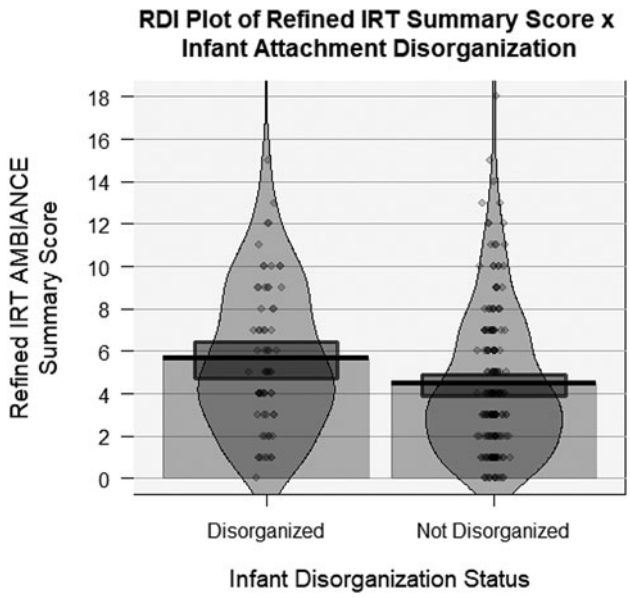

Figure 8. Raw data, descriptive statistics, and inferential statistics (RDI) plot of significant mean difference in item response theory (IRT) based refined AMBIANCE summary scores between disorganized and not disorganized infants. Note that the RDI plot contains four main elements that allow for greater empirical resolution into the patterning of data (relative, e.g., to bar plots): points reflected by darkened dots reflect raw data points; the vertical shaded bar reflects central tendencies; the bean reflects a smoothed density; and the shaded rectangle reflects an inference interval (e.g., frequentist confidence interval, as in this example).

than maternal sensitivity. As noted earlier, meta-analytic data have generated a mean effect size of $r = .10$ for the association between maternal (in)sensitivity and disorganized attachment (van IJzendoorn et al., 1999) and $r = .35$ for the association between disrupted communication and disorganized attachment (Madigan et al., 2006). Both the full ($r = .22$) and the refined ($r = .23$) AMBIANCE scores were significantly ($p < .01$) associated with infant attachment disorganization in the SECCYD subsample, whereas maternal sensitivity was not ($r = –.06$, *ns*). Note that the association between maternal sensitivity and infant disorganization in this subsample of SECCYD data was virtually identical to the zero-order association using the full SECCYD cohort ($r = –.05$; Haltigan & Roisman, 2015). We also assessed the reliability of the difference between the correlation of disorganization with sensitivity and the correlation of disorganization with the refined AMBIANCE using the Fisher r to z transformation. The correlation of the refined (45-item) AMBIANCE summary measure with infant disorganization was significantly stronger than the association of maternal sensitivity with infant disorganization ($z = 2.90$, $p < .01$). In a final analysis, we also examined the 9-point continuous attachment disorganization scale. These analyses yielded a similar pattern of effects as those using the categorical measure of infant disorganization described above (full AMBIANCE $r = .25$, 45-item refined AMBIANCE $r = .24$, both $ps < .01$; maternal sensitivity $r = –.11$, *ns*; for the difference between refined AMBIANCE and maternal sensitivity associations with continuous infant disorganization scores, see the Steiger, 1980, test for dependent correlations, $t = 3.18$, $p < .01$, case A).

Discussion

To the best of our knowledge, this is the first investigation to attempt to extend the IRT methodology to the study of observed maternal caregiving behavior. Using IRT modeling, we examined the functioning of the individual behavioral items included in the AMBIANCE coding system to assess which items were the most informative indicators of the 15 latent dimensions of disturbed communication. Identifying the most informative indicators is important to honing our understanding of disrupted maternal communication, as well as a critical first step toward the related goal of developing an efficient screening instrument for clinical use. The results of our latent trait analyses for each of the 15 AMBIANCE subdimensions provided the necessary psychometric information to cull a refined set of 45 indicators that were maximally informative. Furthermore, the results of our 2PLM models suggested that the identified items were moderately to strongly related to their underlying latent traits and were reliably measuring the severe end of those latent traits.

In addition, the latent trait analyses yielded valuable insights into the defining characteristics of each dimension. For example, both "threatens to cry" and "prioritizes own [parent] needs over infant needs" were identified by the latent trait analyses as possessing high discriminatory value at the severe end of the role confusion latent trait. In the current sample, these items are indexing codable behaviors that carry maximal information regarding the broader theoretical construct of role confusion (Macfie, McElwain, Houts, & Cox, 2005; Mayseless, Bartholomew, Henderson, & Trinke, 2004; Sroufe, Jacobvitiz, Mangelsdorf, DeAngelo, & Ward, 1985). Similarly, "exhibits highly vigilant posture in presence of infant" and "Exhibits sudden loss of affect," each of which emerged as possessing relatively high discriminatory value on the subdimensions measuring fearful and disoriented behavior, respectively, are central to theoretical constructs regarding the frightened or dissociative parental behaviors thought to contribute to infant disorganization (Main & Hesse, 1990). Frightened or frightening parental behavior is hypothesized to be a key mechanism mediating the link between parental unresolved trauma on the Adult Attachment Interview and attachment disorganization in the child. Thus, these analyses inform a more molecular understanding of the aspects of parental frightened or frightening behavior that may be central to the construct. Similarly, items identified across the physical communications latent trait, including "attempts to grab infant," "turns infant's head," and "tickles infant when infant resists" offer insight into the core defining features of specific parenting behaviors that may underlie current conceptualizations of the construct of parental intrusiveness in the context of early parent–child interaction (Egeland, Pianta, & O'Brien, 1993; Haltigan, Leerkes, Supple, & Calkins, 2013; Lyons-Ruth, Alpern, & Repacholi, 1993). What is striking to us about these particular behaviors is their physically invasive, and coarse kinesthetic nature. Finally, the very different items on the withdrawing behavior dimension, such as "backs away from the infant," "puts infant down too soon before cue from infant," and "adopts posture designed to keep infant at a distance" index a maternal stance that has been differentially associated with poor self-regulation in late adolescence, including borderline features, suicidality, and antisocial personality disorder (Lyons-Ruth et al., 2013; Shi, Bureau, Easterbrooks, Zhao, & Lyons-Ruth, 2012). The identification of such particularly informative disrupted behaviors for each subdimension offers much more specificity to our conceptualizations of both the threatening and emotionally neglecting caregiving contexts that have been highlighted as potential contributors to risk for infant and child psychopathology and maladaptation, as well as altered trajectories of brain functioning (Lyons-Ruth, Pechtel, Yoon, Anderson, & Teicher, 2016; Sheridan & McLaughlin, 2014).

The results of the IRT models thus provide important points of basic and applied departure for further empirical work exploring central features of disturbed caregiving. It is also notable that initial qualitative review of the identified items by experienced clinicians and AMBIANCE coders has confirmed that the identified behaviors were all behaviors that were theoretically and clinically central to the construct being coded. This initial qualitative review must be further confirmed by studies on clinical utility, but it is important that the item set selected by the IRT analyses was felt to be clinically rich and informative on initial review.

Further, analyses exploring the initial construct validity of the refined 45-item set suggested that it performed similarly to the full AMBIANCE item set in relation to key validity criteria. First, the refined 45-item set demonstrated good diagnostic accuracy with respect to discriminating between disrupted and not disrupted maternal classifications generated from the full AMBIANCE coding protocol. Thus, the results of our ROC analyses indicate that the IRT models were performing well in identifying highly discriminating items at the more severe end of the latent traits, in somewhat the same way that coder judgment would be used to capture severity on the overall rating scale for level of disrupted communication. Second, in relation to infant attachment disorganization, the refined AMBIANCE 45-item set generated associations similar to those using the full AMBIANCE item set, for both continuous and categorical measures of disorganization. Third, in the SECCYD subsample, both the full AMBIANCE and the smaller IRT-refined item set showed discriminant validity in relation to maternal sensitivity, in that they were significantly more strongly associated with infant disorganization than was the sensitivity measure. This latter finding is consistent with meta-analytic evidence that disrupted maternal behavior demonstrates stronger associations with infant disorganization (Madigan et al., 2006) than does maternal (in)sensitivity in normative-risk populations (Haltigan & Roisman, 2015; van IJzendoorn et al., 1999).

Limitations

As we have noted, the use of latent trait analyses with dichotomous items poses several modeling challenges, in particular when there is a large preponderance of extremely low base-rate items. To address this issue and mitigate the possibility that extreme sampling properties may unduly influence the accuracy and precision of parameter estimates, we utilized bootstrapping procedures to generate latent trait parameter estimates, presenting trimmed mean estimate values as our point estimates for all relevant parameters. Although all latent trait models converged, the direction and magnitude of parameter estimates from 4 of the 15 models (1A, 1B, 2B, and 4C) should be considered especially provisional given that these models did not reliably converge at a consistent log-likelihood value. However, we have included results for these subdimensions because, given the absence of other available item-level frequency data, we considered it important to include parameter estimates for all subdimensions of the coding system, rather than omitting 4 clinically important subdimensions entirely. Omitting subdimensions considered clinically and conceptually important from the refined 45-item set would have substantively changed the meaning of the refined set in relation to the full AMBIANCE system. Moreover, by providing these estimates while also noting the issue of their imprecision (Maxwell, 2004), we allow for comparison with future work examining the latent trait structure of the AMBIANCE coding system and these subdimensions in particular.

It is also possible that, in samples with higher rates of occurrence of a given disrupted caregiving behavior, latent trait models for these 15 subdimensions would yield different absolute magnitudes for IRT parameter estimates, as well as different relative rankings of AMBIANCE indicators with respect to the magnitude of their discrimination parameter estimates. As such, pending replication efforts, we anticipate there may be some fluidity in the specific indicators from these subdimensions that ultimately emerge as those consistently demonstrated to be the most strongly related to their underlying latent traits. Similarly, the somewhat ambiguous findings regarding the unidimensionality of AMBIANCE subdimensions 4A and 5A, indexing physical intrusiveness and physical withdrawing behaviors, respectively, warrant additional research investigating their dimensionality before firm conclusions regarding their latent trait structure are suggested.

Related to the above points, it is important to note that relatively large discrimination and location parameters for some of the latent trait models are not surprising, given the rare nature of the behaviors and the consequent sparse number of observations for each item. Consequently, their absolute magnitudes should be considered cautiously in view of the nature of the data. It is likely that the absolute magnitude of these discrimination parameters may be smaller in samples where there are more events per variable for particular AMBIANCE indicators. However, given that our chief aim was not definitive parameter generalization to the population but data refinement and reduction by selecting the most informative items in the larger set, different levels of precision may be acceptable based on the nature and intent of the investigation (Edelen & Reeve, 2007). Parameter estimates need not be accepted uncritically as highly precise to argue that their relative magnitude, and thus their relative importance for measuring the disrupted maternal behavior latent trait, reflect the best available information concerning these disrupted maternal behavior dimensions. To this end, all existing AMBIANCE data sets with item-level data that we are aware of were included in these analyses. Although serving as an empirical starting point, it will be critically important to assess the replicability of these parameter estimates and continue to refine them as new data is acquired.

A final limitation to stress is that this refined set of indicators is not yet ready for widespread use in research settings. The presence of a larger pool of disrupted behaviors, as currently described in the coding manual, may be important in helping coders to develop a template of the dimension being coded in a way that the inclusion of only three items could not. This is an empirical question that needs to be addressed. A refined AMBIANCE measure would also need to be assessed in relation to its association with a variety of other relevant maternal risk factors and child outcomes. While the larger AMBIANCE measure now has a sound track record of reliability, stability over time, and convergent validity with relevant maternal and child constructs, the reduced measure has not yet received this level of scrutiny.

Despite these issues, we believe these analyses resulted in a well-chosen set of indicators, with good initial validity, that warrant taking further steps toward a more efficient measure of maternal disrupted communication for use in clinical set-

tings. We might expect such a reduced measure to have somewhat less precision and prediction than the full measure, while still offering a more standard clinical training format and yielding information with higher validity and specificity than the varying judgments of individual clinicians. Thus, a next important step in moving toward a clinically efficient instrument will be to evaluate the reliability, validity, and prediction of the reduced item set, and any associated training procedures, when used in clinical settings.

Future directions

Results of the current work also offer a number of potential future directions for clinicians and researchers interested in studying disrupted caregiving behaviors and their predictive significance for child maladaptation. Examinations of the convergent and predictive validity of the refined AMBIANCE item set in relation to additional domains, such as maternal risk factors and measures of child psychopathology, are now needed in independent samples. In addition, additional independent large-sample studies using *item-level* AMBIANCE data are especially needed to continue to refine our understanding of the latent structure of disturbed maternal communication. Because coding systems designed to quantify disrupted parental communication often require extended training and are labor intensive to code, the development of large enough data sets at the item level presents a challenge. In future work, it will be important for researchers to develop consortia that allow for the pooling of data sets, to allow more granular analysis of the process of parent–child communication and to yield more stable parameter estimates of underlying constructs. Moreover, with appropriate sample sizes, modeling the five higher order AMBIANCE dimensions as a function of lower order subdimensions and their indictors will also be an important objective for future research investigating the latent structure of disrupted parenting.

From a measurement science perspective, new developments in IRT mixture modeling (Finkelman, Green, Gruber, & Zaslavsky, 2011; Wall, Park, & Moustaki, 2015) and Bayesian IRT modeling (Swaminathan et al., 2003) offer additional strategies to deal with low-base rate behaviors, which presents challenges when modeling the latent structure of disrupted caregiving traits. In addition, it will be important to assess whether particular disrupted caregiving behaviors show the same degree of severity and discrimination on their relevant latent traits when considering parent–child interaction at different child ages, across different geographic subsamples, and across other demographic characteristics (e.g., sex and ethnicity; Haltigan et al., 2014). Differential item functioning in the IRT framework is well suited to address these issues (Osterlind & Everson, 2009). The full AMBIANCE coding protocol has been used successfully among mothers with children ranging in age from 4 months to 7 years. However, the item-level data used in the IRT analyses here were only available for mothers of children aged 12–54 months.

The need for the translation of research to practice within developmental psychopathology is crucial for the discipline to realize its full potential. The present work represents an initial step in the development of shorter protocols, which are more efficient for clinical and applied use. From a clinical perspective, it will be important to determine whether the specific items identified herein can be coded accurately in real time, which is a practical requirement of many agencies working with high-risk families. This work is currently under way in a group of collaborating clinical agencies. Methodologically, there is great potential for the IRT framework to bridge research and clinical objectives. Understanding the salience of discrete caregiving behaviors for the developing human organism in the earliest years of life is of immense importance. We believe the current work represents an important step in a larger enterprise aimed at identifying the neurobiological and psychological signatures of early social–environmental experiences. For example, this work is especially relevant to recent research conceptualizing childhood adversity around dimensions of deprivation and threat (Sheridan & McLaughin, 2014; Teicher, Samson, Anderson, & Ohashi, 2016), because it offers a fine-grained examination of disrupted caregiving behaviors characterizing both maternal withdrawal and maternal intrusiveness, which may be thought of as downward extensions of concepts of deprivation and threat to infancy and early childhood. The behaviors identified in these analyses, then, are anchored in strong conceptual frameworks from which to launch focused investigations of the effects of early social experience on neural and physiological development.

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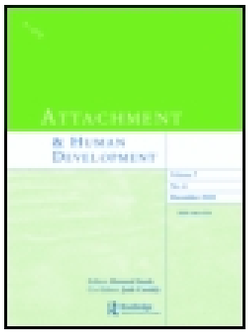

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Growing points in attachment disorganization: looking back to advance forward

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ABSTRACT

In this special issue paper we reflect on the next generation of attachment research with a focus on disorganization, a central but still poorly understood topic in this area. We suggest that progress will be facilitated by a return to attachment theory's evolutionary roots, and to the emphasis on biological function that inspired Bowlby's original thinking. Increased interdisciplinary cross-fertilization and collaborations would enable novel and generative research on some of the long-standing questions surrounding attachment disorganization. Accordingly, we present an agenda for future research that encompasses contributions of modern ethology and neurobiology, novel hypotheses based on the concept of adaptive decanalization, connections with neurodevelopmental vulnerability and risk for mental disorders such as schizophrenia, and the possibility of sex differences in the behavioral manifestations of attachment disorganization. We believe that these avenues of theory and research offer exciting potential for innovative work in attachment disorganization in the years ahead.

KEYWORDS

Bowlby; disorganized attachment; canalization; ethology; evolution; neurodevelopmental disorders; psychosis

As so artfully chronicled in *Cornerstones of Attachment Research* (Duschinsky, 2020), the first two generations of attachment researchers have provided an exceptionally generative body of research and scholarship. In the spirit of foundational "Growing Points" monographs (Bretherton & Waters, 1985; Waters et al., 1995) that took stock of attachment theory and method while looking ahead, here we offer our answer to Duschinsky's question concerning what genre of story the third generation of attachment scholars finds themselves in. In our view, the field is ready for a gripping tale of mystery, one in which emerging perspectives will raise new (and old) questions and prompt a new phase of theoretical exploration. One of the most pressing tasks we envision is the long overdue reintegration with evolutionary biology and psychology, in line with Bowlby's original thinking regarding attachment's fundamental role in survival, and – ultimately – in the promotion of biological fitness (Bowlby, 1969; Main, 1979; Simpson & Belsky, 2016).

Over time, the evolutionary foundations of Bowlby's theory and the ethological observational approach to the identification infant attachment patterns (e.g. Ainsworth et al., 1978; Main & Solomon, 1990) have faded from mainstream attachment scholarship. As Chisholm (1996) remarked more than twenty years ago, attachment

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research has strayed from its "early safe haven in evolutionary theory;" as a result, our functional understanding of the basic patterns of attachment has remained limited. This is especially true of atypical and disorganized/disoriented patterns (i.e. "D"; Main & Solomon, 1990), despite their likely evolutionary importance as correlates of atypical parental behaviors and elevated environmental risk. Surprisingly, evolutionary analyses of individual differences in attachment have focused almost exclusively on Ainsworth et al.'s (1978) original organized attachment patterns (i.e. avoidant [A], secure [B], and resistant [C]), and the conceptually related dimensions of anxiety and avoidance (Belsky et al., 1991; Chisholm, 1996; Del Giudice, 2009; Szepsenwol & Simpson, 2019). Disorganization has remained virtually unexplored by evolutionarily-minded scholars, except as a dysfunctional outcome of conflicts between attachment and other motivational systems (Liotti, 2016).

Developmental research on disorganized attachment also remains saddled with theoretical gaps and unanswered questions, despite the remarkable amount of work carried out in the last three decades (Duschinsky, 2020). The idea that the disorganized classification may contain multiple subtypes with potentially distinct etiologies and functional underpinnings was briefly considered in the early years (Carlson et al., 1989a; P. Crittenden, 1999; Main & Solomon, 1990) of disorganization research, in particular by Lyons-Ruth and colleagues (Lyons-Ruth et al., 1999, 1991, 1989). However, with the exception of Lyons-Ruth and colleagues who have continued to pursue this strand of work (e.g. David & Lyons-Ruth, 2005; Lyons-Ruth et al., 2013, 2016), this issue has received relatively little empirical scrutiny since, even though a few recent studies (e.g. Padrón et al., 2014) indicate the existence of meaningful heterogeneity (Green & Goldwyn, 2002; Solomon et al., 2017). The incomplete state of current knowledge is further illustrated by the case of dissociation. The expectation of an association between infant disorganization and later dissociative symptoms is supported by both theory (Liotti, 1992, 2016) and some evidence (e.g. Carlson, 1998), with additional studies finding higher dissociation scores in adolescents and adults with unresolved states of mind (e.g. Schuengel et al., 1999; West et al., 2001); but findings from recent large-sample work failed to support the infant disorganization – dissociation link (Haltigan & Roisman, 2015), suggesting it should not be reified as a core claim of attachment theory.

Precisely because it is still puzzling in a number of ways, disorganization may prove an ideal bridge between mainstream attachment and evolutionary scholars. On the one hand, a biological perspective can be an invaluable guide to generate new ideas and hypotheses on the nature of attachment disorganization. On the other hand, existing evolutionary models of attachment are clearly incomplete, and need to be extended and revised to integrate the phenomenology of disorganization. A crucial task for the third generation is to deepen the field's understanding of the *variability* in attachment behaviors – especially those observed in infants classified as disorganized – by returning to the study of their potential evolutionary logic, in the footsteps of Main (1979, 1990) and others (Crittenden & Ainsworth, 1989). In what follows, we articulate a research agenda that synthesizes some of Bowlby's ethological and evolutionary insights in the development of attachment theory with new ideas and findings from the biological sciences.

Ethological perspectives on disorganization

It is seldom fully appreciated that the development of a coding system for previously unclassifiable infants seen in the Strange Situation procedure (SSP; Ainsworth et al., 1978) was undertaken against the backdrop of not only Bowlby's early ethological thinking around conflicting behavioral systems (cf. Bowlby, 1969, pp. 224–228; Green & Goldwyn, 2002), but also a painstaking observational approach informed by biological ideas (cf. Main et al., 2011, p. 435; Main & Solomon, 1990, p. 39). Indeed, Main's original motivation for pursuing work on the anomalous behaviors she observed in her dissertation sample was due in large part to her interest in ethology, including observations of odd-appearing behavior of animals in situations of motivational conflict (Main et al., 2011).

Among the disorganized phenomena that have captured the attention of attachment researchers, the most clear-cut exemplars of ethologically relevant behaviors are freezing, stilling, and slowed movements. In nonhuman primates, stilling is often observed in ambiguous or threatening communicative contexts with conspecifics (Hinde, 1966). Evolutionary biologists describe freezing (or "attentive immobility") as a functional response characterized by the rapid simultaneous activation of the sympathetic and parasympathetic systems, which allows the individual to quickly suppress current activities (thus avoiding detection), monitor the source of danger with heightened attention, and prepare for fight or flight if necessary (Hagenaars et al., 2014; Roelofs, 2017). Similarly, stilling and freezing behaviors for some infants and very young children may play a functional protective role, affording them time to form a "best estimate" of the parent's current state and intentions, especially in high-risk contexts in which caregiver behavior is often highly ambiguous and/or potentially harmful (see P. Crittenden, 1999).

The biological understanding of freezing has made considerable progress since Main's initial observations. For example, there is evidence that freezing is inhibited by GABA and potentiated by cortisol (Hagenaars et al., 2014; Nijenhuis et al., 1998). Of particular interest, serotonergic projections in the brain suppress fight-flight behaviors while promoting freezing (Graeff, 2004; Paul et al., 2014; Roelofs, 2017); hence, frequent occurrences of freezing should be associated with elevated serotonergic activity. If supported, this hypothesis would point to novel connections between freezing in the SSP and later psychopathology. Intriguingly, a longitudinal study by Niermann et al. (2019) found that patterns of internalizing symptoms across adolescence were predicted by a long duration of freezing in infancy, but also by the *absence* of freezing (in response to a surprising stimulus). Regardless of how the serotonergic hypothesis stands up to empirical scrutiny, it highlights the idea supported by contemporary evidence that neurobiological mechanisms involved in freezing may suggest a functional basis for the existence of distinct subtypes within the D classification.

As a second example, consider an infant's hand-to-mouth gesture on reunion with the parent with a clearly confused or wary expression, which is considered a direct index of disorganization. A first interpretation of this behavior is that the infant may have learned to cover their mouth to stifle cries of distress in a rejecting or insensitive caregiving environment, so as to avoid further rejection and hence maximize safety and availability of the caregiver (P. Crittenden, 1999; Hesse & Main, 2000; Landa & Duschinsky, 2013). Somewhat related to this interpretation, there is considerable evidence that, in humans as well as other primates, gestures are not only communication tools but also play multiple

cognitive roles for the individual who produces them (Cartmill et al., 2012). For example, non-human primates have been observed performing non-functional gestures during complex problem-solving tasks; these gestures are thought to be an indication of the primate exploring different options before settling on a solution (Cartmill et al., 2012).

Analogously, seemingly out-of-context behaviors and incoherent action sequences in disorganized infants may indicate the rapid exploration of alternative responses when confronted with an unpredictable caregiver, adding cognitive depth to the behavioral focus of classic ethological accounts. The fact that sequential and simultaneous blends of (contradictory) avoidant and resistant behaviors are observed more often in infants exposed to high levels of abuse and neglect (Cicchetti & Barnett, 1991; Cyr et al., 2010; Green & Goldwyn, 2002; P. M. Crittenden, 1985; Van Ijzendoorn et al., 1999) support this possibility and underscore the importance of developing novel approaches to analyzing the sequencing and content of disorganized behaviors.

Disorganization as adaptive decanalization?

The functional underpinnings of disorganized behaviors may be further illuminated with the biological concepts of *canalization* and *phenotypic integration* (Armbruster et al., 2014; Debat & David, 2001; Murren, 2012; Pigliucci & Preston, 2004). The notion of canalization was originally introduced by Waddington (1942, 1957) and Bowlby (1973) drew on Waddington's work to ground his theory in a sophisticated understanding of developmental biology. Decades later, research on plasticity and canalization has made tremendous progress (e.g. Debat & David, 2001; Hallgrimsson et al., 2019; Siegal & Bergman, 2002), and attachment scholars may once again benefit by exploring the many potential connections with this area of research.

Canalization is a ubiquitous feature of developmental processes: by buffering development against genetic and/or environmental perturbations, canalization mechanisms ensure that the organism's traits will demonstrate robustness and develop reliably. Consequently, canalized traits show constrained variation across genotypes and environments (see Figure 1(a)). Phenotypic integration occurs when distinct traits of an organism are not free to vary independently but show specific correlation patterns. Thus, integration can be understood as a multivariate analogue of canalization, as it constrains *independent* variation among linked traits. Canalization can be implemented by specialized mechanisms, or evolve as an emergent property of complex developmental systems, which involve extensive redundancy and multiple layers of regulation (Badyaev, 2005; Geiler-Samerotte et al., 2019; Hallgrimsson et al., 2019).

Canalization and integration are necessary to enable robust, adaptive trait expression. However, they also carry important costs: they inevitably limit an organism's plasticity, reduce the range of potential responses to the environment, and suppress the expression of potentially adaptive genetic variation (e.g. novel mutations). Thus, canalization/integration processes can themselves evolve to show adaptive plasticity, and produce different patterns of variation and covariation in different kinds of environments (Pigliucci & Preston, 2004). As a rule, highly stressful conditions tend to reduce canalization and increase phenotypic variability (Debat & David, 2001; Rowiński & Rogell, 2017). The literature on integration is less consistent, and different studies have found both stronger and weaker correlations under stress (e.g. Lea et al., 2019; Merrill & Grindstaff, 2018).

A plausible interpretation of these findings is that mild challenges can be successfully addressed with the available responses, leading to reinforce existing correlations among traits ("more of the same"). In contrast, severe stress implies that an organism is experiencing extreme unpredictability and/or uncontrollability, and hence that the available responses are not functioning as intended (Del Giudice et al., 2018a). In such conditions, reduced integration allows the organism to explore novel solutions – including responses that would be too costly, counterproductive, or otherwise detrimental in a more benign environment (Badyaev, 2005). For simplicity, we use *decanalization* as a shorthand for the release of variation and loosening of trait correlations under severe stress (see Figure 1(b)). Adaptive decanalization can be viewed as a risky, last-resort adaptive strategy that pushes the envelope of the organism's response in an attempt to regain control over the environment. The concept of adaptive decanalization corrects the unwarranted but common assumption that a loss of canalization is necessarily maladaptive and/or pathological (e.g. Gibson, 2009).

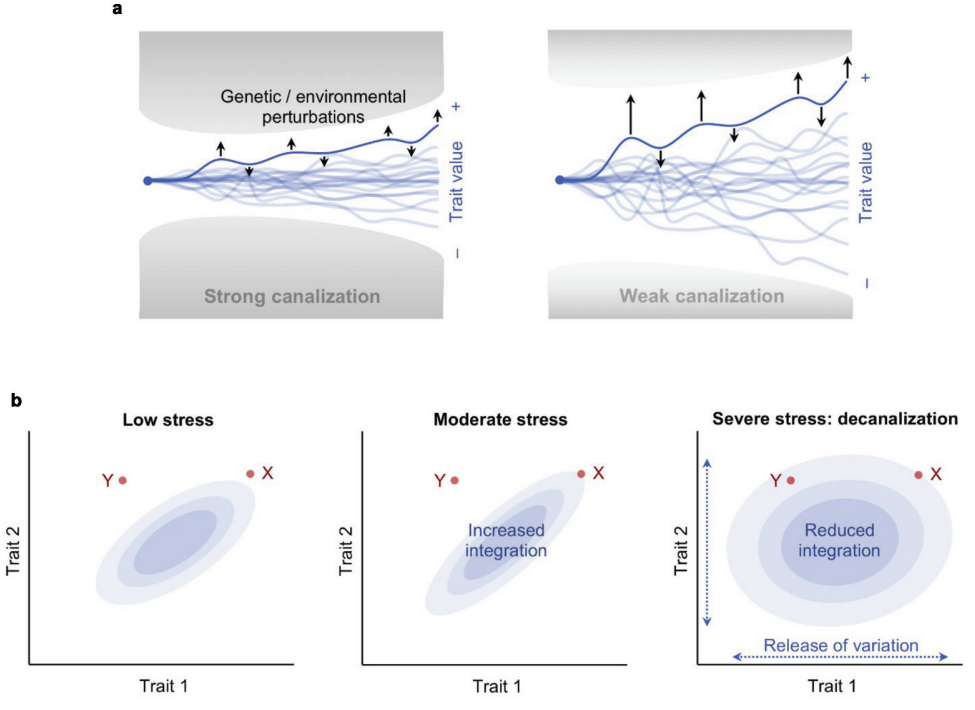

Figure 1. Schematic illustration of canalization and decanalization. (a) Strongly canalized traits (left panel) are buffered against genetic and environmental perturbations; as a result, they tend to develop within a narrow range and show restricted variation. Weakly canalized traits (right panel) show larger responses to environmental and genetic factors and tend to be more variable. Each line in the figure shows the developmental trajectory of an individual. Arrows represent genetic and developmental perturbations that tend to either increase (up) or decrease (down) the value of the trait. (b) Exposure to moderate stress during development tends to increase phenotypic integration (center panel). Stronger correlations among traits reinforce the organism's existing responses by promoting the cohesive phenotypic expression of specific patterns of traits (e.g. point X). In contrast, severe stress tends to release trait variation and reduce phenotypic integration (decanalization; right panel). Decanalization allows the organism to express novel, less constrained patterns of traits (e.g. point Y), which would normally be detrimental but may prove effective against the present threat.

The biology of decanalization offers a novel, intriguing perspective on the phenomenology of disorganized attachment – most notably the simultaneous or sequential juxtaposition of avoidant and resistant behaviors. In some instances of disorganization, seemingly contradictory behaviors are expressed together or in sequence, often accompanied by unusual or out-of-context responses. This is usually interpreted as a maladaptive breakdown of attachment strategies; the alternative possibility we propose is that these behavioral patterns may reflect adaptive decanalization, in response to extreme stress and unpredictability in the relationship with the caregiver. From this vantage point, it is noteworthy that mixed avoidant/resistant behaviors are especially common in maltreated infants and children (see above). When organized attachment behaviors systematically fail to reduce stress and prevent actual or perceived threats from the caregiver, decanalization may allow the child to "explore" alternative coping options and (hopefully) arrive upon behavioral combinations that reduce the heightened activation of the attachment system. It is worth restating that "risky" strategies such as decanalization can be biologically adaptive, despite their costs, if they are better than the alternatives *on average* (Frankenhuis & Del Giudice, 2012).

From a neurobiological standpoint, reduced serotonergic activity is a plausible candidate mechanism for behavioral decanalization. Serotonin promotes behavioral persistence and response inhibition, especially in response to threatening and aversive events (e.g. Cools et al., 2011; Moore & Depue, 2016; Moran et al., 2018). Reduced serotonergic tone should lower the sequential coherence of behavioral strategies, and – by releasing inhibitory constraints – should permit a broader range of responses in challenging situations (such as the SSP). Intriguingly, this could possibly explain why chronically undernourished infants show high and persistent levels of mixed avoidant/resistant behaviors (both sequentially and simultaneously), without other disorganization indices such as freezing, interruptions, and fear displays (Valenzuela, 1990; Waters & Valenzuela, 1999). Serotonin is synthesized from tryptophan, an essential amino acid that can only be obtained from the diet; hence, chronically undernourished infants are very likely to experience serotonin deficiency. The diet-related (i.e. food deprivation) suppression of serotonergic activity could potentially explain some variation in the anomalous frequency of mixed avoidance/resistance, even in absence of maltreatment or especially threatening caregivers. Moreover, serotonin deficiency could explain the surprising absence of freezing and stilling in undernourished D infants (Waters & Valenzuela, 1999).

The adaptive decanalization hypothesis is of course speculative, but it can facilitate new ways of thinking about specific kinds of disorganized behaviors. The concept of decanalization can be valuable even if the "adaptive" part of the hypothesis fails to be supported, or is relevant only to a subset of cases (e.g. behavioral decanalization in undernourished children). The adaptive decanalization hypothesis complements Bowlby's original use of canalization to explain the development of organized attachment behaviors, while preserving the idea that both the organized and the later-identified non-organized patterns (P. M. Crittenden, 1985; Main & Solmon, 1986) arise from the action of functional evolved mechanisms.

Links with neurodevelopmental vulnerabilities

The phenotypic resemblance between some aspects of infant disorganization and behaviors typically found in neurodevelopmental disorders (e.g. freezing, stilling, atypical

postures and behavioral stereotypies) was recognized early on by Main in her coding of samples of children diagnosed with autism (Capps et al., 1994; Rozga et al., 2018). While there are codified procedures to account for this overlap (e.g. Pipp-Siegel et al., 1999), in practice they are utilized only when examining samples with known neurodevelopmental disorders or risk for such disorders (e.g. autism spectrum disorders, ASDs; e.g. Haltigan et al., 2011).

This overlap may have broader implications for understanding the etiological and phenotypic aspects of infant disorganization. Specifically, it is likely that one or more unique subpopulations of disorganized infants exist which are characterized by trait vulnerability to various neurodevelopmental conditions (Barnett et al., 1999) or neurobehavioral atypicalities (e.g. Padrón et al., 2014; Spanger et al., 2009; Spangler, 2019; Spangler et al., 1996), including schizophrenia spectrum disorders (SSDs). There is considerable evidence that both SSDs and certain variants of ASDs (particularly low-functioning syndromes) are strongly influenced by early insults and deleterious mutations, including chromosomal abnormalities and rare/de novo copy number variants (CNVs; Keller, 2018; Ronemus et al., 2014).

Surprisingly, the links between attachment disorganization, dissociation, and risk for schizophrenia have remained virtually unexplored (although see: Liotti & Gumley, 2008), despite evidence of associations between unresolved adult attachment classifications and disorders on the psychosis spectrum (Dozier, 1990; MacBeth et al., 2011; Tyrrell et al., 1999). Moreover, a high prevalence of unresolved classifications has been found in individuals with borderline personality disorder (BPD; Macfie et al., 2014) – a condition with strong genetic overlap with psychosis and substantial comorbidity with disorders in the psychosis spectrum (Witt et al., 2017). For example, Macfie et al. (2014) found that mothers with BPD show higher scores on the preoccupied/unresolved dimension of the AAI, which predicted the frequency of children's narrative representations conceptually relevant to infant disorganization (e.g. incongruent child and self/fantasy confusion). These findings linking BPD or borderline personality features with early caregiving, infant disorganization, dissociation, and unresolved classifications on the AAI are consistent with prior work connecting these phenomena (e.g. E. A. Carlson et al., 2009; Khoury et al., 2019; Lyons-Ruth, 2008; Lyons-Ruth & Brumariu, 2020; Lyons-Ruth et al., 2007).

Taken together, this corpus of work suggests the possibility of a "schizotypal" class of disorganized infants with increased risk for later SSDs and other disorders on the psychosis spectrum (e.g. bipolar disorders); in dimensional models of psychiatric nosology, these disorders are subsumed under the psychosis and thought disorder spectra (see Kotov et al., 2020, 2017). Despite its clinical distinctiveness, BPD is also genetically linked to the psychosis spectrum; thus, investigating the links between attachment and psychosis may illuminate previous research on borderline phenomena in unexpected ways.

From an evolutionary perspective, subthreshold schizotypal traits (e.g. magical thinking and perceptual distortions) can be viewed as "risky" phenotypes that may increase genetic fitness through enhanced mating success, but also lead to catastrophic dysfunctions (i.e. severe psychotic symptoms) in individuals who carry additional genetic or environmental vulnerabilities (Del Giudice, 2017; Shaner et al., 2004). Of note, the sexual selection model of schizophrenia is conceptually linked to the decanalization hypothesis we presented above: according to the model, schizotypal traits increase phenotypic variance by amplifying the positive/negative effects of multiple genetic and

environmental factors. Decanalization has been repeatedly proposed as an important etiological process in SSDs and other neurodevelopmental disorders (e.g. Burrows & Hannan, 2013; McGrath et al., 2011), under the default assumption that decanalization is maladaptive. Both the sexual selection model of schizophrenia and our hypothesis on disorganization consider the possibility that decanalization may function as a risky, yet potentially beneficial strategy. Other evolutionary approaches have focused on the potential role of mismatches between ancestral and modern environments; an example is the unprecedented exposure to out-group members, which may be perceived as threatening, brought about by urbanization and mass migration (Abed & Abbas, 2011, 2014; Del Giudice, 2018). This perspective could suggest novel hypotheses about the epidemiology and demographics of disorganization.

Disorganization in males and females

Although prior meta-analytic work did not find evidence of sex differences in infant attachment disorganization (Van Ijzendoorn et al., 1999), the set of studies included in the meta-analysis was heterogeneous, and some studies of infant disorganization in high-risk samples have found evidence for increased rates of attachment disorganization in males (e.g. Carlson et al., 1989b; Lyons-Ruth et al., 1999). It is worth noting that these studies (most of which were included in van IJzendoorn et al.'s meta-analysis) only probed differences between male and female distributions at the level of attachment categories (Lyons-Ruth et al., 1999 the exception); it is not clear whether more robust differences would emerge if analyses were conducted using continuous disorganization scores. The pattern of sex differences becomes stronger and more consistent in the studies of attachment in early and middle childhood, using both separation-reunion procedures and doll-play vignettes (Barone et al., 2009; Del Giudice, 2008; Gloger-Tippelt & Kappler, 2016; Solomon & George, 2011; Tóth et al., 2013).

Of course, the relations between disorganized phenomena in infancy and middle childhood are complex, as is the empirical meaning of disorganization itself, and one must exercise care and diligence when drawing the necessary distinctions. That said, the totality of the data does point to the possibility that attachment disorganization may be more prevalent and/or expressed more intensely in boys, especially as children mature and sex differences in cognition and behavior become more pronounced. Middle childhood is a phase of rapid neurobehavioral maturation, driven by the onset of adrenal androgen secretion or *adrenarche*. Many sex differences in behavior – from aggression and social play to psychopathology – emerge or intensify during middle childhood (see Del Giudice, 2014); accordingly, there is good reason to suspect that disorganized phenomena may follow a similar trajectory.

The idea that disorganization may occur at different rates or with different characteristics in the two sexes is conceptually plausible for a number of reasons. First, males are at heightened risk for perinatal mortality and morbidity (Elsmén et al., 2004; Wells, 2000) and more vulnerable to most types of neurodevelopmental disorders (Polyak et al., 2015). Indeed, the Lyons-Ruth et al. (1997) study was consistent with this notion: infant disorganization was associated with lower mental developmental index (MDI) scores as measured by the Bayley Scales of Infant Development, and males were overexpressed

within those disorganized infants whose MDI scores were below 100, albeit low cell sizes precluded formal significance testing of this three-way effect.

Secondly, males also tend to be phenotypically more variable (Del Giudice et al., 2018b; Wyman & Rowe, 2014) and less developmentally canalized. Sexual selection is generally stronger on males, and traits subject to sexual selection tend to become exaggerated, more variable, and more sensitive to an individual's condition (Del Giudice et al., 2018b; Geary, 2015, 2017). In addition, selection to preserve adult reproductive function in the face of environmental perturbation should lead to stronger developmental buffering in females (Stinson, 1985) and is consistent with the idea that early stress tends to affect males more severely than females (Wells, 2000; for some exceptions see Geary, 2017).

David and Lyons-Ruth (2005) discussed sex differences in disorganization drawing on the "tend and befriend" hypothesis (Taylor, 2006; Taylor et al., 2000). The core idea is that females often react to threats and stressors with affiliation, caregiving, and search for social support – a pattern markedly different from the male-biased "fight or flight" response. Specifically, David and Lyons-Ruth (2005) suggested that sex differences in disorganization may become apparent when considering variation within the disorganized classification based on the best-fitting secondary "organized" classification. They found that female infants were more likely to be classified as D/Secure and male infants D/Insecure; when the mother's behavior became more frightening, female infants continued to approach them consistent with the D/Secure classification, whereas male infants evidenced displays of conflicted and disorganized behaviors characteristic of the D/Insecure classification (David & Lyons-Ruth, 2005).

Based on the decanalization hypothesis we proposed, disorganized males should show high levels of behavioral inconsistency, such as mixed intense displays of avoidance and resistance. Also, to the extent that broader neurodevelopmental vulnerabilities (e.g. for ASDs and SSDs) are linked to specific types of disorganized behavior, such as stilling and atypical postures, there may be patterns of sex differences in the frequency and correlates of these behaviors. Autism is substantially more common in males (French et al., 2013), and males tend to develop more severe forms schizophrenia and other SSD, and are strongly overrepresented in childhood-onset SSDs (Bartlett, 2014). There are also indications that freezing responses to danger and trauma may be more strongly expressed in females (Hagenaars, 2016; Kalaf et al., 2015), suggesting another possible pathway for sex-differentiated patterns of behavior within the D category. Collectively, these strands of work suggest that sex-linked patterns of developmental plasticity and stress-response functioning may plausibly underlie different subtypes of infant disorganization that could be validated by examining associations with increased liability to specific psychopathology syndromes and mental disorders in adolescence and adulthood.

Conclusion

Bowlby (1960) noted that a main reason he valued ethology was because it provided a wide range of new concepts to "try out in our theorizing." In sketching our vision for the third generation of attachment research through the lens of infant disorganization, we close by reemphasizing Bowlby's original view and suggest that a good deal of value is to be found in the application of contemporary evolutionary-developmental psychology (Ellis & Bjorklund, 2005) to attachment scholarship, and specifically attachment disorganization; this is because

within the current D classification system, varied behaviors might reflect one or more of a range of possible behavioral strategies, thus making the articulation of theoretically well-grounded explanatory hypotheses regarding their appearance and meaning challenging, yet potentially insightful.

Advances in the current understanding of attachment disorganization would strengthen attachment theory, increase its theoretical richness, and help bridge the gap with biological approaches in psychology. The evolutionary ideas discussed here also have potential value for informing policy and applied practice (Ellis et al., 2011). For example, evolutionary-minded scholars have used related ideas to explore novel treatment approaches (e.g. Ellis et al., 2017). As Schuengel et al. (2021) highlight, optimizing societal contributions of attachment research is a critical issue for the next generation of attachment researchers to embrace. We look forward to a vibrant third generation of attachment scholars who make new, unexpected discoveries, and write the next chapter in the story of attachment research.

Disclosure statement

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NEW RESEARCH

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"P" and "DP:" Examining Symptom-Level Bifactor Models of Psychopathology and Dysregulation in Clinically Referred Children and Adolescents

John D. Haltigan, PhD, Madison Aitken, PhD, Tracey Skilling, PhD, Joanna Henderson, PhD, Lisa Hawke, PhD, Marco Battaglia, MD, John Strauss, MD, Peter Szatmari, MD, Brendan F. Andrade, PhD

Objective: This study examined cross-informant evidence for a general factor of psychopathology ("P") and a narrower, clinically oriented dysregulation general factor based on the Dysregulation Profile ("DP") in a large clinical sample of children and adolescents. We also compared the magnitude of P and DP general factor associations with self-harm and suicidal ideation as an indicator of criterion validity.

Method: Itemwise data from the Child Behavior Checklist (N = 2,934; 4–18 years of age) were analyzed using confirmatory bifactor modeling and replicated in a supplementary analysis using Youth Self Report data (N = 2,395).

Results: General P and DP bifactor models fit the data better than single-factor and correlated factor models. Cross-informant criterion analyses on a subset of youth (n = 1,552) suggested that whether modeled as latent P or DP, associations with a brief composite index of self-harm and suicidal ideation are essentially of the same magnitude.

Conclusion: Our findings provide novel, large-sample support for the existence of general factors of psychopathology and dysregulation in clinically referred children and adolescents using a standardized rating system of psychopathology symptoms. Moreover, our results provide preliminary evidence that general psychopathology and dysregulation factors are clinically meaningful constructs. In addition, our findings raise the possibility that the DP general factor may serve as an efficient proxy for the general psychopathology factor in future clinical applications. Further efforts are necessary to understand the core empirical meaning of the P factor and to determine how it can be applied to clinical assessment and intervention.

Key words: dysregulation, externalizing, internalizing, p-factor, psychopathology

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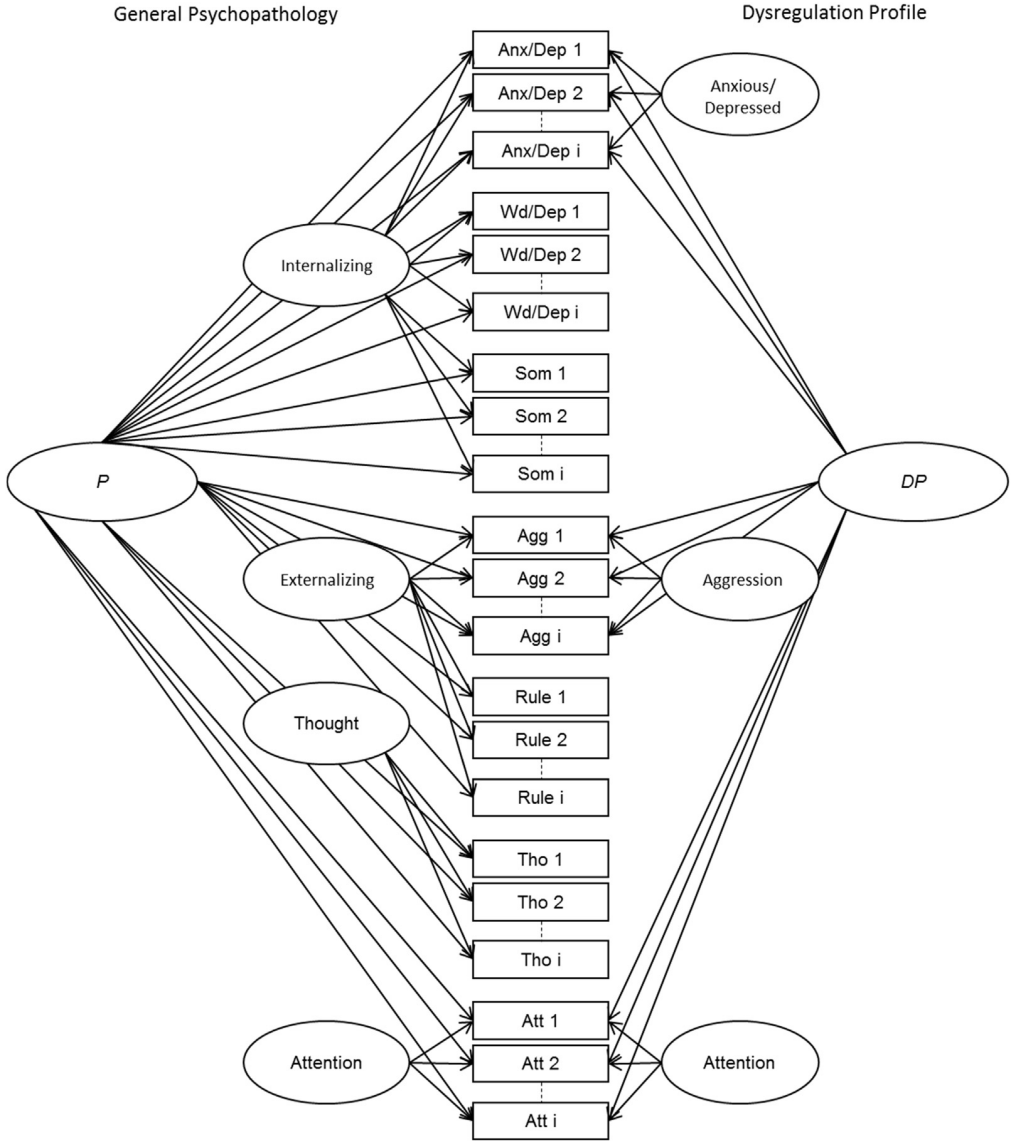
Categorical approaches to the study of psychopathology are limited in their ability to explain widespread phenomena, including high rates of comorbidity between disorders and heterogeneity within disorders.[1] In response to these limitations, there has been a recent resurgence of factor-analytic work on the metastructure of psychopathology. This work has yielded compelling evidence for the existence of a general factor of psychopathology (i.e., a "P" factor; Figure 1)[2] that accounts for common variation among a diverse array of mental health symptoms, including the internalizing and externalizing dimensions.[2-5] The P factor is thought to reflect shared etiological or transdiagnostic mechanisms, such as genetic vulnerabilities, environmental risk factors, or neurobiological atypicalities.[1,6]

Despite increasing empirical support for a general factor of psychopathology, several conceptual and methodological issues remain, and increased applicability of general psychopathology research to clinical practice with children and adolescents is needed. First, to the best of our knowledge, although the presence of a general psychopathology factor has been consistently reported in general population samples, it has yet to be demonstrated empirically in clinically referred children and adolescents. Verifying the presence of a general factor of psychopathology in a large sample of clinically referred children and adolescents would extend the growing body of work in population-based samples[2,7] and would provide a needed foundation for future research examining the utility of the P factor for assessment and treatment decisions. For example, establishing the presence of a general factor of psychopathology in clinical samples opens up the possibility for measurement invariance investigations across different risk group populations. Investigations of this kind have the potential to provide clinically relevant between-group information concerning the behavior of general psychopathology symptom

FIGURE 1 Orthogonal Bifactor Models of the General Psychopathology (P) and Dysregulation Profile (DP) Factors



Note: *Agg = aggression; Anx/Dep = anxious–depressed; Att = attention problems; DP = dysregulation profile; P = general psychopathology factor; Rule = rule breaking; Tho = thought problems; Wd/Dep = withdrawn/depressed.*

indicators that may differ in their factor loadings. Such information may provide more insightful clues regarding the symptoms and behaviors that drive the manifestation of the underlying mental illness, and may inform both basic understanding of etiopathology and the identification of clear treatment targets for transdiagnostic interventions.

Second, a number of investigations into the general psychopathology factor have used combinations of different measurement tools as symptom indicators.[7-9] Investigating the P factor using a well-known, multi-informant measure of psychopathology, such as the Achenbach System of Empirically Based Assessment (ASEBA),[10] which includes the Child Behavior Checklist (CBCL) and the Youth Self-Report (YSR), would have the benefit of producing findings that could be translated to both clinical and research applications. Third, given that there are discrepancies among informants regarding child and adolescent psychopathology,[11] examining separate models of behavioral rating data from various informants is important to further establish the validity of the P factor. Fourth, a good deal of work on the P factor has not included indicators of psychosis, mania, psychotic spectrum symptoms,[12] or thought

problems more generally (for exceptions, see Laceulle *et al.*[8] and Carragher *et al.*[13]). Including symptom-level indicators of thought problem/psychosis spectrum symptoms is warranted, given the increasing evidence of a thought/psychotic factor in structural analyses of psychopathology symptoms in adults,[1,14,15] as well as evidence that psychotic symptoms are fairly common in young people[16] and are associated with debilitating outcomes for a subset of individuals.[17]

In addition to methodological limitations, conceptual and empirical clarity is needed to better understand what the general psychopathology construct reflects.[8,18,19] It has been suggested that deficits in emotion regulation and self-control are a core feature of the P factor.[2] Paralleling the burgeoning literature on the P factor, a clinically oriented body of research on the structure of psychopathology has developed around the notion of an emotion dysregulation general factor. This dysregulation general factor is conceptually rooted in previous clinical research that identified a "Dysregulation Profile" of psychopathology indicators that was used to identify children with, or at risk for, pediatric bipolar disorder.[20,21] Most often, the dysregulation general factor is defined by items from the Aggressive Behavior, Attention Problems, and Anxious/Depressed syndrome scales of the CBCL,[22] which formed the basis for the original Dysregulation Profile index score.[20,21] Although more recent studies suggest the Dysregulation Profile does not accurately identify pediatric bipolar disorder,[23] it has proved useful in identifying children who are at high risk for persistent psychopathology and mental health impairment.[24] Recent item-level analyses support a dysregulation bifactor structure similar to that of the P factor, in which models including a dysregulation general factor (i.e., a "DP" factor; Figure 1) best account for symptom-level covariation among the lower-order syndrome dimensions.[22,25] Although there is considerable conceptual and measurement overlap between these 2 streams of research, previous studies have not examined bifactor models of general psychopathology and dysregulation in the same investigation. Empirical integration and synthesis of these complementary bodies of work may provide clinically useful information. For example, comparisons among these models may suggest the possibility that a general dysregulation factor could serve as a more psychometrically and clinically efficient proxy for the more expansive general psychopathology factor with respect to clinical risk prediction.[22]

The present study uses item-level data from the CBCL form of the ASEBA,[10,26,27] a widely used and well-validated behavior rating scale, to examine the structure of psychopathology based on parent-ratings in a large clinical sample of children and youth. We evaluated whether a bifactor model of general psychopathology fit behavioral and emotional problem data better than conventional hierarchical latent conceptualizations of correlated psychopathology dimensions (internalizing, externalizing, thought, and attention problem syndromes). A recent item-level structural investigation in 2 large clinical samples using the Strengths and Difficulties Questionnaire (SDQ),[28,29] a brief screening measure, found that a correlated factors model fit the data better than a bifactor or hierarchical model. Further research in large clinical samples using a comprehensive, widely used measure such as the CBCL is necessary to determine the extent to which bifactor representations of psychopathology are valid in clinic-referred children and adolescents. In addition, we add to existing research on the metastructure of psychopathology by examining a bifactor representation of CBCL items comprising the Dysregulation Profile, and evaluate the extent to which both bifactor representations of psychopathology are associated with a composite index of self-harm and suicidal ideation.[22,30] We also attempt to replicate the above analyses using item-level self-report data.

Based on their respective literature bases, we hypothesized that bifactor operationalizations of general psychopathology and dysregulation would better account for relations among items of lower-order dimensions than models without a bifactor. In addition, we reasoned that if the P factor is, in fact, largely indexing the construct of emotion dysregulation or negative emotionality, as has been suggested in the literature,[2,6,31,32] then a more circumscribed general factor of emotion dysregulation, empirically defined using a subset of items from the same standardized rating system, should demonstrate associations of similar magnitude with self-harm and suicidality, key indicators of psychological distress.

METHOD

Setting

Data were drawn from children and youth either referred or self-presenting to a large, urban, specialized mental health hospital in Toronto, Ontario, Canada, for clinical assessment and/or treatment. All procedures were approved by the hospital's Research Ethics Board.

Study Population

Participant assessments included one or more CBCL and/or YSR measures between January 1, 2003, and April 12, 2017, reflecting the full range of potentially useable data in the program-wide computer repository (the Assessment Data Manage [ADM]),[33] from its inception as a central data warehouse to when data were extracted for use in the current

report. Current clinic staff provided identification codes for youth who were assessed in their clinic and whose data were available in the ADM, or existing item-level ASEBA data from standalone databases. After data quality control and aggregation (see Supplement 1, available online, for additional detail), complete and reliable data were available for 2,934 caregiver/parent reports on the CBCL (76.8% boys; mean age = 12.52 years, SD = 3.91, range 4−18) and for 2,395 youth self-reports on the YSR (77.5% boys; mean age = 15.59 years, SD = 1.72, range 11−18. There were 1,552 participants who had both valid CBCL and YSR data available. Percentages of data drawn from the various clinics are as follows: mood and anxiety (7% CBCL; 6% YSR), child disruptive behavior (30% CBCL; 8% YSR), adolescent disruptive behavior/forensic (31% CBCL; 57% YSR), youth substance use (6% CBCL; 13% YSR), fire setting (8% CBCLs; 8% YSR), and general psychiatric consultation (19% CBCL; 8% YSR).

Measures

Child Behavior Checklist. The present sample included data from the Child Behavior Checklist (CBCL)/4-18[26] and the CBCL/6-18[10] forms. Six ineffective items from the CBCL/4-18 were replaced in the CBCL/6-18.[34] Both versions of the CBCL ask parents/caregivers to rate specific child behaviors (e.g., argues a lot) as 0 (not true of the child), 1 (somewhat or sometimes true), or 2 (very true or often true) within the past 6 months. Importantly, we used only those items that were consistent in content across the CBCL/4-18 and CBCL/6-18 forms.

Youth Self-Report. The Youth Self-Report (YSR)[10,27] is normed for youth 11 to 18 years of age and uses the same Likert rating response format as the CBCL. Item-to-syndrome profiles are analogous to those reported above for the CBCL with the exception that some parent/caregiver-report items (e.g., talks or walks in sleep) are not present on the YSR.

Factor analyses of the CBCL[10,26] have identified 8 common emotional/behavioral problem syndromes and 2 higher-order, broad-band symptom domains of internalizing and externalizing problems. The broad-band internalizing and externalizing domains have been widely featured in research on child and youth psychopathology.[7,8,13,35-37] In addition to these 2 broad-band domains, we examined items comprising 2 syndromes (i.e., dimensions) measured by the CBCL that have been included in previous research on the meta-structure of psychopathology, namely attention problems and thought problems.[1,8,13] Although attention problems are sometimes included within the externalizing domain,[8] we considered them as a separate dimension, in keeping with the factor structure of the CBCL.[10]

Data Analysis

We used confirmatory factor analysis to test models of general psychopathology using item-level CBCL data. All analyses were performed in M*plus* version 7.4[38] using the robust weighted least-squares mean variance (WLSMV) estimator, which is appropriate for ordered categorical and non-multivariate normal data.[39] Because the χ^2 statistic is sensitive to large sample sizes, evaluation of model fit was primarily made using the root-mean-square error of approximation (RMSEA), the magnitude of factor loadings, and the comparative fit index (CFI). Generally, a nonsignificant χ^2, RMSEA values ≤ 0.05, and CFI > 0.90 suggest adequate model fit.[40] Note, however, that these cutoffs are rules of thumb,[41] and our focal interest was in the relative comparison between multidimensional models with and without a general factor of psychopathology (see Supplement 1, available online, for further detail regarding evaluation of model fit indices, including some discrepancy between model-fit indices described below, as well as additional model-based reliability estimates for psychopathology dimensions).

Selection of core factor models to test was informed by previously reported theoretical models and those most frequently examined in empirical investigations of a general psychopathology factor.[2,6,8,13,42-44] As such, we tested 1-factor (model A), 2-factor (model B), and 4-factor (model C) models of item-level CBCL data based on the internalizing, externalizing, thought, and attention symptoms. Model B (2-factor) comprised only internalizing and externalizing items and was included because it is perhaps the most well-known, classic representation of psychopathology.[45] We compared the best-fitting of models A and C to both a standard bifactor model (model D) in which the lower-order symptom domains are orthogonal,[7,9] as well as an alternative bifactor model (model E), in which the underlying symptom domains are allowed to correlate [2,6,8] (see Figures S1−S7, available online, for these and additional sensitivity models tested). For these models, we used multiple-indicator, multiple-causes modeling (MIMIC)[46] to examine the impact of age and sex on psychopathology dimensions. In these models, sex was dummy coded (males = 0, females = 1).

These same model-fitting tests were repeated for analyses examining the DP bifactor. We tested 1-factor (model A) and 3-factor (model B) models based on the syndrome scales defining the original DP construct (i.e., anxious−depressed, aggressive, and attention),[21] and compared the best-fitting of these models to both a standard orthogonal bifactor model

(model C), as well as an alternative bifactor model in which the underlying DP symptom dimensions were allowed to correlate (model D; note there is one less model in these comparisons relative to P factor models).

For a subset of participants with both CBCL and YSR data (n = 1552), we regressed the latent dimensions of the P and DP bifactor models on a composite measure of 2 ASEBA items indexing self-harm (item 18) and suicidal ideation (item 91). To avoid shared method artifact, we regressed the P and DP bifactors on the self-harm and suicidality composite as indexed by the YSR and excluded items 18 and 91 from the bifactor models used in these analyses. Because these models are not nested, to formally test the magnitude of these associations, we used estimated factor scores computed from these models to generate bivariate associations among manifest factor score variables and the self-harm and suicidality composite, which we then compared using Steiger tests for dependent, overlapping correlations.[47] Further details are available in Supplement 1, available online, for this article. For all analyses presented, coefficients are standardized.

Finally, the above analyses were repeated using item-level YSR data, to provide a replication of the primary findings based on a different informant, namely the youth themselves (i.e., self-report). In these supplementary analyses, we regressed the P and DP bifactors on the self-harm and suicidality composite, as indexed by CBCL data.

RESULTS

General Psychopathology P Factor Models

Both the standard bifactor (model D) and correlated bifactor (model E) models exhibited better fit, compared to the 4-factor model without a P factor (model C), itself the best fitting of the models with no P factor (Table 1). Average factor loadings in the correlated 4-factor model for internalizing, externalizing, thought, and attention problem factors were moderate to strong in magnitude (0.57, 0.65, 0.57, and 0.67 respectively; p values for all factor loadings $<.001$; see Table S1, available online), suggesting strong correspondence between items and psychopathology factors.

For both the standard orthogonal and correlated bifactor models, loadings on the P factor were all positive and significant, with average factor loadings of 0.47 for the standard bifactor model and 0.44 for the correlated bifactor model (all p values $<.001$; see Table 2 and Table S1, available online).

TABLE 1 Model Fit Indices for Child Behavior Checklist (CBCL) Data General Psychopathology ("P") and Dysregulation Profile ("DP") Factor Models

Model	FP	χ^2	χ^2 Diff	RMSEA	RMSEA 90% CI	CFI
General psychopathology "P"						
One factor (model A)	258	55723.67	—	0.071	0.070–0.071	0.662
Two correlated factors (model B)	190	123752.52	—	0.069	0.068–0.070	0.783
Four correlated factors (model C)	264	38402.62	—	0.058	0.057–0.058	0.774
Orthogonal (standard bifactor; model D)	344	33908.10	N/A[a]	0.055	0.054–0.055	0.803
Nonorthogonal (correlated bifactor; model E)	350	29703.37	χ^2 (86) = 4406.64, $p < .001$[b]	0.051	0.050–0.051	0.830
Dysregulation profile "DP"						
One factor (model A)	117	21568.28	—	0.101	0.100–0.102	0.771
Three correlated factors (model B)	120	12852.33	—	0.077	0.076–0.078	0.867
Orthogonal (standard bifactor; model C)	156	9537.67	χ^2 (36) = 2234.02, $p < .001$[c]	0.068	0.066–0.069	0.903
Nonorthogonal (correlated bifactor; model D)	159	7240.93	χ^2 (39) = 2690.65, $p < .001$[c]	0.058	0.057–0.060	0.928

Note: N = 2,932. Two cases were missing data on all relevant indicators and were not included in model estimation. ASEBA behavior problem items 5 (enjoys little), 2 (drinks alcohol), 28 (breaks rules), and 99 (uses tobacco) were not used, as they are not common to both the 1991 CBCL 4/18[26] and 2001 CBCL 6/18[10] factor structures for internalizing and externalizing dimensions, respectively. CFI = comparative fit index; Diff = difference; DP = general dysregulation factor; FP = free parameters; P = general psychopathology factor; RMSEA = root-mean-square error of approximation; χ^2 = model chi-square.

[a]*As noted recently (see: www.statmodel.com/download/Bi-factor%20compared%20to%20correlated%20factors%20model.pdf), the classic orthogonal bifactor model in this case is not nested within the 4–correlated factors model. As such, MLR model estimation was also used for both the 4–correlated factors model and the standard orthogonal bifactor model. In this way, the Bayesian Information Criterion (BIC) was generated and used to compare the fit of these 2 models. The smaller BIC of the standard orthogonal bifactor model (360512.533) relative to the 4–correlated factors model (364842.165) suggests that the bifactor model provides a better fit to the data.*

[b]*Reference for χ^2 test for different testing (4 correlated factors; model C).*

[c]*Reference for χ^2 test for different testing (3 correlated factors; model B).*

TABLE 2 Factor Loadings for Child Behavior Checklist (CBCL) Data General Psychopathology ("P") and Dysregulation Profile ("DP") Orthogonal (Standard Bifactor) Models

	General Psychopathology (Model D)					Dysregulation Profile (Model C)			
Item	**P**	**INT**	**EXT**	**THO**	**ATT**	**DP**	**AD**	**AGG**	**ATT**
Internalizing									
Cries a lot	0.534	0.459				0.497	0.353		
Fears	0.312	0.226				0.323	0.464		
Fears school	0.522	0.401				0.284	0.469		
Fears doing bad	0.601	0.203				0.385	0.456		
Must be perfect	0.420	0.371				0.253	0.514		
Feels unloved	0.611	0.356				0.612	0.287		
Feels worthless	0.689	0.464				0.519	0.524		
Nervous, tense	0.663	0.467				0.543	0.487		
Fearful, anxious	0.589	0.531				0.407	0.750		
Feels too guilty	0.467	0.439				0.200	0.616		
Self-conscious	0.560	0.458				0.402	0.447		
Talk, thinks suicide	0.739	0.168				0.526	0.226		
Worries	0.616	0.508				0.396	0.707		
Rather be alone	0.425	0.216							
Won't talk	0.420	-0.003							
Secretive	0.585	0.052							
Shy, timid	0.329	0.495							
Lacks energy	0.598	0.328							
Sad	0.712	0.422							
Withdrawn	0.342	0.086							
Nightmares	0.542	0.220							
Constipated	N/A	N/A							
Feels dizzy	0.641	0.349							
Overtired	0.651	0.285							
Aches, pains	0.529	0.252							
Headaches	0.495	0.386							
Nausea	0.544	0.530							
Eye problems	0.453	0.141							
Skin problems	0.344	0.110							
Stomachaches	0.464	0.526							
Vomiting	0.469	0.385							
Externalizing									
Argues a lot	0.508		0.384			0.770		0.299	
Mean to others	0.411		0.558			0.582		0.558	
Demands attention	0.383		0.233			0.736		0.172	
Destroys own things	0.558		0.258			0.591		0.526	
Destroys others' things	0.422		0.531			0.605		0.595	
Disobedient at home	0.527		0.525			0.739		0.377	
Disobedient at school	0.359		0.659			0.535		0.356	
Gets in fights	0.317		0.612			0.527		0.486	
Attacks people	0.306		0.614			0.537		0.610	
Screams a lot	0.585		0.255			0.706		0.252	
Stubborn, sullen	0.524		0.305			0.806		0.124	
Mood changes	0.767		0.077			0.838		0.013	
Sulks	N/A		N/A			0.768		-0.141	
Suspicious	0.532		0.206			0.658		0.026	
Teases a lot	0.363		0.540			0.527		0.374	

(continued)

TABLE 2 Continued

	General Psychopathology (Model D)					Dysregulation Profile (Model C)			
Item	**P**	**INT**	**EXT**	**THO**	**ATT**	**DP**	**AD**	**AGG**	**ATT**
Temper	0.487		0.474			0.784		0.318	
Threatens others	0.446		0.649			0.572		0.584	
Loud	0.431		0.313			0.644		0.188	
Lacks guilt	0.284		0.505						
Bad friends	0.344		0.591						
Lies, cheats	0.484		0.494						
Prefers older kids	0.334		0.303						
Runs away	0.484		0.384						
Sets fires	0.410		0.356						
Sex problems	N/A		N/A						
Steals at home	0.458		0.468						
Steals outside home	0.341		0.617						
Swearing	0.470		0.515						
Thinks of sex	0.378		0.366						
Truant	0.360		0.476						
Uses drugs	0.363		0.496						
Vandalism	N/A		N/A						
Thought									
Can't get mind off	0.634			0.083					
Harms self	0.777			−0.122					
Hears things	0.577			0.572					
Twitching	0.672			0.125					
Picks skin	0.582			0.055					
Sex parts in public	N/A			N/A					
Sex parts too much	N/A			N/A					
Repeats acts	0.505			0.222					
Sees things	0.534			0.611					
Sleeps less	0.534			-0.115					
Stores things	0.437			0.220					
Strange behavior	0.596			0.512					
Strange ideas	0.674			0.490					
Sleep talks/walks	N/A			N/A					
Trouble sleeping	0.671			−0.109					
Attention									
Acts young	0.289				0.241	0.446			0.234
Can't concentrate	0.609				0.635	0.575			0.612
Can't sit still	0.570				0.489	0.599			0.357
Confused	0.794				−0.153	0.423			0.573
Daydreams	0.552				0.110	0.398			0.581
Impulsive	0.604				0.178	0.796			0.140
Poor schoolwork	0.448				0.231	0.433			0.338
Stares	N/A				N/A	0.466			0.558

Note: N = 2,932. Two cases were missing data on all relevant indicators and were not included in model estimation. ASEBA behavior problem items 5 (enjoys little), 2 (drinks alcohol), 28 (breaks rules), and 99 (uses tobacco) were not used as they are not common to both the 1991 CBCL 4/18[26] and 2001 CBCL 6/18[10] factor structures for internalizing and externalizing dimensions, respectively. AD = anxious–depressed; AGG = aggressive; ATT = attention; DP = general dysregulation factor; EXT = externalizing; FP = free parameters; INT = internalizing; N/A = not applicable; P = general psychopathology factor; THO = thought; UNI = unidimensional.

Loadings of lower-order, syndrome-specific indicators that drop in size, but are high loaders on the P factor, suggest that these items more directly predict general psychopathology. Of note, in the correlated bifactor model, the association between the specific internalizing and externalizing factors reversed in direction ($r = -0.35$; $p < .001$) from that which was observed in the correlated 4-factor model ($r = 0.54$; $p < .001$).

Although the standard orthogonal and correlated bifactor models were approximately equally well fitting, with marginally stronger CFI and RMSEA values for the correlated bifactor model, for the sake of parsimony and also to more closely follow standard bifactor modeling conventions in which specific factors are *not* permitted to correlate,[7,9,48] we chose model D (standard orthogonal bifactor) as the model on which we subsequently examined covariates. In this model, the P factor is a general psychopathology factor accounting for shared variance across all CBCL behavioral problem items, whereas the internalizing, externalizing, thought, and attention problem factors represent the common variance among their respective indicators. The structure of the P factor was reflected by salient loadings from each of the internalizing, externalizing, thought, and attention problem lower-order dimensions, defined here as ≥0.40. Nonetheless, it is noteworthy that a somewhat higher proportion of thought (87%) and attention (100%) problem items, relative to internalizing (44%) and externalizing items (73%), were salient loaders on the P factor.

The MIMIC model, in which the latent factors of model D were regressed on participant sex and age, provided adequate fit to CBCL data (CFI = 0.83, RMSEA = 0.050, 90% CI = 0.049 −0.050). A stronger association with the internalizing factor was observed for girls ($\beta = 0.38$, $p < .001$), whereas stronger associations with the externalizing ($\beta = -0.37$, $p < .001$), thought ($\beta = -0.34$, $p < .001$), and attention ($\beta = -0.52$, $p < .001$) problem factors were observed for boys. A stronger association with the P factor was found for girls ($\beta = 0.29$, $p < .001$). Finally, model results revealed small positive associations between age and the internalizing factor ($\beta = 0.13$, $p < .001$), as well as modest negative associations between age and the thought factor ($\beta = -0.15$, $p < .001$), and the P factor ($\beta = -0.16$, $p < .001$).

DP Factor Models

For the DP factor models, both the standard bifactor (model C) and correlated bifactor (model D) models exhibited better fit than the 4-factor model without a general DP factor (model B), itself the best fitting of comparison models with no DP factor (see Tables 1, 2, and Table S2, available online). We regressed the factors of model C on participant sex and age. This model provided satisfactory fit to the data (CFI = 0.89, RMSEA = 0.067, 90% CI = 0.066−0.068). Consistent with the general psychopathology MIMIC model, a stronger association with the anxious−depressed factor was found for girls ($\beta = 0.42$, $p < .001$), whereas stronger associations with the aggressive ($\beta = -0.37$, $p < .001$) and attention problem ($\beta = -0.32$, $p < .001$) factors were observed for boys. As well, paralleling general psychopathology (P) models, a stronger association with the DP general factor was found for girls ($\beta = 0.23$, $p < .001$). Finally, positive associations between age and the attention problems factor ($\beta = 0.10$, $p < .001$) and negative associations with the aggressive factor ($\beta = -0.18$, $p < .001$) and the DP factor ($\beta = -0.24$, $p < .001$) were also observed. In general, these associations were of modest magnitude.

Relations of General and Symptom-Specific Factors With Suicidal Behavior

In our P factor model, both the general P factor ($\beta = 0.22$, $p < .001$) and the symptom-specific internalizing factor ($\beta = 0.22$, $p < .001$) were positively associated with the self-harm and suicidality composite. In contrast, the symptom-specific externalizing factor was negatively, and more weakly ($\beta = -0.90$, $p < .002$), associated with the self-harm and suicidality composite. Neither the symptom-specific thought ($\beta = 0.01$, $p = .884$) nor the attention problem ($\beta = -0.04$, $p = .305$) factors were significantly associated with the composite. A Wald test of parameter constraints confirmed that the strength of associations between the P factor and symptom-specific internalizing factor with the self-harm and suicidality composite were not significantly different from one another [Wald χ^2 (1) = 0.002, $p = .964$]. Similarly, in our DP model, the DP factor ($\beta = 0.17$, $p < .001$), as well as the symptom-specific anxious−depressed factor ($\beta = 0.26$, $p < .001$) were positively associated with the self-reported self-harm and suicidality composite. Neither the symptom-specific aggression ($\beta = -0.05$, $p = .149$) nor the attention problem ($\beta = 0.04$, $p = .271$) factors were associated with the composite. However, a Wald test of parameter constraints demonstrated that the anxious−depressed factor was a stronger predictor of self-harm and suicidality than was the DP general factor [Wald χ^2 (1) = 4.78, $p < .05$]. Finally, although the absolute magnitudes of bivariate associations were similar, correlations between estimated general factor scores and the self-harm and suicidality composite were significantly stronger for P factor scores ($r = 0.24$, $p < .001$), than for DP factor scores ($r = 0.19$, $p < .01$), $t = -5.87$, $p < .001$.

Replication Using Youth Self-Report (YSR) Data

The general pattern of findings reported above based on CBCL data was replicated using YSR data, with some notable exceptions. In particular, in these analyses, positive associations of the general P and DP factors with the self-harm and suicidality composite were definitively the strongest in magnitude relative to symptom-specific factors (see Tables S3–S6, available online, for full results). These analyses provide additional support for the bifactor structure of psychopathology in clinic-referred children and youth, whether defined by the broader P factor or the narrower DP factor.

DISCUSSION

The current study extends prior work investigating the general factor of psychopathology in adolescents and adults,[2,4,8,9,13] as well as the general factor of dysregulation,[22] using itemwise behavioral problem data from a large sample of clinically referred children and adolescents. To our knowledge, this is the first study in which the general psychopathology factor and the more specific Dysregulation Profile dimension have been examined in the same sample.

Consistent with prior reports in children, adolescents, and adults,[2,4,8,22,25] findings from bifactor analyses of underlying symptom dimensions provided evidence for the existence of general factors of psychopathology, whether considered more expansively (i.e., the P factor) or more narrowly (i.e., the Dysregulation Profile). We also observed the reversal of the direction of association (from positive to negative) between the internalizing and externalizing factors once the P factor was taken into account, as observed in previous bifactor studies of general psychopathology.[2,8] This was also the case for our DP models, in which we observed a similar reversal of association between the anxious–depressed and aggressive behavior factors after taking into account the DP factor. This suggests that the original positive correlation in the population is because these dimensions share a common liability to generalized psychopathology and dysregulation, respectively. Once the general P or DP factor is taken into account, the lower-order behavioral syndromes (i.e., factors) become "purified" constructs to the extent that they describe unique syndromes or behavioral phenotypes with potentially distinct etiology.[14,49]

Importantly, consistent with prior evidence,[2,8,19] the P factor was most strongly defined by items from the thought and attention problem dimensions, with these domains containing the largest proportion of items with salient loadings on the P factor, relative to internalizing and externalizing factors. The characterization of the P factor in the current work provides further evidence that the empirical definition of the P factor may evidence some tilt toward thought and attention disturbance. The substantive meaning reflected by this empirical characterization should not be overlooked in our view, and warrants continued attention as investigations of a general psychopathology construct across the lifespan continue to accumulate.

To the extent that comprehensive symptom-level systems (e.g., the ASEBA) and/or investigations of a general psychopathology dimension among indicator pools with wide scope (i.e., using dimensional measures tapping multiple syndrome domains) converge on this empirical pattern of thought and attention problem indicators loading more strongly on the general psychopathology factor, it may illuminate a more precisely operationalized psychopathology phenotype. Such converging evidence may inform the search for neurobiological and behavioral correlates that provide clues to its underlying etiopathology. This is underscored in the present investigation by the observation that ASEBA thought problem indicators contain items that possess face validity with respect to both psychotic and bipolar disorders, and have been used in CBCL item–based development of additional screening measures for juvenile bipolar disorder (distinct from the DP profile) predicated upon DSM operational criteria for mania, as well as attention to childhood and adolescent psychosis.[50] Of note, the scale developed by Papachristou *et al.* showed superior predictive ability in a general, population-based sample with respect to bipolar disorder type 1 over that of the DP profile, which does not contain thought problem items.

Not unrelated to the empirical characterization of the P factor described above, our findings also offer further evidence for a reliable thought problem dimension as measured by the CBCL in children and adolescents. This is consistent with the balance of evidence for its existence as a separate psychopathology domain alongside the internalizing and externalizing domains in adults.[1,15,51] The proclivity for boys to show elevations on this dimension after accounting for general psychopathology is consistent with work showing stronger male sex effects on problems indexing psychosis spectrum symptoms and mania.[12] Although we are not suggesting the thought dimension in the current study is isomorphic with other thought problem factors enumerated in work with adolescents and adults,[51,52] we retain the Thought Problem label given that the ASEBA system[10] uses this nomenclature, and the items themselves share both conceptual and content overlap with the symptoms and disorders that load on thought disorder factors.[51,52] Nonetheless, it remains

crucially important to replicate the present results in clinical samples using more comprehensive measures of thought problems.

Associations of sex and age with the internalizing, externalizing, and attention problem dimensions, as well as the general P factor, across both parent and youth informants, are consistent with those found in community sample investigations of the P factor.[2,7,13,35,53,54] We also found similar associations between sex and the narrower anxious–depressed, aggression, attention problems, and DP factors. Nonetheless, the relatively small effect sizes for age should be kept in mind, and some differences in our findings with previous work should be noted. For example, whereas we found sex-specific effects for girls on the P factor, Patalay *et al.*[7] did not. It is possible, however, that the use of different measures between the current work and that of Patalay *et al.*,[7] who used the SDQ,[29] may have contributed to these divergent findings. As well, it is important to note that our MIMIC models involving the P factor were conducted without formal evaluation of measurement invariance, as the large number of symptom-level indicators resulted in model convergence problems. Thus, associations with sex and age should be interpreted bearing this in mind. That said, follow-up analyses revealed that the P factor model used in our MIMIC analyses (model D) showed virtually the same fit for both boys and girls when analyzed separately, suggesting evidence for its configural invariance (i.e., same factor structure in boys and girls). Moreover, for the narrower DP general factor model, additional multiple group measurement invariance analyses (see Table S9, available online) provided evidence for its invariance across sex, both replicating a previous study that modeled the DP factor using CBCL data,[22] as well as extending these measurement invariance findings to youth self-report (YSR) data. Taken together, these additional analyses lend strong confidence to observed associations with sex in our MIMIC models, and especially so in the case of the DP construct.

This work also builds on prior factor-analytic investigations of the DP general factor in children[25] and adolescents[22] using the CBCL, and is the first, to our knowledge, to replicate evidence for a dysregulation or DP bifactor[22] in clinically referred school-aged children and adolescents. Our findings provide preliminary evidence that the addition of a general psychopathology (P) factor meaningfully describes variation and consistency in psychopathology symptoms in clinically referred children and adolescents. Moreover, the significant association between the P factor and a composite measure of self-harm and suicidal ideation, a marker of clinical severity and risk, provides some evidence, albeit provisional, that the P factor is a clinically meaningful construct. Correlational analyses also suggested that the more narrow-band DP factor is quantitatively similar to the P factor with respect to its associations with this composite measure of self-harm and suicidal ideation. That said, these results should be balanced against findings that the symptom-specific internalizing (P factor model) and anxious–depressed (DP factor model) factors were associated with the self-harm and suicidality composite at equal (P factor model) or stronger (DP factor model) magnitudes, raising the possibility that, at least for parent-reported behavioral symptoms in relation to youth-reports on the self-harm and suicidality composite, internalizing symptoms may have a particularly important, and perhaps more practically significant, meaning. Intriguingly, as both the P and DP general factors derived from youth self-report data were definitively more strongly and positively associated with our self-harm and suicidality composite derived from parent-reported data than were symptom-specific factors (see Supplement 1, available online), it is possible that caregivers' own internalizing symptoms in rating their children may have contributed to this somewhat differing pattern of results across informants. Future work examining the predictive significance of general factors of adolescent psychopathology should consider this possibility. Nonetheless, taken together, the present results provide tentative evidence that the DP general factor may serve as a "clinical proxy" for the P factor, and may be useful to clinicians in terms of providing a more efficient transdiagnostic indicator of psychopathology severity and behavioral risk.

The present results also provide the necessary structural evidence for future research examining the clinical utility of general P and DP factors for both assessment and treatment applications. In particular, future clinically oriented research that may lead to the development of efficient and psychometrically valid measurement approaches that assess both general factors and purified, lower-level psychopathology constructs would be of value, as would the examination of the prognostic value of these constructs. Although further research is needed to determine how general factors of psychopathology can best be integrated into clinical practice, our results support efforts by clinicians and researchers to consider shared factors across disorders, such as emotion dysregulation, that may account for disorder comorbidity and overlapping clinical presentations across disorders.

Significant strengths of the current study include our large sample of item-level behavioral problem ratings for clinically referred children and youth from a publicly funded system in a culturally and linguistically diverse large

urban city. There are also limitations to our data and analyses that merit comment. Most importantly in our view, because data were not collected as part of an a priori designed research investigation, additional variables and measures across clinics that could be used to externally validate the CBCL-derived factors were not collected; only sex and age were available for all participants, and YSR suicidal thoughts and behavior for a subset of participants. In addition, our self-harm and suicidality composite was based on only 2 behavioral indicators, necessarily limiting the scope and precision of the measurement of this concurrent "clinical risk" criterion. Second, all models are cross-sectional and, as such, we were not able to interrogate longitudinal measurement invariance and construct stability, nor predictive validity of the P or DP factors.[9,19] As discussed above, further investigation of the external validity and measurement invariance of the general psychopathology factor, especially that modeled using itemwise data with standardized instruments such as the CBCL, is urgently needed, as it has the potential to yield valuable insight into both etiopathological correlates (e.g., brain structures) and between-group (e.g., biological sex) differences in various phenotypic expressions of child and adolescent psychopathology. Third, our sample was disproportionate in terms of sex composition, with boys overrepresented. However, this sex composition is consistent with the clinical and forensic nature of the sample, which includes children and youth with diverse externalizing clinical presentations (e.g., fire setting, delinquency),[55] and has been observed in prior investigations of the bifactor structure of the Dysregulation Profile in clinically referred preschool children.[25] Nevertheless, results favoring the bifactor model of psychopathology were consistent with extant work, and sensitivity analyses with boys only were virtually identical to the full sample (see Tables S5−S8, available online). Finally, all participants were assessed at a single facility, which may limit the generalizability of the results.

The current empirical work is, to the best of our knowledge, the first large-sample, item-wise extension of evidence for a general factor of psychopathology, as well as a narrower general factor construct of emotion dysregulation, using the CBCL in clinically referred children and youth. Our results underscore the clinical significance of both the P and DP general factors as transdiagnostic indicators of mental illness. Results supporting a general factor of psychopathology in clinically referred children and youth provide an evidence-based impetus to advance scientific understanding of mental illness etiology and to further the development of transdiagnostic approaches to assessment and intervention,[56,57] which current psychiatric nosologies have largely failed to do.[58] Continued empirical work investigating the metastructure and external correlates of the general psychopathology factor, as well as more narrow-band factors or spectra,[1] such as the DP factor and other factor-analytically derived dimensions of potentially disorder-specific specific disturbance[50] that account for underlying symptom covariation, are essential to provide a cumulative and reliable empirical platform that can facilitate efforts to develop transdiagnostic intervention approaches based on children's individual symptom presentations.[1,59]

Despite their considerable descriptive power and heuristic value, general factor models of psychopathology are limited in their explanatory power, given that they are empirical generalizations rather than predictions derived from theoretical principles.[49] Thus, in addition to clinical integration of dimensional schemes of psychopathology with general factor models of psychopathology, it remains important to militate against reification of the P and DP factors by encouraging principled thought around what they may reflect.[14,49] The present study adds to the existing empirical evidence suggesting that the underlying structure of psychopathology is organized at a general level accounting for both disorder comorbidity and heterogeneity; that is, a common association among symptoms across the spectrum of psychopathology exists, represented by a general psychopathology factor, along with separable dimensions of psychopathology (i.e., syndromes). With continued empirical evidence supporting this metastructure of psychopathology, it is important that conceptual and structural investigations of transdiagnostic general factors continue to inform basic research (e.g., the Research Domain Criteria Initiative [RDoC][60]) and clinical practice.

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Supplementary Materials

"P" and "DP": Examining Symptom-Level Bifactor Models of Psychopathology in Clinically Referred Children and Adolescents

Methodological and Analytical Details

Detailed Information about Data Quality Control and Aggregation

Prior to analysis, cases from the program-wide data repository that were outside the age range (4-18) for the CBCL (n = 10) or YSR (age 11-18; n = 24), or who were missing age data (n = 10 for CBCL; n = 14 for YSR) were excluded. For cases in which multiple respondents' CBCL ratings for a given youth were available (e.g., biological mother and father, or they were assessed more than once across or within clinics; < 25% of CBCL data), or where there were multiple occasions on which the youth completed the YSR (e.g., they were assessed in more than one clinic that contributed data, or they were assessed on multiple occasions in the same clinic; < 5% of YSR data), we used the maximum rating for a given indicator so as to index the highest level of psychopathology reported for each individual. This was done to leverage all available data for that youth, thus creating a more reliable overall index of the strongest level of that problem behavior (i.e., item). Using the highest rating was also necessary as weighted least squares mean variance estimation (WLSMV) requires integer values for the declared categorical outcome variables (see below). Note that in cases where a youth had assessment data from multiple occasions, the participant's mean age was computed during data aggregation. Because the occurrence of multiple CBCL respondents was not uniform across participants, any exclusionary decision regarding a particular respondent would have been arbitrary and lead to a loss of raw CBCL data. Likewise, because multiple YSR ratings either within or across clinics were not uniform and were not directly tied to any formalized treatment program within the hospital, any decision to exclude a given YSR form would have been arbitrary.

Previous Factor Analytic Work with the ASEBA Rating System: Relevance to Current Analyses

Our use of item-level ASEBA response data in the current investigation was motivated by our belief that an item-wise analysis would allow for more comprehensive symptom coverage than an analysis based on composite symptom-level scales, thus allowing for a more granular structural mapping of behavior problems to their higher-level psychopathology dimension. Although the focus of our analyses was not to examine the construct validity of ASEBA CBCL and YSR measurement structures described in Achenbach and Rescorla[1], our results do provide data relevant to such issues. Below we describe additional details concerning how confirmatory models in the current report map onto the factor structure of ASEBA behavioral problem items as outlined in Achenbach and Rescorla.[1]

Confirmatory modeling of internalizing and externalizing factors using items which comprise lower-level syndrome scales (e.g., anxious/depressed, withdrawn/depressed, and somatic complaints comprising internalizing problems) departs from the derivation of internalizing and externalizing scales which were based on factor analyses of raw *scale* scores on the lower level syndromes themselves (i.e., not items).[1] In extended sensitivity analyses, we examined various additional configurations of correlated factors models with these items included and compared them to bifactor models in the same manner as our focal analyses (see figures S6-S7, models F and G, respectively). As with our focal analyses, models with a general psychopathology factor (standard orthogonal bifactor model) generally fit better than did their counterparts without a general psychopathology factor (i.e., correlated factors) as adjudicated by model fit indices (see Figures S6 and S7 for model fit indices for correlated and bifactor models).

Technical Details Pertaining to Model Fit Indices for ASEBA CBCL data

Our focus in the current report was on comparisons between models of psychopathology with and without a general psychopathology factor. We make this point as conventional indices of absolute model fit were somewhat tentative for CBCL models based on the comparative fit index (CFI), which was rather discrepant with the RMSEA model fit index for the same models. This issue of discrepancies between CFI and RMSEA values has recently received research attention in its own right,[2] and concerns about the CFI index with large numbers of (ordered) categorical items have been noted in prior investigations of ASEBA data,[3] which have relied primarily on the RMSEA as an indicator of model fit. Indeed, Achenbach and Rescorla[1] and Dumenci, Erol, Achenbach, and Simsek[4] do not report CFI values for their analyses of ASEBA data. Moreover, simulation work has suggested that RMSEA was the best performing index for WLSMV in large samples (i.e., N = 1,000).[5] Lastly, as has been noted in prior confirmatory modeling work with CBCL data,[6] and reiterated in Lai and Green,[2] the extent to which data characteristics (e.g., non-normality, ordered categorical indicators, sample sizes, indicator number) influence the values of fit indices is difficult to determine precisely. Fixed cutoff values for 'adequate' fit may not work well with large models, large sample sizes, and categorically skewed variables, and as such whether cutoff rules of thumb apply to the present situation is unknown. For a more extended discussion of these issues relevant to confirmatory tests of ASEBA data, see Ivanova et al.[7], who also note in citing Marsh, Hau, and Wen[8] that overreliance on fixed cutoff values to evaluate model fit (e.g., Hu & Bentler[9]) can be problematic if applied without attention to the particular characteristics of the data being analyzed (e.g., indicator scale type), and that a more balanced

(and less stringent 'cutoff' threshold heuristics) approach to model fit evaluation is warranted (e.g., 'adequate' model fit for CFI ranging from .80 - .90).

As noted above, these issues are particularly salient in the current analyses with respect to the CFI, as it is an incremental fit index and calculation of the CFI value is made relative to a baseline 'null' model (in the case of M*plus* this model reflects *no* covariation among factor indicators). To the extent that average correlations amongst indicators in the hypothesized model are not particularly strong (which may be more likely with large numbers of items reflecting conceptually different syndromes), the strength of the CFI will be adversely impacted. Evidence that this may be the case in our data is suggested by the more favorable CFI values (based on conventional rules of thumb) for our CBCL DP factor models, which are based on a much smaller subset of items thought to reflect a more narrow construct. Whatever the case may be, average factor loadings for all modeled behavior problem dimensions were remarkably consistent with prior work,[1,3,10,11] pointing to the reliability of the empirical meaning of these domains for CBCL data. Most importantly, relative comparisons between models with and without a bifactor dimension, as judged by inspection of fit indices, favored models incorporating a general psychopathology P factor with smaller more favorable cutoff values for these models.

Technical Details Pertaining to General Bifactor Relations with Suicidality

Given items 18 and 91 (the suicidality index items for both CBCL and YSR data) are worded almost identically across informants, and because we wanted to avoid shared-method bias in these analyses, we did not include these items when constructing the bifactor P and DP models for these analytic models (i.e., these items were used to create the suicidality index for each informant). In each analysis, the latent general and symptom-specific factors from a given informant was regressed on the suicidality composite (the sum of items 18 and 91) from the opposite informant. As such, *n*s for these analyses reflect participants with both CBCL and YSR data for each run (for the CBCL bifactor models, n = 1529; for the YSR bifactor models, n = 1518). This was also the case for the corresponding Steiger tests of factor-based general P and DP score associations with the CBCL and YSR-based suicidality composites. Note also that because YSR data were used in both sets of analyses, the age range for these analyses is restricted to youth aged 11-18 years.

Technical Details Pertaining to Reliability Coefficients

To address conceptual and methodological concerns regarding the use of bifactor models of the P factor,[12] we computed model-based reliability statistics,[13,14] which can be used as an index of the amount of reliable variance that can

be attributed to various dimensions of a bifactor model. Reliability coefficients were calculated using the THETA parameterization of the ANALYSIS command with the WLSMV in conjunction with the MODEL CONSTRAINT command. Note that the THETA parametrization allows for residual variances for continuous latent response variables of observed categorical dependent variables to be parameters in the model, which is necessary for calculation of Omega statistics.

For both sets of confirmatory bifactor analyses, we computed model-based estimates of factor variance proportions and factor reliability, including: (a) Lucke's omega (ω),[13] which is analogous to the alpha reliability coefficient but allows items to possess different loadings; (b) the hierarchical omega coefficient (ω*H*),[15] which judges the degree to which composite scores are interpretable as a measure of a single common factor; and (c) the omega subscale (ω*S*) reliability estimate for a residualized subscale, an index of the reliability of the specific factor net of the P factor.[16] Values of the omega reliability coefficients may vary between 0 and 1, and higher scores indicate greater reliability.

For CBCL data, model-based omega coefficients demonstrated the P factor exhibited high reliability (ω = .98, ω*H* = .81), indicating that ~81% of the variance in the unit-weighted total score can be attributed to individual differences in the P factor. As such, ~83% (.81 divided by .98) of the reliable variance can be attributed to individual differences in overall psychopathology while only ~17% can be attributed to the specific internalizing, externalizing, thought, and attention problem factors. For YSR data, model-based reliability estimates again suggested the P factor exhibited high reliability (ω = .98, ω*H* = .83), indicating that ~83% of the variance in the unit-weighted total score can be attributed to individual differences in the P factor. As such, ~85% of the reliable variance can be attributed to individual differences in overall psychopathology while only ~15% can be attributed to the specific internalizing, externalizing, thought, and attention problem factors.

Related to these analyses, it is important to note that Omega coefficients are 'model-based' estimates in the sense that they are based on the polychoric correlation matrix of the continuous latent response variables thought to underlie the observed categorical dependent variables (Y*ij**), rather than the observed ordered categorical (ordinal) indicator variables (Y*ij*) themselves. Thus, to the extent that inferences based on these reliability estimates bear on practical decisions (e.g., clinical assessment), this empirical distinction should be kept in mind. Currently, to the best of our knowledge, there is no best practice regarding the presentation of model-based Omega statistics (in the case of ordered categorical data and the use of the WLSMV estimator as was the case here). Nonetheless, methodological work pertaining to this issue exists,[17,18]

and our decision to present these statistics was motivated in part by the fact that they have been presented previously in work exploring the general structure of psychopathology,[19] and we wanted to provide a point of comparison with this work.

Supplementary Analyses

Replication using YSR data

The steps followed in the primary analysis using CBCL data were repeated using YSR data (N = 2,395; 77.5% boys; *M*age = 15.59 [*SD* = 1.72], range 11 - 18).

General Psychopathology P Factor Models. As was the case in the primary analysis, both the standard bifactor (Model D) and correlated bifactor (Model E) models exhibited better fit to YSR data, compared to the four-factor model without a P factor (Model C), itself the best fitting of the correlated factors models with no P factor (see Table S3). Average factor loadings in the correlated four-factor model for internalizing, externalizing, thought, and attention problem factors were moderate to strong in magnitude (.63, .62, .64 and .59, respectively; *p*s for all factor loadings < .001; see Table S3), suggesting strong correspondence between YSR items and YSR psychopathology factors. For both the standard orthogonal and correlated bifactor models, loadings on the P factor were all positive and significant, with average factor loadings of .51 for the standard bifactor model and .48 for the correlated bifactor model for the YSR (all *p*s < .001; see Table S3). Replicating the findings from the primary analysis, in the correlated bifactor model, the association between the specific internalizing and externalizing factors reversed in direction (r = -.27; p < .001) from that which was observed in the correlated four-factor model (r = .58; p < .001).

The standard orthogonal and correlated bifactor models were again approximately equally well-fitting, with marginally stronger CFI and RMSEA values for the correlated bifactor model. We again chose Model D (standard orthogonal bifactor) as the model on which we subsequently examined covariates, for the sake of parsimony and to be consistent with standard bifactor modeling conventions.[20–22] In some contrast to the results of the primary analysis, the nature of the P factor as defined based on YSR items was primarily reflected by salient loadings (≥ .40) from the internalizing, thought, and attention problem dimensions (81%, 100%, and 86% of items with salient loadings, respectively), with relatively less contribution from externalizing items (52%).

The MIMIC model, in which the latent factors of Model D were regressed on participant sex and age, provided adequate fit to YSR data (CFI = .88, RMSEA = .043 [90% CI .043 - .044]). Consistent with the primary analysis, girls (β

= 0.59, $p < .001$) scored higher on the internalizing factor than boys, whereas boys scored higher on the externalizing (β = -0.29, $p < .001$), thought (β = -0.21, $p < .003$), and attention (β = -0.37, $p < .001$) problem factors than girls. Finally, consistent with the primary analysis, girls (β = 0.63, $p < .001$) scored higher than boys on the P factor. Significant associations were also observed between age and the internalizing, thought, and P factors, although patterns differed slightly from those found in the primary analysis. As was the case in the primary analysis, there were small positive associations between age and the internalizing factor (β = 0.06, $p < .001$) and small negative associations between age and the thought factor (β = -0.12, $p < .001$). In addition, a small negative association between age and the attention problem factor was found for YSR data only (β = -0.16, $p < .001$). Finally, whereas in the primary analysis there was a small negative association between age and the P factor, in YSR data, there was a small positive association between age and the P factor (β = 0.04, $p < .05$).

DP Factor Models. As was the case in the primary analyses, the standard bifactor (Model C) and correlated bifactor (Model D) models exhibited better fit to YSR data for the DP factor models than the four-factor model without a general DP factor (Model B), itself the best fitting of comparison models with no DP factor (see Tables S4). We regressed the factors of Model C on participant sex and age. This model provided satisfactory fit to the data (CFI = .90, RMSEA = .060 [90% CI .058 - .061]). Consistent with the primary analyses of DP factors and with the general psychopathology MIMIC models of YSR data, boys scored higher on the aggressive (β = -0.26, $p < .001$) and attention problem factors (β = -0.36, $p < .001$) than girls. In addition, girls scored higher on the anxious-depressed (β = 0.64, $p < .001$) and DP (β = 0.60 $p < .001$) factors than boys, with these sex differences being more pronounced for YSR than for CBCL models. Finally, associations of small magnitude were observed for age. Results differed slightly for YSR models in comparison to those found in the primary models using CBCL data. Specifically, in the models using YSR data, age was positively associated with the anxious-depressed factor (β = 0.08, $p < .001$), and negatively associated with the aggressive (β = -0.09, $p < .001$) and attention problem (β = -0.16, $p < .001$) factors.

Relations of General and Symptom-Specific Factors with Suicidal Behavior. In our P factor model, the YSR P factor (β = 0.31, $p < .001$) and the symptom-specific internalizing (β = 0.13, $p < .001$) and externalizing (β = 0.12, $p < .001$) factors were all positively and significantly associated with the CBCL self-harm and suicidality composite. Separate Wald tests of parameter constraints confirmed what might be surmised from the magnitude of the above positive associations; namely, that the P factor was more strongly associated with the self-harm and suicidality composite than was

either the internalizing (Wald χ^2 [1] = 29.38, $p < .001$) or externalizing (Wald χ^2 [1] = 30.56, $p < .001$) symptom-specific factors. Similarly, in our DP model, the YSR DP factor ($\beta = 0.31$, $p < .001$) was positively associated with the CBCL self-harm and suicidality composite, as were the symptom-specific anxious-depressed ($\beta = 0.12$, $p < .001$) and aggression ($\beta = 0.10$, $p < .001$) factors. Separate Wald tests of parameter constraints revealed that the general DP factor was a stronger predictor of the CBCL self-harm and suicidality composite than either the anxious-depressed (Wald χ^2 [1] = 24.04, $p < .001$) or aggression (Wald χ^2 [1] = 29.85, $p < .001$) symptom-specific factors. Finally, associations of P ($r = .33$, $p < .01$) and DP ($r = .32$, $p < .01$) factor scores with the CBCL-measured self-harm and suicidality composite were not significantly different from one another, $t = -.879$, $p = .379$.

In general, the pattern of findings in the primary analyses based on CBCL data was replicated in the supplementary analyses based on YSR data, providing evidence of the reliability of our primary findings. However, there were some notable differences: 1) items from the internalizing dimension showed more salient loadings on the P factor as defined based on YSR items than as defined based on CBCL items; 2) similarly, sex differences in anxious-depressed and DP factor scores, with girls scoring higher than boys, were more pronounced based on YSR than CBCL data; 3) slight differences in associations were found between some factors, sex, and age based on the P and DP models for YSR compared to CBCL data. In addition, relative to our CBCL-based P and DP factor models, the association of the P and DP factors with the self-harm and suicidality composite, *as indexed by parent reports*, was definitively the strongest in magnitude when formally compared with additional, symptom-specific P and DP factors (recall in the parent-reported CBCL-based factor models where the self-harm and suicidality composite was *indexed by youth reports*, both the P factor and the symptom-specific internalizing factor were *equally associated* with the self-harm and suicidality composite; moreover, in the CBCL DP model, the symptom-specific anxious-depressed factor was *more strongly* associated with the self-harm and suicidality composite than was the DP general factor). The additional empirical characterization of the YSR P factor by internalizing items is consistent with prior work indicating that youth, and male youth in particular, may show somewhat higher endorsement rates of internalizing and thought symptoms than their parents.[23,24] Such findings may reflect the 'internal' nature of internalizing symptoms, which may make them less readily observable to parents, underscoring the importance of chronicling differences in the empirical characterization of the P factor, particularly at the symptom level, given the oft-noted discrepancies between parent and youth ratings of youth mental health functioning.[25]

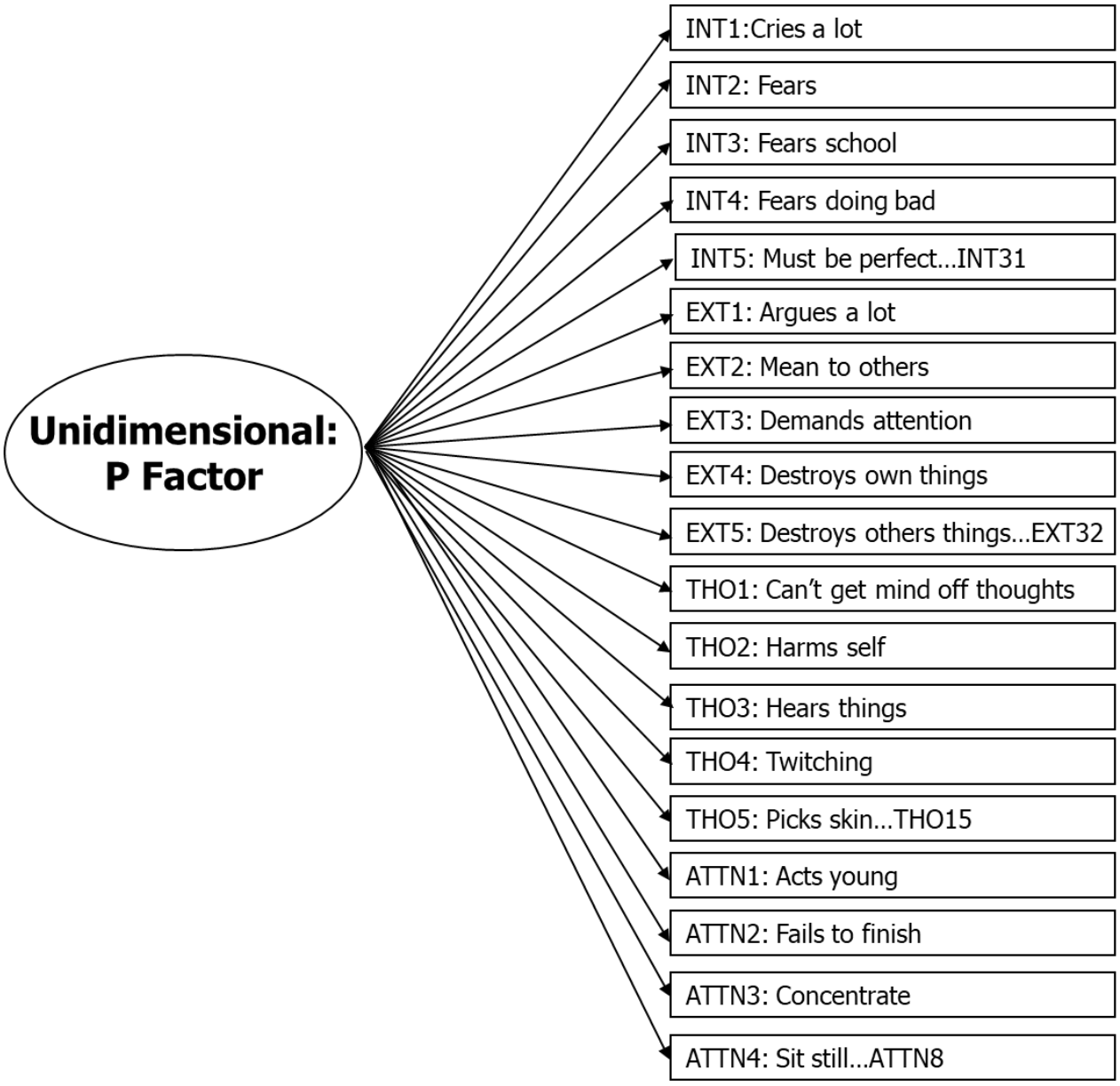
Factor Models for Boys Only Sample

Given that both the CBCL and YSR samples were disproportionate with respect to youth sex, we reran all focal P factor and ancillary DP factor models for boys only given that they constituted the large majority of the overall sample, and we wanted to inspect whether overall models might be disproportionately influenced by the inclusion of girls). Results of P factor models are reported in Tables S5 (CBCL) and S6 (YSR) and results of DP factor models are reported in supplementary tables S7 (CBCL) and S8 (YSR). As can be seen, model results were substantively very similar to full sample models, both with respect to structural coefficients (i.e., item-factor pattern loadings) and model fit indices (models for girls were also run separately and yielded a generally similar pattern of results; they can be obtained from the first author upon request).

Multiple-Group Measurement Invariance Analysis of CBCL and YSR DP Models across Sex

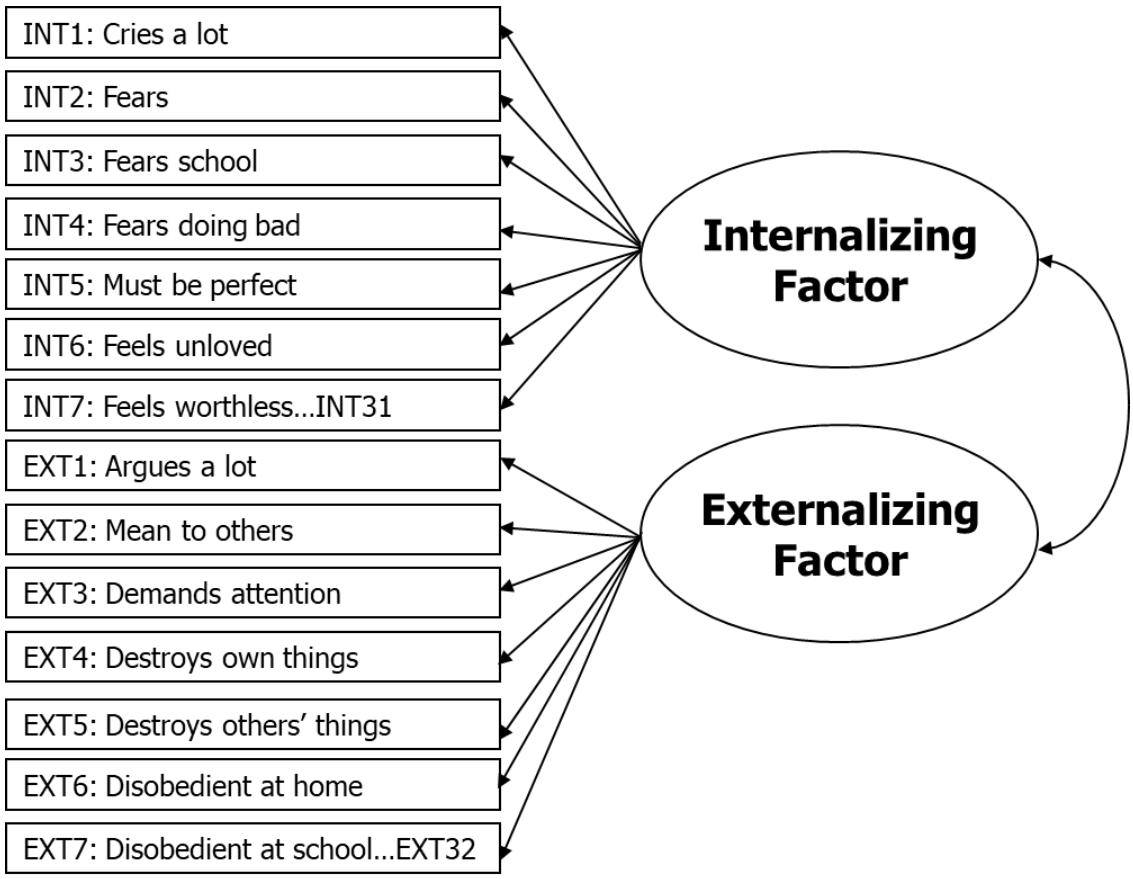
The reduced item content of the DP construct relative to the P construct afforded us the ability to formally evaluate the measurement invariance of our DP factor model using a multiple-group approach (a similar model with the larger P factor construct [i.e., substantially more indicators] was analytically intractable and did not converge in our data). As such, we examined the measurement invariance of the DP factor model used in our MIMIC analyses (Model C) across sex to lend additional rigor to our analyses, as well as provide a replication of earlier work,[26] which found the DP construct to be invariant across sex using CBCL data. Using the same procedures and criteria as in Deutz et al. (2016)[26] to examine the measurement invariance of the DP construct across sex, we found that the DP construct as modeled in our CBCL data was invariant across participant sex, thus replicating their earlier findings and lending strong support to our MIMIC analyses involving the DP construct. Consistent with our MIMIC results, formal tests of latent means in these analyses indicated that girls scored higher than boys on the DP and Anxious-Depressed factors (.22 and .48 units, respectively, $p < .01$) and that boys scored higher than girls on the Aggression and Attention Problems factors (.37 and .28 units, respectively, $p < .01$). Likewise, and of complete novelty, we also found that the DP construct as modeled in our YSR data was invariant across participant sex. Once again, formal tests of latent means in these analyses indicated that girls scored higher than boys on the DP and Anxious-Depressed factors (.50 and .73 units, respectively, $p < .001$) and that boys scored higher than girls on the Aggression and Attention Problems factors (.22 and .47 units, respectively, $p < .002$ and $p < .001$). Of note, mean difference effects for YSR data were, in general, stronger in magnitude than those for CBCL data. See Table S9 for tests of measurement invariance for the CBCL and YSR DP constructs.

Figure S1. One general psychopathology factor (unidimensional) model (**model A**).



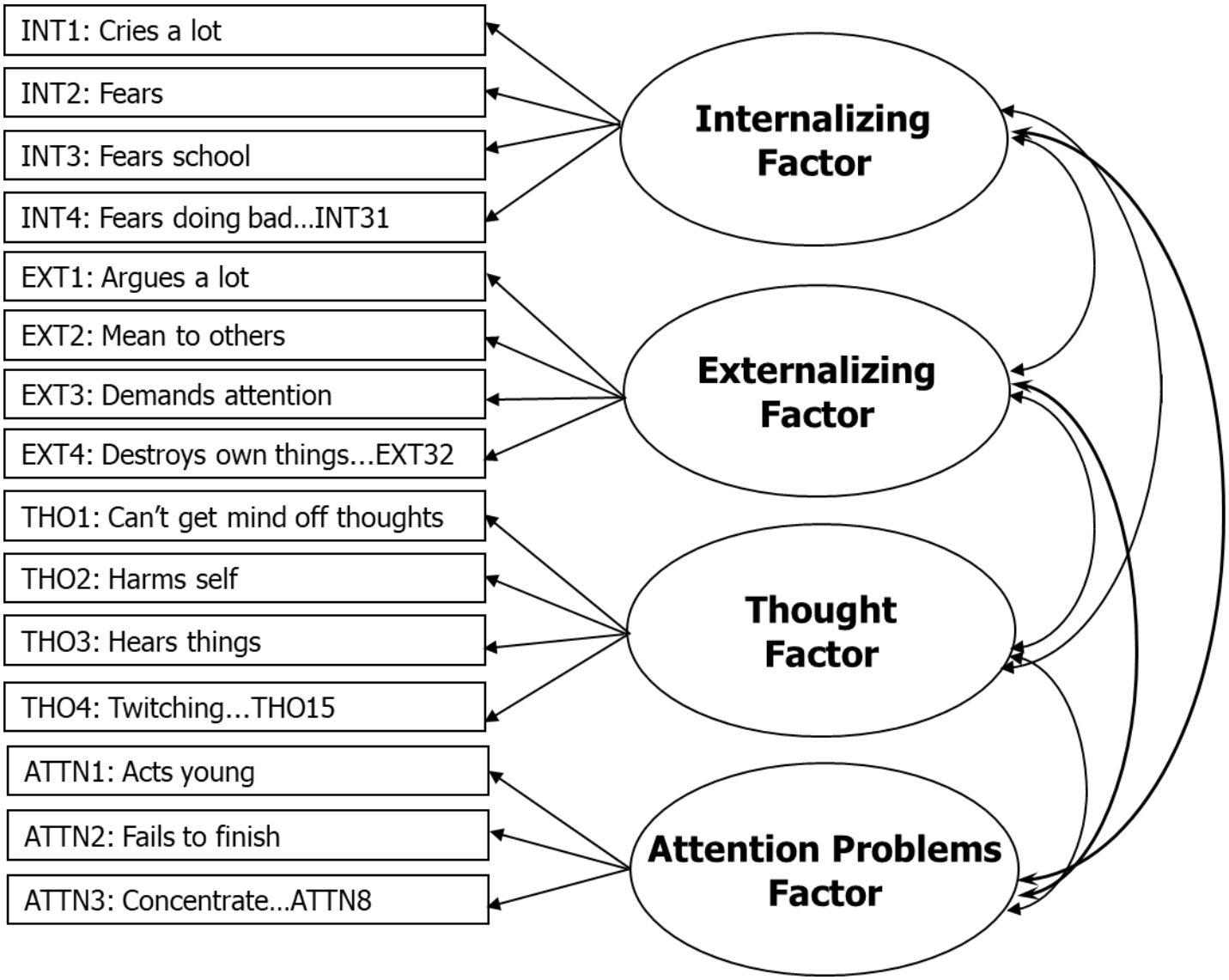
Note. Total number of items varies slightly between CBCL and YSR. Items shown in figure are for representational purposes; for full item descriptions, see supplementary tables below.

Figure S2. Two correlated factors model of psychopathology (**model B**).



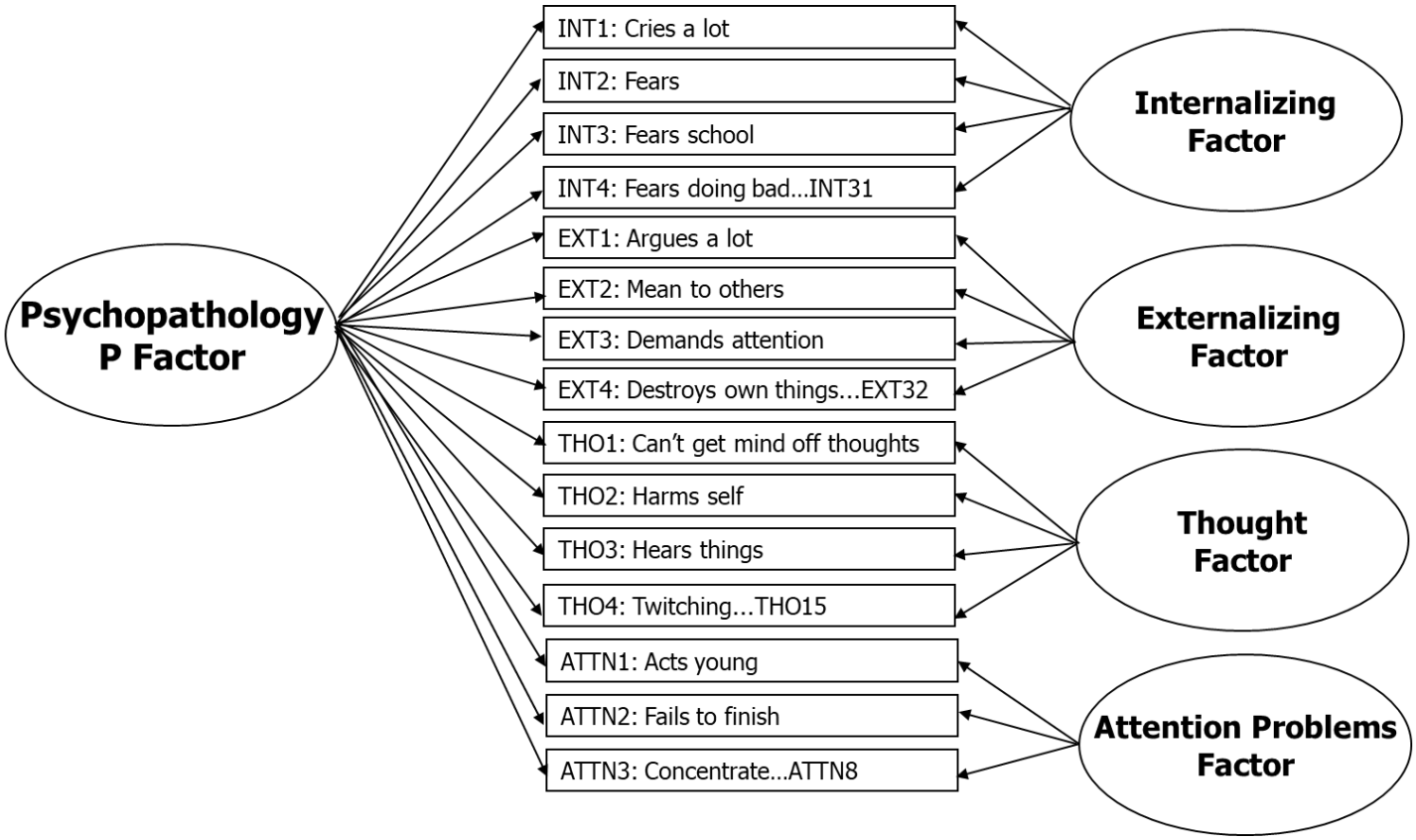
Note. Total number of items varies slightly between CBCL and YSR. Items shown in figure are for representational purposes; for full item descriptions, see supplementary tables below.

Figure S3. Four correlated factors model of psychopathology (**model C**).



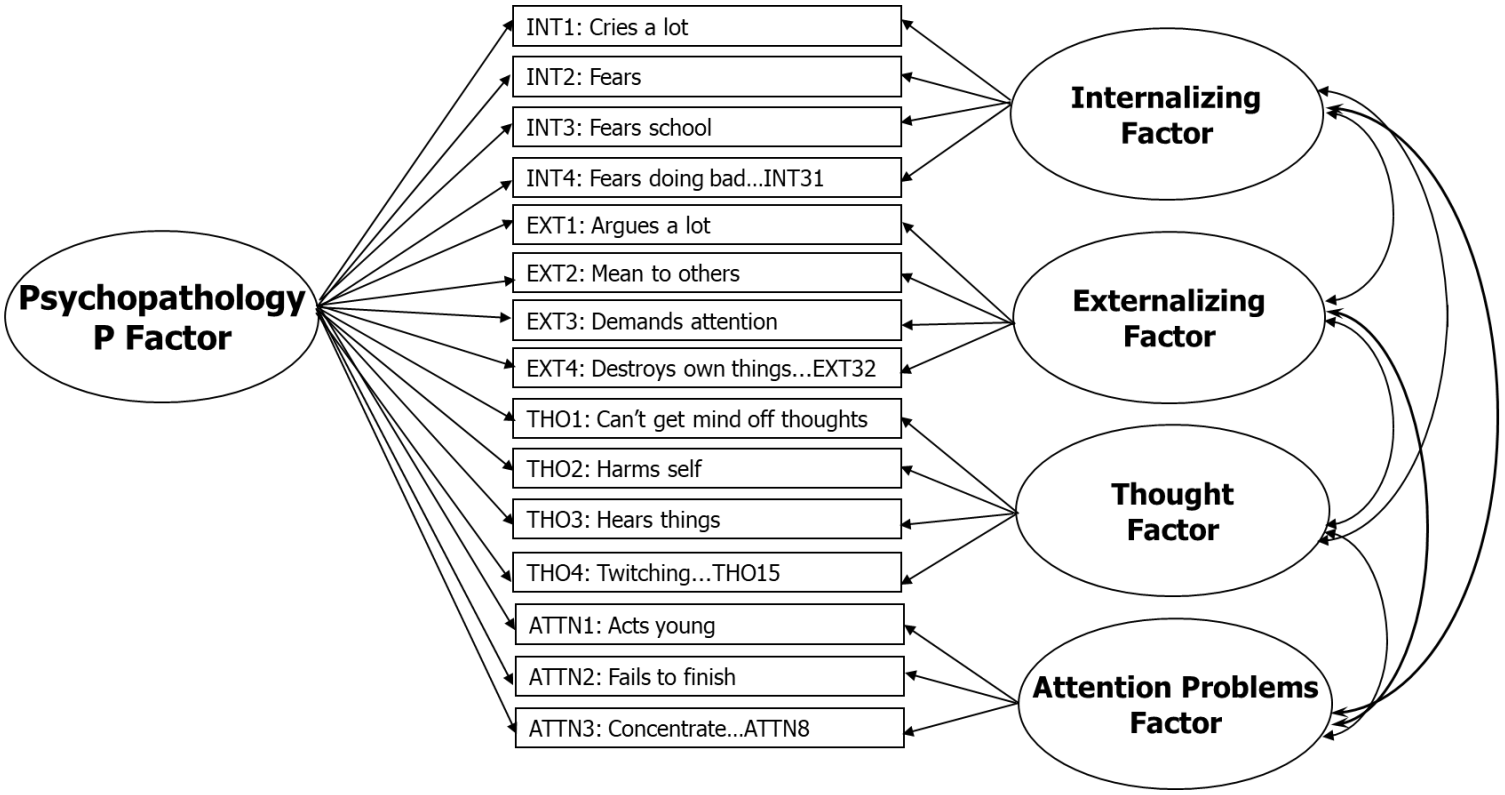
Note. Total number of items varies slightly between CBCL and YSR. Items shown in figure are for representational purposes; for full item descriptions, see supplementary tables below.

Figure S4. Orthogonal bifactor four specific factors model of psychopathology (**model D**).



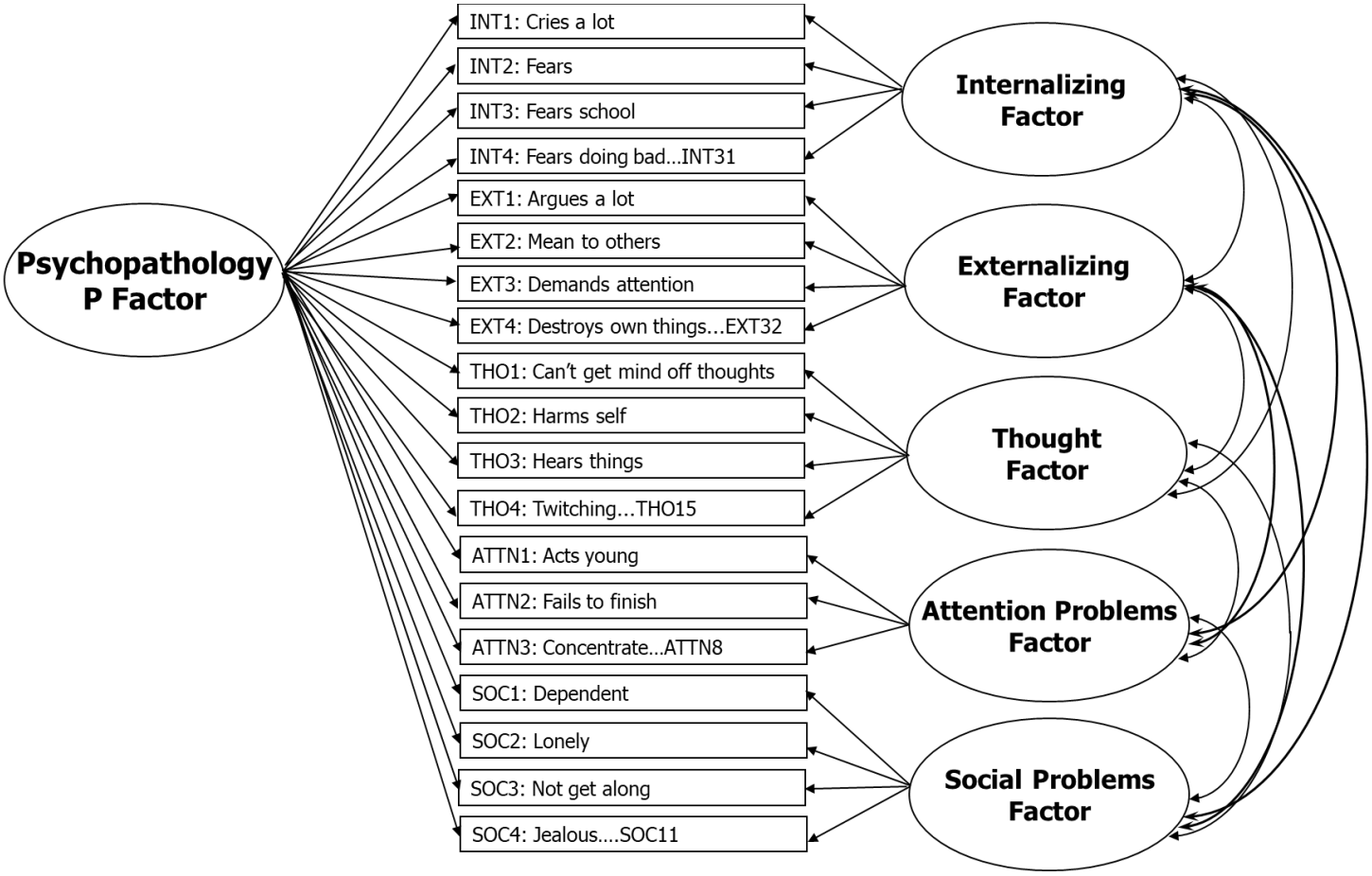
Note. Total number of items varies slightly between CBCL and YSR. Items shown in figure are for representational purposes; for full item descriptions, see supplementary tables below.

Figure S5. Correlated bifactor four specific factors model of psychopathology (**model E**).



Note. Total number of items varies slightly between CBCL and YSR. Items shown in figure are for representational purposes; for full item descriptions, see supplementary tables below.

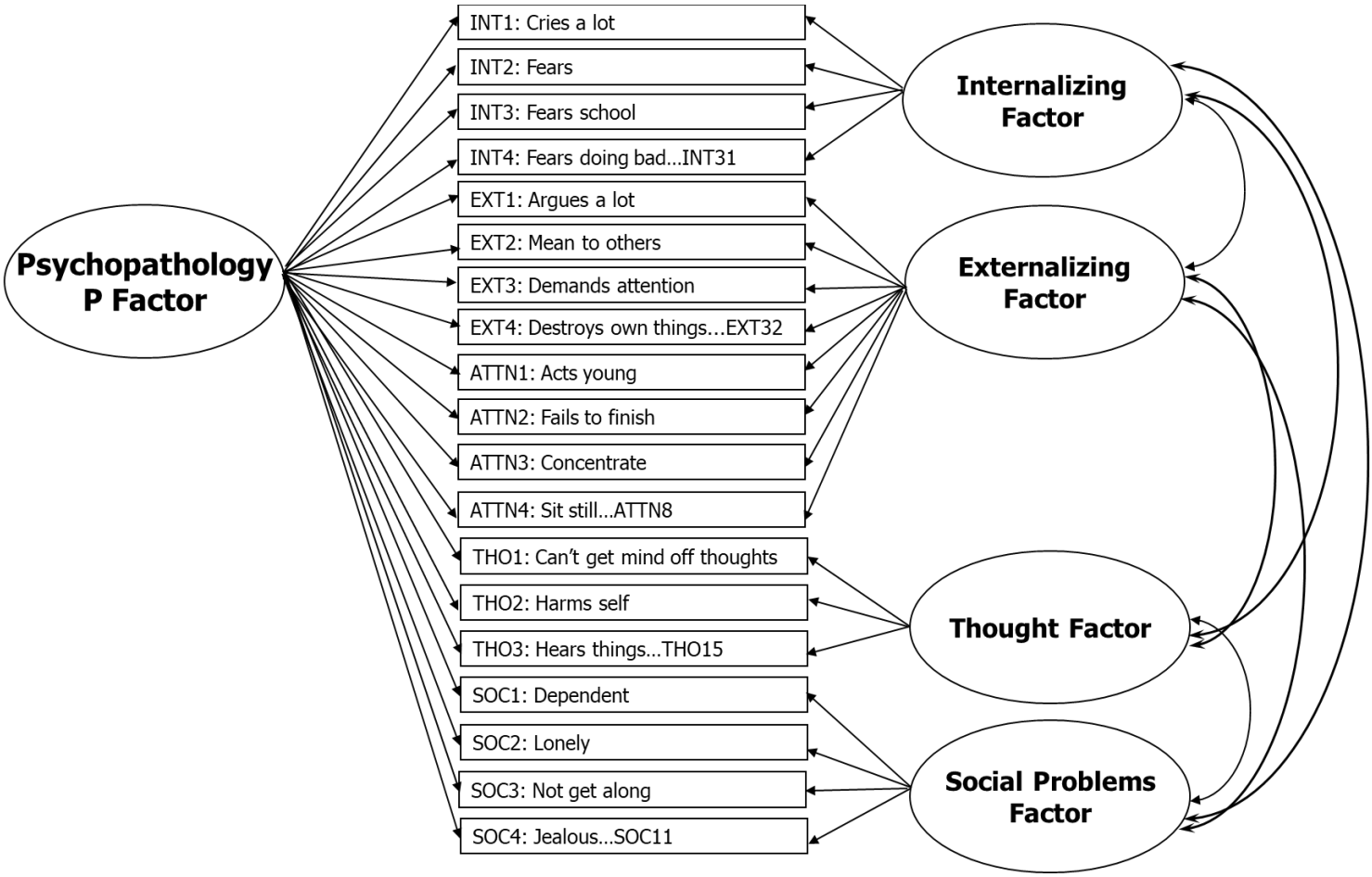
Figure S6. Alternate bifactor five factors model of psychopathology compared to a five correlated factors model of psychopathology (**model F**).



Note. Total number of items varies slightly between CBCL and YSR. In this model, the CBCL/YSR Social Problems syndrome is added as a separate psychopathology dimension. Items shown in figure are for representational purposes; for full internalizing, externalizing, thought, and attention item descriptions, see supplementary tables below. Social problem items are provided below. Note as with our focal models, only ASEBA items consistent in content across the 1991 and 2001 CBCL/YSR versions were modeled. **For CBCL**: correlated factors model, CFI = .77, RMSEA = .055 (90% C.I.: .054-.055); standard orthogonal bifactor (pictured above), CFI = .80, RMSEA = .051 (90% CI: .051-.052). **For YSR**: correlated factors model, CFI = .86, RMSEA = .046 (90% CI: .045 - .046); standard orthogonal bifactor (pictured above), CFI = .88, RMSEA = .044 (90% CI: .041 - .043).

Social Problems
Dependent
Lonely
Not get along
Jealous
Out to get
Accidents
Teased
Not liked
Clumsy
Prefer young
Speech problems

Figure S7. Revised bifactor 4 factors model of psychopathology compared to a revised correlated four factors model of psychopathology: Attention problems loading on the externalizing factor (**model G**).



Note. Total number of items varies slightly between CBCL and YSR. In this model, the CBCL/YSR Social Problems syndrome is added as a psychopathology dimension. Unlike Model F, however, in this model Attention Problem items are conceptualized as loading on the Externalizing factor rather than as comprising a separate problem dimension (factor). Items shown in figure are for representational purposes; for full internalizing, externalizing, thought, and attention problem item descriptions, see supplementary tables below. Social problem items are provided below. Note as with our focal models, only ASEBA items consistent in item content across the 1991 and 2001 CBCL/YSR versions were modeled. **For CBCL** correlated factors model, CFI = .76, RMSEA = .056 (90% CI: .056-.057); standard orthogonal bifactor (pictured above), CFI = .81, RMSEA = .050 (90% CI: .050 - .051). **For YSR** correlated factors model, CFI = .85, RMSEA = .048 (90% CI: .048 - .049); standard orthogonal bifactor (pictured above), CFI = .90, RMSEA = .039 (90% CI: .038 - .039).

Social Problems
Dependent
Lonely
Not get along
Jealous
Out to get
Accidents
Teased
Not liked
Clumsy
Prefer young
Speech problems

Supplementary Table S1. *Factor Loadings and Correlations for Additional Models for Child Behavior Checklist (CBCL) Data General Psychopathology "P" Factor Models*

Items	One Factor (Model A)	Two Correlated Factors (Model B)		Four Correlated Factors (Model C)				Nonorthogonal Bifactor (Correlated symptoms; Model E)				
	UNI	INT	EXT	INT	EXT	THO	ATT	**P**	INT	EXT	THO	ATT
Internalizing												
Cries a lot	.503	.589		.598				.320	.496			
Fears	.396	.474		.495				.179	.519			
Fears school	.413	.527		.518				.212	.497			
Fears doing bad	.462	.539		.557				.301	.447			
Must be perfect	.345	.457		.445				.137	.492			
Feels unloved	.604	.710		.699				.507	.381			
Feels worthless	.621	.746		.742				.400	.595			
Nervous, tense	.638	.726		.759				.433	.579			
Fearful, anxious	.583	.714		.721				.231	.768			
Feels too guilty	.361	.484		.482				.097	.583			
Self-conscious	.477	.597		.587				.270	.536			
Talk, thinks suicide	.564	.639		.651				.477	.347			
Worries	.568	.699		.701				.247	.721			
Rather be alone	.402	.497		.507				.203	.496			
Won't talk	.434	.503		.499				.395	.214			
Secretive	.504	.580		.575				.491	.183			
Shy, timid	.299	.424		.413				.052	.546			
Lacks energy	.484	.596		.594				.295	.497			
Sad	.640	.783		.768				.419	.602			
Withdrawn	.500	.607		.616				.282	.553			
Nightmares	.459	.509		.549				.288	.461			
Constipated	.328	.393		.402				.186	.367			
Feels dizzy	.460	.587		.587				.173	.637			
Overtired	.512	.618		.622				.314	.520			
Aches, pains	.399	.505		.502				.186	.507			
Headaches	.396	.532		.509				.149	.553			
Nausea	.529	.703		.674				.147	.768			
Eye problems	.304	.360		.376				.164	.353			
Skin problems	.292	.347		.353				.190	.285			
Stomachaches	.493	.653		.627				.151	.705			
Vomiting	.416	.545		.522				.189	.522			
Externalizing												
Argues a lot	.767		.812		.813			.753		-.321		
Mean to others	.679		.742		.730			.709		-.187		
Demands attention	.678		.693		.720			.624		-.446		
Destroys own things	.697		.741		.743			.729		-.153		

Destroys others' things	.739	.794	.788		.788	-.099		
Disobedient at home	.779	.828	.825		.809	-.177		
Disobedient at school	.578	.621	.636		.688	.122		
Gets in fights	.594	.650	.647		.644	-.101		
Attacks people	.634	.694	.684		.646	-.246		
Screams a lot	.679	.726	.721		.620	-.449		
Stubborn, sullen	.776	.812	.813		.712	-.442		
Mood changes	.784	.808	.817		.690	-.528		
Sulks	.658	.676	.688		.545	-.577		
Suspicious	.669	.693	.707		.677	-.202		
Teases a lot	.564	.608	.613		.590	-.177		
Temper	.776	.824	.817		.720	-.439		
Threatens others	.690	.757	.742		.727	-.167		
Loud	.604	.620	.648		.577	-.353		
Lacks guilt	.621	.674	.674		.695	-.017		
Bad friends	.450	.509	.501		.599	.405		
Lies, cheats	.684	.733	.737		.789	.150		
Prefers older kids	.355	.393	.392		.424	.072		
Runs away	.502	.551	.546		.597	.143		
Sets fires	.403	.444	.447		.503	.180		
Sex problems	.424	.415	.452		.444	-.090		
Steals at home	.611	.680	.669		.740	.338		
Steals outside home	.520	.592	.579		.667	.461		
Swearing	.584	.653	.636		.679	.083		
Thinks of sex	.505	.522	.544		.551	-.042		
Truant	.335	.398	.368		.476	.530		
Uses drugs	.367	.439	.409		.522	.600		
Vandalism	.593	.658	.651		.708	.188		
Thought								
Can't get mind off	.588			.700	.433		.547	
Harms self	.548			.645	.422		.457	
Hears things	.509			.615	.270		.660	
Twitching	.461			.553	.294		.518	
Picks skin	.451			.531	.354		.374	
Sex parts in public	.373			.441	.360		.175	
Sex parts too much	.417			.490	.390		.216	
Repeats acts	.492			.579	.429		.318	
Sees things	.497			.600	.260		.648	
Sleeps less	.436			.521	.293		.462	
Stores things	.422			.495	.339		.329	
Strange behavior	.570			.672	.491		.379	
Strange ideas	.540			.638	.445		.401	
Sleep talks/walks	.342			.401	.258		.301	
Trouble sleeping	.511			.614	.299		.622	

Attention												
Acts young	.418					.510	.384				-.285	
Can't concentrate	.596					.729	.535				-.460	
Can't sit still	.550					.670	.535				-.292	
Confused	.547					.660	.356				-.747	
Daydreams	.487					.593	.294				-.729	
Impulsive	.734					.896	.784				-.128	
Poor schoolwork	.485					.589	.509				-.156	
Stares	.558					.677	.398				-.674	
Factor correlations												
Internalizing		--	.537	--	.538	.747	.572		--	-.353	.689	-.488
Externalizing			--		--	.665	.730			--	-.393	.176
Thought						--	.739				--	-.589
Attention							--					--

Note. N = 2932. 2 cases were missing data on all relevant indicators and were not included in model estimation. ASEBA behavior problem items 5 (enjoys little), 2 (drinks alcohol), 28 (breaks rules), and 99 (uses tobacco) were not used as they are not common to both the 1991 CBCL 4/18 [27] and 2001 CBCL 6/18 [28] factor structures for internalizing and externalizing dimensions, respectively.

FP = free parameters, χ^2 = model chi-square; RMSEA = root mean square error of approximation; CI = confidence interval; CFI = comparative fit index; UNI = unidimensional; INT = internalizing; EXT = externalizing; THO = thought; ATT = attention; P = general psychopathology factor.

[a] As noted recently (see: http://www.statmodel.com/download/Bi-factor%20compared%20to%20correlated%20factors%20model.pdf) the classic orthogonal bifactor model in this case is not nested within the four correlated factors model. As such, MLR model estimation was also used for both the four correlated factors model and the standard orthogonal bifactor model. In this way, the Bayesian Information Criterion (BIC) was generated and used to compare the fit of these two models. The smaller BIC of the standard orthogonal bifactor model (360512.533) relative to the four correlated factors model (364842.165) suggests the bifactor model provides a better fit to the data. [b] Reference for χ^2 test for different testing (four correlated factors; Model C).

Supplementary Table S2. *Factor Loadings and Correlations for Additional Models for Child Behavior Checklist (CBCL) Data Dysregulation Profile "DP" General Factor Models*

Items	One Factor (Model A)	Three Correlated Factors (Model B)			Nonorthogonal Bifactor (Correlated Symptoms; Model D)			
	UNI	AD	AGG	ATT	**DP**	AD	AGG	ATT
Anxious Depressed								
Cries a lot	.529	.662			.164	.617		
Fears	.403	.528			.036	.549		
Fears school	.368	.491			.001	.531		
Fears doing bad	.455	.589			.093	.578		
Must be perfect	.346	.476			-.058	.556		
Feels unloved	.621	.771			.344	.596		
Feels worthless	.596	.758			.135	.733		
Nervous, tense	.611	.768			.171	.718		
Fearful, anxious	.575	.747			-.058	.830		
Feels too guilty	.328	.471			-.172	.618		
Self-conscious	.464	.600			.071	.609		
Talks or thinks of suicide	.530	.652			.317	.487		
Worries	.549	.717			-.051	.795		
Aggressive Behavior								
Argues a lot	.805		.829		.654		-.512	
Mean to others	.719		.745		.736		-.285	
Demands attention	.733		.756		.531		-.539	
Destroys own things	.725		.749		.708		-.317	
Destroys others' things	.763		.787		.777		-.292	
Disobedient at home	.800		.823		.726		-.423	
Disobedient at school	.596		.624		.669		-.184	
Gets in fights	.639		.666		.654		-.260	
Attacks people	.702		.728		.727		-.265	
Screams a lot	.729		.751		.543		-.520	
Stubborn, sullen	.789		.809		.491		-.658	
Mood changes	.785		.804		.386		-.759	
Sulks	.678		.696		.252		-.730	
Suspicious	.619		.638		.304		-.599	
Teases a lot	.597		.624		.584		-.283	
Temper	.826		.846		.636		-.560	
Threatens others	.724		.750		.731		-.297	
Loud	.648		.672		.530		-.417	
Attention Problems								
Acts young	.441			.536	.311			.427
Concentrate	.614			.749	.384			.662
Sit still	.605			.727	.494			.499

Confused	.475			.585	.021			.796
Daydream	.454			.562	.044			.741
Impulsive	.753			.923	.722			.482
Poor school	.445			.545	.355			.393
Stares	.512			.629	.136			.738
Factor correlations								
Anxious Depressed		--	.593	.549		--	-.780	.570
Aggressive Behavior			--	.722			--	-.583
Attention Problems				--				--

Note. N = 2932. 2 cases were missing data on all relevant indicators and were not included in model estimation.

FP = free parameters, χ^2 = model chi-square; RMSEA = root mean square error of approximation; CI = confidence interval; CFI = comparative fit index; UNI = unidimensional; AD = anxious-depressed; AGG = aggressive; ATT = attention; DP = general dysregulation factor.

Supplementary Table S3. *Confirmatory Factor Analysis Models for Youth Self Report (YSR) Data General Psychopathology "P" Factor Models*

		Correlated Models						Bifactor Models									
Items	One Factor (Model A)	Two Correlated Factors (Model B)		Four Correlated Factors (Model C)				Orthogonal (Standard bifactor; Model D)					Nonorthogonal (Correlated symptoms; Model E)				
	UNI	INT	EXT	INT	EXT	THO	ATT	**P**	INT	EXT	THO	ATT	**P**	INT	EXT	THO	ATT
Internalizing																	
Cries a lot	.615	.675		.671				.534	.459				.695	.088			
Fears	.338	.360		.378				.312	.226				.397	.065			
Fears school	.589	.642		.641				.522	.401				.657	.058			
Fears doing bad	.608	.640		.655				.601	.203				.643	-.110			
Must be perfect	.480	.533		.529				.420	.371				.567	.175			
Feels unloved	.663	.720		.712				.611	.356				.724	.054			
Feels worthless	.773	.827		.826				.689	.464				.843	.122			
Nervous, tense	.750	.802		.804				.663	.467				.815	.061			
Fearful, anxious	.699	.758		.753				.589	.531				.780	.169			
Feels too guilty	.545	.605		.600				.467	.439				.627	.127			
Self-conscious	.642	.702		.698				.560	.458				.727	.154			
Talk, thinks suicide	.727	.728		.779				.739	.168				.777	-.042			
Worries	.714	.774		.771				.616	.508				.800	.200			
Rather be alone	.442	.488		.488				.425	.216				.491	-.023			
Won't talk	.391	.424		.414				.420	-.003				.375	-.231			
Secretive	.555	.591		.594				.585	.052				.564	-.214			
Shy, timid	.429	.504		.485				.329	.495				.533	.256			
Lacks energy	.640	.694		.691				.598	.328				.680	-.125			
Sad	.781	.835		.835				.712	.422				.843	.053			
Withdrawn	.336	.364		.365				.342	.086				.360	-.051			
Nightmares	.556	.591		.601				.542	.220				.576	-.196			
Constipated	N/A	N/A		N/A				N/A	N/A				N/A	N/A			
Feels dizzy	.687	.735		.740				.641	.349				.695	-.314			
Overtired	.677	.724		.729				.651	.285				.706	-.195			
Aches, pains	.554	.594		.599				.529	.252				.538	-.375			
Headaches	.561	.622		.611				.495	.386				.528	-.471			
Nausea	.654	.725		.710				.544	.530				.614	-.527			
Eye problems	.452	.473		.491				.453	.141				.427	-.366			
Skin problems	.343	.364		.372				.344	.110				.336	-.229			
Stomachaches	.576	.648		.630				.464	.526				.532	-.518			
Vomiting	.539	.600		.586				.469	.385				.483	-.533			
Externalizing																	
Argues a lot	.577		.653		.661			.508		.384			.405		.501		
Mean to others	.555		.661		.646			.411		.558			.304		.613		
Demands attention	.417		.463		.478			.383		.233			.309		.337		
Destroys own things	.585		.641		.656			.558		.258			.482		.376		

Destroys others' things	.554	.655	.646	.422	.531	.313	.600
Disobedient at home	.647	.743	.741	.527	.525	.414	.615
Disobedient at school	.545	.656	.648	.359	.659	.214	.731
Gets in fights	.488	.606	.585	.317	.612	.202	.652
Attacks people	.481	.597	.576	.306	.614	.194	.646
Screams a lot	.607	.674	.683	.585	.255	.504	.385
Stubborn, sullen	.567	.637	.642	.524	.305	.439	.418
Mood changes	.726	.774	.808	.767	.077	.695	.264
Sulks	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Suspicious	.545	.597	.610	.532	.206	.473	.305
Teases a lot	.502	.604	.593	.363	.540	.254	.597
Temper	.588	.681	.678	.487	.474	.376	.570
Threatens others	.624	.741	.725	.446	.649	.322	.708
Loud	.482	.535	.558	.431	.313	.329	.441
Lacks guilt	.411	.504	.498	.284	.505	.178	.556
Bad friends	.501	.608	.597	.344	.591	.226	.642
Lies, cheats	.595	.689	.687	.484	.494	.376	.582
Prefers older kids	.392	.458	.453	.334	.303	.270	.357
Runs away	.557	.637	.635	.484	.384	.399	.468
Sets fires	.476	.531	.551	.410	.356	.308	.464
Sex problems	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Steals at home	.563	.650	.650	.458	.468	.362	.539
Steals outside home	.511	.626	.609	.341	.617	.231	.648
Swearing	.587	.681	.681	.470	.515	.356	.602
Thinks of sex	.452	.524	.524	.378	.366	.305	.420
Truant	.475	.564	.558	.360	.476	.270	.523
Uses drugs	.483	.577	.568	.363	.496	.277	.531
Vandalism	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Thought							
Can't get mind off	.588		.665	.634	.083	.602	.198
Harms self	.714		.801	.777	-.122	.751	.088
Hears things	.580		.655	.577	.572	.486	.564
Twitching	.625		.705	.672	.125	.627	.256
Picks skin	.539		.606	.582	.055	.535	.243
Sex parts in public	N/A		N/A	N/A	N/A	N/A	N/A
Sex parts too much	N/A		N/A	N/A	N/A	N/A	N/A
Repeats acts	.479		.537	.505	.222	.396	.486
Sees things	.546		.618	.534	.611	.448	.563
Sleeps less	.487		.547	.534	-.115	.456	.312
Stores things	.413		.467	.437	.220	.373	.329
Strange behavior	.587		.661	.596	.512	.471	.642
Strange ideas	.657		.740	.674	.490	.580	.541
Sleep talks/walks	N/A		N/A	N/A	N/A	N/A	N/A
Trouble sleeping	.612		.691	.671	-.109	.611	.258

Attention																				
Acts young	.277					.315	.289			.241	.220					.254				
Can't concentrate	.594					.672	.609			.635	.490					.493				
Can't sit still	.557					.630	.570			.489	.457					.464				
Confused	.725					.819	.794			-.153	.764					.079				
Daydreams	.512					.575	.552			.110	.519					.159				
Impulsive	.578					.654	.604			.178	.361					.753				
Poor schoolwork	.432					.491	.448			.231	.296					.506				
Stares	N/A					N/A	N/A			N/A	N/A					N/A				
Factor correlations																				
Internalizing		--	.579	--	.581	.841	.772							--	-.272	-.291	-.198			
Externalizing			--		--	.651	.777								--	.382	.690			
Thought						--	.759									--	.321			
Attention							--										--			
Model Fit																				
FP	234		178			240				312				318						
χ^2	32095.96		13243.23			19430.90				16220.07				12569.89						
χ^2 test for difference	--		--			--				N/A[a]				χ^2 (78) = 3054.38, p < .001[a]						
RMSEA	.065		.054			.049				.044				.038						
RMSEA 90% CI	[.064 - .065]		[.053 - .055]			[.048 - .049]				[.044 - .045]				[.037 - .039]						
CFI	.755					.862				.888				.918						

Note. N = 2390. 5 cases were missing data on all relevant indicators and were not included in model estimation. N/A = items not applicable on the YSR.

FP = free parameters, χ^2 = model chi-square; RMSEA = root mean square error of approximation; CI = confidence interval; CFI = comparative fit index; UNI = unidimensional; INT = internalizing; EXT = externalizing; THO = thought; ATT = attention; P = general psychopathology factor.

[a] As described recently (see: http://www.statmodel.com/download/Bi-factor%20compared%20to%20correlated%20factors%20model.pdf) the classic orthogonal bifactor model in this case is not nested within the four correlated factors model. As such, MLR model estimation was also used for both the four correlated factors model and the standard orthogonal bifactor model. In this way, the Bayesian Information Criterion (BIC) was generated and used to compare the fit of these two models. The smaller BIC of the standard orthogonal bifactor model (278037.861) relative to the four correlated factors model (280627.395) suggests the bifactor model provides a better fit to the data. [b] Reference for χ^2 test for different testing (four correlated factors; Model C).

Supplementary Table S4. *Confirmatory Factor Analysis Models for Youth Self-Report (YSR) Data Dysregulation Profile "DP" General Factor Models*

					Bifactor Models							
	One Factor (Model A)	Three Correlated Factors (Model B)			Orthogonal (Standard bifactor; Model C)				Nonorthogonal (Correlated Symptoms; Model D)			
Items	UNI	AD	AGG	ATT	**DP**	AD	AGG	ATT	**DP**	AD	AGG	ATT
Anxious Depressed												
Cries a lot	.621	.702			.523	.475			.691	.203		
Fears	.329	.386			.271	.301			.383	.110		
Fears school	.592	.669			.493	.460			.653	.130		
Fears doing bad	.610	.682			.576	.297			.649	-.060		
Must be perfect	.504	.583			.400	.470			.579	.212		
Feels unloved	.663	.735			.578	.428			.714	.095		
Feels worthless	.775	.854			.647	.549			.830	.186		
Nervous, tense	.758	.837			.634	.530			.809	.144		
Fearful, anxious	.718	.795			.563	.592			.776	.236		
Feels too guilty	.549	.632			.427	.521			.624	.243		
Self-conscious	.644	.726			.534	.504			.712	.183		
Talks or thinks of suicide	.662	.734			.612	.349			.709	.002		
Worries	.722	.806			.575	.591			.787	.258		
Aggressive Behavior												
Argues a lot	.626		.693		.565		.381		.478		.492	
Mean to others	.601		.672		.423		.614		.358		.631	
Demands attention	.466		.513		.452		.206		.385		.312	
Destroys own things	.601		.653		.577		.268		.516		.354	
Destroys others' things	.588		.659		.438		.556		.365		.600	
Disobedient at home	.649		.717		.562		.444		.474		.539	
Disobedient at school	.552		.629		.410		.555		.292		.665	
Gets in fights	.529		.605		.331		.648		.257		.664	
Attacks people	.520		.596		.293		.686		.234		.675	
Screams a lot	.646		.705		.630		.266		.560		.371	
Stubborn, sullen	.578		.637		.540		.305		.469		.399	
Mood changes	.715		.773		.783		.048		.705		.207	
Sulks	N/A		N/A		N/A		N/A		N/A		N/A	
Suspicious	.531		.574		.537		.159		.493		.230	
Teases a lot	.531		.602		.382		.545		.311		.582	
Temper	.628		.699		.508		.516		.425		.585	
Threatens others	.652		.730		.441		.688		.368		.707	
Loud	.511		.571		.468		.307		.382		.425	
Attention Problems												
Acts young	.331			.368	.358			.173	.293			.267
Concentrate	.623			.694	.655			.551	.574			.426

Sit still	.575			.639	.596			.474	.526			.397
Confused	.714			.796	.829			-.288	.768			-.046
Daydream	.492			.544	.544			.088	.522			.079
Impulsive	.602			.669	.654			.129	.476			.656
Poor school	.426			.475	.459			.201	.357			.404
Stares	N/A			N/A	N/A			N/A	N/A			N/A
Factor correlations												
Anxious Depressed	--		--	.564	.750					--	-.574	-.911
Aggressive Behavior	--			--	.787						--	.647
Attention Problems	--				--							--
Model Fit												
FP	111			114		148			151			
χ^2	14013.373			7887.689		5948.351			4011.850			
χ^2 test for difference						χ^2 (34) = 1439.499, p < .001[a]			χ^2 (37) = 1772.382, p < .001[a]			
RMSEA	.094			.070		.062			.049			
RMSEA 90% CI	[.093 - .096]			[.068 - .071]		[.060 - .063]			[.048 - .051]			
CFI	.766			.873		.906			.940			

Note. N = 2390. 5 cases were missing data on all relevant indicators and were not included in model estimation. N/A = items not applicable for the YSR.

FP = free parameters, χ^2 = model chi-square; RMSEA = root mean square error of approximation; CI = confidence interval; CFI = comparative fit index; UNI = unidimensional; AD = anxious-depressed; AGG = aggressive; ATT = attention; DP = general dysregulation factor.

[a] Reference for χ^2 test for different testing (three correlated factors; Model B).

Supplementary Table S5. *Confirmatory Factor Analysis Models for Child Behavior Checklist (CBCL) Data General Psychopathology "P" Factor Models (Boys Only)*

		Correlated Models						Bifactor Models									
Items	One Factor (Model A)	Two Correlated Factors (Model B)		Four Correlated Factors (Model C)				Orthogonal (Standard bifactor; Model D)					Nonorthogonal (Correlated symptoms; Model E)				
	UNI	INT	EXT	INT	EXT	THO	ATT	**P**	INT	EXT	THO	ATT	**P**	INT	EXT	THO	ATT
Internalizing																	
Cries a lot	.470	.542		.555				.458	.256				.317	.452			
Fears	.400	.464		.489				.366	.322				.212	.497			
Fears school	.413	.514		.505				.343	.401				.257	.444			
Fears doing bad	.470	.545		.560				.439	.297				.324	.440			
Must be perfect	.336	.432		.423				.285	.358				.167	.448			
Feels unloved	.595	.700		.687				.601	.183				.521	.339			
Feels worthless	.628	.751		.745				.568	.435				.445	.561			
Nervous, tense	.646	.726		.761				.618	.335				.478	.540			
Fearful, anxious	.591	.704		.717				.494	.563				.294	.734			
Feels too guilty	.364	.480		.473				.265	.513				.133	.566			
Self-conscious	.479	.591		.582				.425	.403				.312	.496			
Talk, thinks suicide	.550	.630		.633				.560	.146				.483	.309			
Worries	.580	.698		.703				.481	.555				.301	.700			
Rather be alone	.410	.492		.504				.362	.351				.250	.455			
Won't talk	.434	.511		.498				.425	.147				.411	.180			
Secretive	.500	.582		.569				.490	.128				.508	.130			
Shy, timid	.285	.393		.383				.200	.461				.081	.504			
Lacks energy	.467	.572		.569				.373	.465				.310	.462			
Sad	.633	.775		.755				.552	.490				.459	.549			
Withdrawn	.488	.586		.593				.421	.422				.310	.509			
Nightmares	.476	.524		.567				.462	.263				.305	.487			
Constipated	.331	.397		.407				.286	.308				.186	.395			
Feels dizzy	.458	.569		.573				.337	.571				.210	.614			
Overtired	.504	.601		.606				.423	.446				.338	.489			
Aches, pains	.404	.502		.501				.303	.492				.206	.512			
Headaches	.373	.498		.475				.238	.571				.158	.531			
Nausea	.504	.667		.638				.287	.788				.162	.757			
Eye problems	.296	.350		.366				.239	.323				.153	.374			
Skin problems	.288	.339		.344				.255	.226				.199	.270			
Stomachaches	.473	.622		.596				.288	.705				.167	.694			
Vomiting	.401	.518		.494				.273	.526				.214	.483			
Externalizing																	
Argues a lot	.755		.798		.800			.716		.311			.747		-.312		
Mean to others	.664		.726		.712			.576		.430			.685		-.217		
Demands attention	.691		.700		.730			.726		.076			.644		-.453		

Destroys own things	.703	.743	.745	.625	.395	.732	-.150
Destroys others' things	.748	.801	.793	.635	.495	.790	-.105
Disobedient at home	.778	.822	.820	.698	.423	.801	-.183
Disobedient at school	.610	.651	.660	.507	.470	.689	.051
Gets in fights	.591	.643	.638	.516	.386	.624	-.146
Attacks people	.636	.692	.682	.579	.345	.625	-.338
Screams a lot	.664	.707	.704	.671	.155	.612	-.459
Stubborn, sullen	.772	.805	.808	.765	.204	.742	-.355
Mood changes	.792	.815	.827	.812	.122	.753	-.381
Sulks	.634	.649	.664	.699	-.061	.572	-.450
Suspicious	.670	.696	.708	.630	.286	.714	-.037
Teases a lot	.568	.609	.612	.534	.266	.580	-.225
Temper	.778	.821	.816	.762	.240	.726	-.450
Threatens others	.696	.759	.744	.602	.450	.716	-.219
Loud	.609	.617	.649	.637	.085	.579	-.385
Lacks guilt	.602	.649	.651	.514	.428	.669	-.003
Bad friends	.440	.497	.484	.267	.600	.567	.437
Lies, cheats	.680	.726	.729	.543	.555	.779	.201
Prefers older kids	.345	.382	.378	.284	.289	.405	.081
Runs away	.479	.526	.520	.373	.440	.570	.198
Sets fires	.411	.450	.450	.316	.393	.494	.180
Sex problems	.397	.375	.419	.423	.027	.406	-.116
Steals at home	.613	.679	.666	.405	.686	.725	.392
Steals outside home	.522	.594	.575	.285	.734	.643	.520
Swearing	.601	.666	.649	.480	.511	.682	.069
Thinks of sex	.483	.495	.518	.463	.199	.523	-.038
Truant	.304	.370	.338	.080	.670	.435	.672
Uses drugs	.351	.426	.389	.099	.741	.480	.707
Vandalism	.606	.671	.659	.444	.598	.707	.204
Thought							
Can't get mind off	.598		.708	.642	.062	.457	.536
Harms self	.551		.643	.588	.020	.454	.404
Hears things	.514		.613	.529	.434	.281	.667
Twitching	.467		.557	.502	.107	.301	.537
Picks skin	.467		.546	.506	.124	.380	.364
Sex parts in public	.393		.463	.379	.744	.343	.261
Sex parts too much	.454		.531	.457	.642	.400	.286
Repeats acts	.515		.603	.553	.187	.448	.335
Sees things	.519		.620	.531	.487	.284	.669
Sleeps less	.455		.539	.501	-.400	.317	.472
Stores things	.427		.499	.460	.110	.356	.310
Strange behavior	.583		.684	.615	.321	.495	.405
Strange ideas	.570		.668	.599	.337	.472	.421
Sleep talks/walks	.352		.412	.381	.058	.268	.313
Trouble sleeping	.523		.622	.570	-.400	.339	.593

Attention																		
Acts young	.415						.502	.432			.242	.362					.328	
Can't concentrate	.591						.715	.573			.669	.507					.502	
Can't sit still	.575						.691	.585			.393	.530					.368	
Confused	.534						.644	.529			.421	.351					.723	
Daydreams	.479						.582	.469			.483	.293					.711	
Impulsive	.746						.902	.790			.116	.777					.168	
Poor schoolwork	.494						.592	.489			.277	.503					.178	
Stares	.559						.676	.563			.426	.396					.681	
Factor correlations																		
Internalizing		--	.569	--	.571	.754	.619						--	-.257	.666	.489		
Externalizing			--		--	.677	.723							--	-.349	-.145		
Thought						--	.752								--	.589		
Attention							--									--		
Model Fit																		
FP	258		190		264			344				350						
χ^2	37843.77		20641.19		27535.35			23881.43				20845.65						
RMSEA	.066		.067		.055			.051				.047						
RMSEA 90% CI	[.065 - .066]		[.066 - .067]		[.054 - .055]			[.051 - .052]				[.047 - .048]						
CFI	.705		.796		.794			.825				.851						

Note. n = 2236. 2 cases were missing data on all relevant indicators and were not included in model estimation. ASEBA behavior problem items 5 (enjoys little), 2 (drinks alcohol), 28 (breaks rules), and 99 (uses tobacco) were not used as they are not common to both the 1991 CBCL 4/18 and 2001 CBCL 6/18 factor structures for internalizing and externalizing dimensions, respectively.

FP = free parameters, χ^2 = model chi-square; RMSEA = root mean square error of approximation; CI = confidence interval; CFI = comparative fit index; UNI = unidimensional; INT = internalizing; EXT = externalizing; THO = thought; ATT = attention; P = general psychopathology factor.

[a] Reference for χ^2 test for different testing (four correlated factors; Model C).

Supplementary Table S6. *Confirmatory Factor Analysis Models for Youth Self Report (YSR) Data General Psychopathology "P" Factor Models (Boys Only).*

		Correlated Models						Bifactor Models									
	One Factor (Model A)	Two Correlated Factors (Model B)		Four Correlated Factors (Model C)				Orthogonal (Standard bifactor; Model D)					Nonorthogonal (Correlated symptoms; Model E)				
Items	UNI	INT	EXT	INT	EXT	THO	ATT	**P**	INT	EXT	THO	ATT	**P**	INT	EXT	THO	ATT
Internalizing																	
Cries a lot	.501	.557		.560				.446	.386				.550	.295			
Fears	.311	.333		.352				.289	.211				.347	.142			
Fears school	.541	.592		.596				.499	.330				.580	.165			
Fears doing bad	.602	.639		.656				.598	.205				.632	.002			
Must be perfect	.430	.485		.482				.385	.328				.475	.220			
Feels unloved	.624	.689		.681				.576	.355				.660	.163			
Feels worthless	.728	.793		.791				.662	.434				.765	.240			
Nervous, tense	.702	.763		.767				.627	.459				.741	.246			
Fearful, anxious	.658	.730		.723				.559	.530				.700	.337			
Feels too guilty	.522	.592		.584				.443	.460				.571	.311			
Self-conscious	.602	.670		.665				.538	.427				.648	.239			
Talk, thinks suicide	.676	.690		.734				.688	.171				.710	.062			
Worries	.668	.738		.735				.584	.496				.712	.318			
Rather be alone	.425	.479		.477				.404	.237				.460	.086			
Won't talk	.444	.494		.478				.460	.064				.448	-.158			
Secretive	.537	.579		.581				.566	.053				.548	-.153			
Shy, timid	.453	.541		.518				.346	.544				.508	.391			
Lacks energy	.580	.639		.635				.548	.297				.614	.083			
Sad	.730	.800		.796				.662	.445				.769	.242			
Withdrawn	.341	.379		.375				.337	.126				.358	-.016			
Nightmares	.509	.549		.560				.497	.219				.541	.051			
Constipated	N/A	N/A		N/A				N/A	N/A				N/A	N/A			
Feels dizzy	.665	.722		.727				.628	.339				.703	.090			
Overtired	.639	.687		.697				.627	.248				.673	.027			
Aches, pains	.542	.582		.594				.526	.236				.572	.017			
Headaches	.538	.604		.595				.480	.377				.575	.106			
Nausea	.614	.694		.677				.513	.521				.656	.192			
Eye problems	.454	.473		.498				.457	.139				.474	-.048			
Skin problems	.335	.359		.369				.334	.127				.356	.007			
Stomachaches	.528	.608		.590				.419	.537				.574	.233			
Vomiting	.512	.579		.568				.434	.432				.551	.148			
Externalizing																	
Argues a lot	.558		.622		.632			.479		.390			.448		.441		
Mean to others	.560		.656		.639			.412		.552			.401		.551		
Demands attention	.437		.468		.488			.421		.175			.396		.223		

Destroys own things	.579	.620	.637	.562	.216	.537	.253
Destroys others' things	.585	.673	.663	.465	.497	.448	.509
Disobedient at home	.659	.738	.738	.541	.501	.516	.525
Disobedient at school	.608	.701	.694	.434	.625	.404	.656
Gets in fights	.497	.605	.581	.326	.604	.316	.600
Attacks people	.536	.636	.616	.370	.586	.361	.580
Screams a lot	.570	.624	.634	.536	.266	.510	.306
Stubborn, sullen	.542	.602	.608	.484	.328	.457	.369
Mood changes	.692	.722	.756	.724	.089	.688	.152
Sulks	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Suspicious	.562	.608	.618	.554	.184	.536	.201
Teases a lot	.514	.604	.591	.381	.512	.366	.521
Temper	.579	.662	.658	.467	.483	.441	.515
Threatens others	.655	.755	.740	.491	.608	.478	.610
Loud	.511	.545	.574	.479	.250	.445	.317
Lacks guilt	.444	.526	.519	.321	.484	.301	.507
Bad friends	.518	.608	.598	.374	.547	.360	.551
Lies, cheats	.607	.688	.686	.498	.476	.475	.501
Prefers older kids	.352	.409	.403	.288	.299	.279	.304
Runs away	.549	.610	.613	.489	.332	.474	.346
Sets fires	.514	.557	.578	.466	.300	.438	.347
Sex problems	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Steals at home	.570	.643	.643	.477	.425	.463	.433
Steals outside home	.538	.636	.621	.382	.577	.374	.569
Swearing	.607	.690	.690	.483	.520	.461	.541
Thinks of sex	.487	.551	.550	.420	.342	.411	.339
Truant	.467	.543	.537	.350	.464	.342	.460
Uses drugs	.459	.546	.531	.331	.494	.331	.470
Vandalism	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Thought							
Can't get mind off	.556		.636	.595	.140	.578	.157
Harms self	.678		.768	.736	-.062	.708	-.029
Hears things	.592		.677	.584	.566	.577	.531
Twitching	.605		.691	.648	.143	.632	.127
Picks skin	.532		.603	.577	.003	.554	.077
Sex parts in public	N/A		N/A	N/A	N/A	N/A	N/A
Sex parts too much	N/A		N/A	N/A	N/A	N/A	N/A
Repeats acts	.514		.583	.541	.220	.514	.346
Sees things	.559		.641	.539	.622	.533	.591
Sleeps less	.517		.588	.566	-.088	.537	.088
Stores things	.388		.446	.405	.264	.391	.301
Strange behavior	.604		.687	.606	.527	.584	.594
Strange ideas	.641		.731	.648	.511	.631	.511
Sleep talks/walks	N/A		N/A	N/A	N/A	N/A	N/A
Trouble sleeping	.602		.687	.658	-.070	.630	.049

Attention																				
Acts young	.336						.382	.354			.217	.322					.198			
Can't concentrate	.592						.674	.605			.639	.548					.431			
Can't sit still	.554						.629	.566			.487	.523					.349			
Confused	.693						.784	.760			-.154	.737					-.113			
Daydreams	.492						.556	.528			.132	.508					.075			
Impulsive	.568						.644	.595			.171	.473					.652			
Poor schoolwork	.482						.547	.506			.174	.432					.415			
Stares	N/A						N/A	N/A			N/A	N/A					N/A			
Factor correlations																				
Internalizing		--	.590	--	.593	.824	.759						--	-.564	-.374	-.550				
Externalizing			--		--	.652	.776							--	.085	.580				
Thought						--	.750								--	-.005				
Attention							--									--				
Model Fit																				
FP	234		178		240				312					318						
χ^2	20950.41		8886.10		13038.64				11136.60					9888.94						
RMSEA	.058		.049		.043				.040					.037						
RMSEA 90% CI	[.057 - .058]		[.048 - .050]		[.043 - .044]				[.039 - .040]					[.036 - .037]						
CFI	.773		.877		.873				.896					.911						

Note. n = 1852. 5 cases were missing data on all relevant indicators and were not included in model estimation. N/A = items not applicable on the YSR.

FP = free parameters, χ^2 = model chi-square; RMSEA = root mean square error of approximation; CI = confidence interval; CFI = comparative fit index; UNI = unidimensional; INT = internalizing; EXT = externalizing; THO = thought; ATT = attention; P = general psychopathology factor.

[a] Reference for χ^2 test for different testing (four correlated factors; Model C).

Supplementary Table S7. *Confirmatory Factor Analysis Models for Child Behavior Checklist (CBCL) Data Dysregulation Profile "DP" General Factor Models (Boys Only).*

					Bifactor Models							
	One Factor (Model A)	Three Correlated Factors (Model B)			Orthogonal (Standard bifactor; Model C)				Nonorthogonal (Correlated Symptoms; Model D)			
Items	UNI	AD	AGG	ATT	**DP**	AD	AGG	ATT	**DP**	AD	AGG	ATT
Anxious Depressed												
Cries a lot	.508	.626			.488	.314			.223	.563		
Fears	.409	.520			.342	.441			.109	.529		
Fears school	.378	.484			.318	.411			.095	.499		
Fears doing bad	.465	.588			.407	.443			.151	.576		
Must be perfect	.347	.459			.273	.476			.029	.527		
Feels unloved	.614	.753			.613	.259			.405	.537		
Feels worthless	.607	.758			.549	.488			.235	.704		
Nervous, tense	.619	.768			.572	.446			.265	.689		
Fearful, anxious	.585	.741			.452	.699			.071	.814		
Feels too guilty	.338	.465			.225	.609			-.096	.633		
Self-conscious	.466	.588			.423	.398			.161	.570		
Talks or thinks of suicide	.528	.642			.527	.215			.362	.445		
Worries	.557	.710			.427	.691			.051	.795		
Aggressive Behavior												
Argues a lot	.793		.817		.745		.331		.702		-.423	
Mean to others	.700		.725		.540		.593		.767		-.173	
Demands attention	.743		.765		.744		.176		.579		-.500	
Destroys own things	.724		.746		.592		.510		.724		-.263	
Destroys others' things	.766		.789		.609		.584		.799		-.231	
Disobedient at home	.799		.821		.728		.387		.751		-.361	
Disobedient at school	.627		.652		.559		.359		.660		-.202	
Gets in fights	.633		.657		.513		.483		.656		-.213	
Attacks people	.701		.725		.533		.601		.743		-.202	
Screams a lot	.719		.740		.682		.283		.599		-.435	
Stubborn, sullen	.787		.806		.788		.178		.566		-.584	
Mood changes	.795		.814		.832		.076		.484		-.703	
Sulks	.654		.672		.740		-.116		.288		-.704	
Suspicious	.608		.627		.637		.061		.351		-.559	
Teases a lot	.593		.617		.521		.362		.586		-.247	
Temper	.827		.846		.773		.340		.687		-.493	
Threatens others	.726		.751		.568		.585		.755		-.232	
Loud	.651		.673		.646		.181		.551		-.387	
Attention Problems												
Acts young	.426			.516	.428			.261	.281			.431
Concentrate	.603			.731	.569			.600	.362			.663

Sit still	.625			.745	.626			.334	.487			.520
Confused	.460			.567	.411			.578	.037			.789
Daydream	.448			.554	.393			.598	.069			.732
Impulsive	.767			.935	.815			.110	.716			.470
Poor school	.451			.547	.442			.330	.334			.411
Stares	.509			.624	.467			.564	.150			.740
Factor correlations												
Anxious Depressed		--	.623	.592					--	-.750	.560	
Aggressive Behavior			--	.722						--	-.606	
Attention Problems				--							--	
Model Fit												
FP	117		120		156			159				
χ^2	14414.393		8839.432		6535.909			5463.729				
RMSEA	.093		.072		.063			.057				
RMSEA 90% CI	[.092 - .095]		[.071 - .074]		[.062 - .064]			[.056 - .058]				
CFI	.804		.883		.916			.931				

Note. n = 2236. 2 cases were missing data on all relevant indicators and were not included in model estimation. ASEBA behavior problem items 5 (enjoys little), 2 (drinks alcohol), 28 (breaks rules), and 99 (uses tobacco) were not used as they are not common to both the 1991 CBCL 4/18 and 2001 CBCL 6/18 factor structures for internalizing and externalizing dimensions, respectively.

FP = free parameters, χ^2 = model chi-square; RMSEA = root mean square error of approximation; CI = confidence interval; CFI = comparative fit index; UNI = unidimensional; AD = anxious-depressed; AGG = aggressive; ATT = attention; DP = general dysregulation factor.

[a] Reference for χ^2 test for different testing (three correlated factors; Model B).

Supplementary Table S8. *Confirmatory Factor Analysis Models for Youth Self-Report (YSR) Data Dysregulation Profile "DP" General Factor Models (Boys Only).*

					Bifactor Models							
	One Factor (Model A)	Three Correlated Factors (Model B)			Orthogonal (Standard bifactor; Model C)				Nonorthogonal (Correlated Symptoms; Model D)			
Items	UNI	AD	AGG	ATT	**DP**	AD	AGG	ATT	**DP**	AD	AGG	ATT
Anxious Depressed												
Cries a lot	.510	.599			.439	.416			.551	.291		
Fears	.303	.366			.260	.275			.339	.158		
Fears school	.544	.630			.476	.398			.586	.194		
Fears doing bad	.598	.684			.566	.309			.643	.067		
Must be perfect	.450	.537			.374	.421			.495	.279		
Feels unloved	.614	.702			.533	.436			.648	.235		
Feels worthless	.724	.823			.613	.541			.755	.313		
Nervous, tense	.705	.807			.602	.520			.740	.282		
Fearful, anxious	.666	.763			.530	.586			.694	.364		
Feels too guilty	.517	.616			.397	.547			.555	.383		
Self-conscious	.593	.691			.509	.470			.639	.272		
Talks or thinks of suicide	.621	.705			.575	.348			.663	.119		
Worries	.668	.775			.542	.590			.703	.391		
Aggressive Behavior												
Argues a lot	.607		.663		.537		.378		.476		.465	
Mean to others	.610		.669		.422		.619		.395		.613	
Demands attention	.487		.523		.500		.123		.453		.213	
Destroys own things	.597		.637		.578		.231		.544		.281	
Destroys others' things	.616		.673		.474		.526		.441		.543	
Disobedient at home	.667		.721		.583		.415		.526		.486	
Disobedient at school	.616		.676		.495		.498		.420		.593	
Gets in fights	.541		.604		.348		.626		.312		.634	
Attacks people	.576		.637		.358		.663		.336		.644	
Screams a lot	.610		.658		.574		.287		.531		.350	
Stubborn, sullen	.558		.608		.505		.319		.453		.393	
Mood changes	.681		.722		.737		.059		.687		.150	
Sulks	N/A		N/A		N/A		N/A		N/A		N/A	
Suspicious	.551		.584		.566		.124		.540		.161	
Teases a lot	.552		.608		.412		.510		.373		.537	
Temper	.623		.683		.490		.524		.441		.567	
Threatens others	.686		.747		.483		.656		.457		.651	
Loud	.541		.589		.520		.237		.461		.341	
Attention Problems												
Acts young	.392			.436	.428			.133	.398			.204
Concentrate	.623			.695	.646			.573	.622			.366

Sit still	.573			.639	.587			.477	.577			.305
Confused	.681			.757	.785			-.249	.741			-.172
Daydream	.470			.521	.512			.130	.507			.042
Impulsive	.597			.662	.647			.104	.555			.544
Poor school	.473			.526	.513			.145	.465			.307
Stares	N/A			N/A	N/A			N/A	N/A			N/A
Factor correlations												
Anxious Depressed		--	.565	.734					--	-.401	-.878	
Aggressive Behavior			--	.788						--	.552	
Attention Problems												
Model Fit												
FP	111		114		148			151				
χ^2	9131.778		5064.167		3835.928			2915.751				
RMSEA	.085		.062		.054			.046				
RMSEA 90% CI	[.084 - .087]		[.060 - .063]		[.053 - .056]			[.045 - .048]				
CFI	.779		.884		.916			.939				

Note. n = 1852. 5 cases were missing data on all relevant indicators and were not included in model estimation. N/A = items not applicable for the YSR.

FP = free parameters, χ^2 = model chi-square; RMSEA = root mean square error of approximation; CI = confidence interval; CFI = comparative fit index; UNI = unidimensional; AD = anxious-depressed; AGG = aggressive; ATT = attention; DP = general dysregulation factor.

[a] Reference for χ^2 test for different testing (three correlated factors; Model B).

Supplementary Table S9. *Measurement Invariance of the Child Behavior Checklist (CBCL) and Youth Self-Report (YSR) Data Dysregulation Profile "DP" General Factor (Model C, Orthogonal, Standard Bifactor) across Sex*

Model	χ^2	df	RMSEA	RMSEA 90% CI	CFI	Δ df	Δ CFI	Δ RMSEA
Child Behavior Checklist (CBCL)								
Model 1: less restrictive model [a]	5637.964	1326	.047	[.046 – .048]	0.938	--	--	--
Model 2: metric and scalar invariance [b]	5288.393	1400	.044	[.042 – .045]	0.944	74	0.006	-0.003
Youth Self-Report (YSR)								
Model 1: less restrictive model [a]	3858.088	1184	.043	[.042 – .045]	0.935	--	--	--
Model 2: metric and scalar invariance [b]	3762.556	1254	.041	[.039 – .042]	0.939	70	0.004	-0.002

Note. [a] = Thresholds and factor loadings were free across groups, scale factors were fixed at one in both groups, and factor means were fixed at zero in both groups. [b] = Thresholds and factor loadings were constrained to be equal across groups, scale factors were fixed at one in one group and free in the other, and factor means were fixed at zero in one group and free in the other. This model tested metric and scalar (intercept) invariance. Δ in CFI and Δ RMSEA were used to adjudicate whether the invariance hypothesis should not be rejected as in Deutz et al. (2016).[26] Values of change in CFI $\leq$ 0.01 and Δ RMSEA $\leq$ 0.015 indicate that the invariance hypothesis should not be rejected.[29]

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Correlates of Childhood vs. Adolescence Internalizing Symptomatology from Infancy to Young Adulthood

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EMPIRICAL RESEARCH

Correlates of Childhood vs. Adolescence Internalizing Symptomatology from Infancy to Young Adulthood

John D. Haltigan[1] · Glenn I. Roisman[2] · Elizabeth Cauffman[3] · Cathryn Booth-LaForce[4]

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Abstract In light of its associations with child and adolescent health and well-being, there remains a need to better understand the etiological underpinnings and developmental course of internalizing symptomatology in children and adolescents. This study leveraged intensive longitudinal data ($N = 959$; 49.6 % females) to test the hypothesis that internalizing symptoms in childhood may be driven more strongly by family experiences whereas internalizing symptoms in adolescence may derive more uniquely from familial loading for affective disorders (i.e., maternal depression). We evaluated the relative contributions of (a) family experiences (b) maternal depression, and (c) peer influences in testing this hypothesis. The results indicated that family predictors were more strongly correlated with childhood (relative to adolescent) internalizing symptoms. In contrast to previous findings, maternal depression also exhibited stronger associations with childhood internalizing symptoms. Although often overlooked in theories concerning potential differential origins of childhood vs. adolescent internalizing symptomatology, peer experiences explained unique variation in *both* childhood and adolescent internalizing problems.

Keywords Environment · Internalizing symptomatology · Peer victimization · Psychopathology · Puberty

Introduction

Internalizing symptomatology refers to an empirically-derived cluster of symptoms that indicate problems in regulating intropunitive emotions and moods, including problems related to anxiety, fear, shyness, low self-esteem, sadness, and depression (Achenbach and Edelbrock 1978; Crawford et al. 2001; Graber and Sontag 2009; Kovacs and Devlin 1998; Ollendick and King 1994; Zahn-Waxler et al. 2000). From a developmental psychopathology perspective, which has as its goal understanding the processes and pathways that lead to developmental success or limitation and to improve the lives of individuals at risk for mental health problems (Cicchetti 1984; Sroufe and Rutter 1984), research efforts that aim to identify and better understand the antecedents, developmental course, and sequalae of internalizing symptomatology are of particular importance.

A unique feature of internalizing symptomatology is its well documented rise during the adolescent period, particularly for girls (Kovacs and Devlin 1998; Zahn-Waxler et al. 2000, Zahn-Waxler 2000). Empirical efforts to illuminate potential causal factors contributing to this rise have been diverse, including a focus on biological, cognitive, and social factors (Graber and Sontag 2009; Zahn-Waxler et al. 2000; Zahn-Waxler 2000). The rise in internalizing symptoms during adolescence also raises the possibility that different etiological factors may underlie internalizing problems that begin prior to adolescence as compared to those that begin during adolescence. Such a possibility is also consistent with the developmental chronology of anxiety

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and depressive symptoms, in which early childhood appears to be the high-risk period for the onset of anxiety symptoms and late childhood or early adolescence to be the high-risk period for the onset of depressive symptoms (Brady and Kendall 1992; Kovacs and Devlin 1998). Given that anxiety and depressive symptoms are moderately to highly correlated both with each other (Brady and Kendall 1992; Seligman and Ollendick 1998) as well as with higher-order internalizing syndrome constructs (e.g., Achenbach and Edelbrock 1978), it is not unreasonable to consider the possibility that, in addition to a large pool of common risk or etiological factors, there may also be unique developmental antecedents and correlates (e.g., family vs. peer relationships) that are differentially associated with internalizing symptomatology in childhood and adolescence. A third stream of evidence for the possibility that different developmental factors may underlie internalizing problems that begin prior to adolescence and those that emerge during adolescence comes from studies investigating distinct correlates of *depressive problems or symptoms* that begin in childhood (prepubertal or early-onset) relative to those that begin in adolescence (pubertal or postpubertal onset; Harrington et al. 1996, 1997; Silberg et al. 1999).

Despite the possibility that childhood and adolescent internalizing symptomatology may reflect distinctive developmental phenomena, large scale, multi-domain developmental investigations of internalizing problems in youth have rarely directly examined this possibility. Indeed, internalizing problems have historically received less theoretical attention and large-scale empirical scrutiny than have longitudinal and classificatory analyses of childhood and adolescent *externalizing* symptomatology and antisocial behavior (Cicchetti and Natsuaki 2014; Ollendick and King 1994; Rubin and Mills 1991), such as Moffitt's (1993) seminal taxonomy of life-course persistent and adolescence-limited antisocial life-course patterns (e.g., Roisman et al. 2010). As such, there remains an important basic and applied need to leverage high-quality prospective data to further explore the possibility of developmental heterogeneity in internalizing symptoms across childhood and adolescence (Cicchetti and Natsuaki 2014). In an effort to address this need, the objective of the current inquiry was to use prospective, multi-informant data from the NICHD Study of Early Child Care and Youth Development (SECCYD) to examine unique and overlapping correlates of childhood and adolescent internalizing symptomatology. Below we briefly review empirical work with *depressive problems or symptoms* that has provided a key stimulus for the broader notion that internalizing symptomatology that begins in childhood and adolescence may reflect distinct developmental phenomena. Using this work as a point of departure, its limitations are discussed and its relevance to the current study is highlighted.

The notion that internalizing symptomatology in childhood and adolescence may reflect distinct developmental phenomena demarcated by timing of occurrence can be traced to research conducted by Harrington and colleagues (1997). They found that a family history of mania or hypomania was more common in postpubertal depressed cases whereas prepubertal cases of depression tended to have lower familial rates of depression, higher rates of criminality among first-degree relatives, and higher rates of retrospectively reported maternal criticism/hostility directed toward the child (i.e., an "environmental risk factor"). Harrington et al. (1997) thus suggested that postpubertal onset depressive disorders may have a higher heritability (i.e., higher familial loading for depression) than prepubertal onset depressive disorders—which in contrast may be more associated with adverse family environments (although see Kovacs and Devlin 1998 for a different interpretation of these findings). Subsequent to the Harrington et al. (1997) finding, a growing body of behavior-genetic research (Murray and Sines 1996; Scourfield et al. 2003; Silberg et al. 1999; Thapar and McGuffin 1996) has provided evidence that family discord and shared environmental factors are more strongly associated with depressive symptoms in childhood whereas genetic factors are more strongly associated with depressive symptoms in adolescence. Nevertheless, findings of developmental change in the etiology of depression symptoms have not been unequivocal and some studies have failed to find an increasing heritability of depressive symptoms in adolescence (e.g., Gjone et al. 1996; O'Connor et al. 1998a, b).

Duggal et al. (2001) were the first to *prospectively* evaluate the possibility of different developmental pathways to depressive and anxious symptomatology in childhood and adolescence using data ($n = 168$) drawn from the Minnesota Longitudinal Study of Risk and Adaptation (MLSRA). A particular strength of their study was the availability of data on observed early family relationships during the first 3.5 years of life that could be used to evaluate more directly the idea that early family adversity would be more strongly associated with depressive problems occurring in childhood rather than adolescence (e.g., Harrington et al. 1996, 1997; Thapar and McGuffin 1996). Duggal et al. (2001) found that psychosocial factors (supportive early care, parenting support, abuse, and early maternal stress) accounted for 13 % of the variance in childhood depressive symptomatology even after accounting for the effects of maternal depression (which accounted for 6 % of the variance in childhood depressive symptoms). In contrast, maternal depression was more strongly associated with *adolescent* depressive symptomatology, accounting for 10 % of the variance in depressive symptomatology while psychosocial variables accounted for 9 % of the variance. When comparing extreme groups (i.e., using

clinical cutoffs), both abuse and early family stress were more strongly associated with childhood than to adolescent depressive symptomatology. Moreover, maternal depression was the only covariate to distinguish the adolescent depressive symptomatology group from controls (i.e., never depressed). These findings were in line with the above work suggesting a higher familial loading for adolescent depression and provided preliminary support for the notion of distinct depressive subgroups defined by their timing of onset (childhood vs. adolescent).

Despite a number of strengths of this study—especially concerning prospective measurement of family adversity—Duggal et al. (2001) did not control for stability in depressive and anxious symptomatology in their continuous analyses of child and adolescent depression. In addition, demarcation of the timing of depressive symptomatology (i.e., childhood or adolescence) was made on the basis of *age* rather than *pubertal status.* Pubertal status and the dynamic changes in hormonal status associated with the pubertal process have been shown to be stronger predictors than chronological age of the gender disparity in unipolar depressive disorders that emerge during adolescence and to the emergence of major depressive disorder (Angold et al. 1998) and negative (depressive and aggressive) affect (Brooks-Gunn and Warren 1989). Moreover, adolescence is often operationalized as a broad interval of maturation encompassing physical, mental, and socioemotional development that results in entry into the social world of adults (Graber and Brooks-Gunn 1996), whereas puberty encompasses a more specific set of processes involved in physical and reproductive functional reorganization that permit greater precision in measurement (Dorn et al. 2006). Finally, in addition to the conceptual and methodological advantages of demarcating childhood and adolescence on the basis of pubertal status, puberty (and school events) are frequently studied as key transitions signaling the entry into adolescence (Graber and Brooks-Gunn 1996).

In addition to the limitations of the Duggal et al. (2001) study, a limitation common to much of the existing work on the possibility of distinctive pathways to child- and adolescent internalizing problems has been that both family and peer researchers have had a tendency to construct models of depressive and internalizing symptomatology that have omitted high quality variables from the others' domain of inquiry, limiting an understanding of the potentially unique effects of family and peer experiences on internalizing problems. In particular, family experience researchers exploring distinctive pathways to child- and adolescent internalizing symptomatology have often neglected to consider the role of peer influences, especially those occurring during later childhood and adolescence (although see, for example, Criss et al. 2009). This is surprising in light of both theoretical work surrounding the importance of peer group socialization (Harris 1995) and peer relationship processes (Rose and Rudolph 2006) for development and empirical evidence that experiences of peer victimization in childhood and adolescence are associated reliably with both concurrent and later depressive and internalizing symptomatology, as well as other forms of maladjustment (Criss et al. 2009; Bowes et al., 2015; Kretschmer et al. 2015; Rose and Rudolph 2006). These findings have been substantiated by both cross-sectional (Hawker and Boulton 2000) and longitudinal (Reijntjes et al. 2010) meta-analytic studies that generally have found modest associations between victimization and internalizing problems. Moreover, given the increasing developmental salience of establishing and maintaining positive peer relations (i.e., peer competence) during later childhood and early adolescence (Sroufe and Rutter 1984), children's friendship quality might also be expected to influence the development of internalizing symptomatology either directly or via its effects on experiences of peer victimization (e.g., Hodges et al. 1999; Waldrip et al. 2008). Increased understanding of whether peer victimization experiences and children's and adolescents' perceived quality of peer friendships track differentially with internalizing symptomatology in childhood or adolescence has the potential to be of both basic and applied value and build upon the extensive body of research that has shown clear evidence for the association of peer victimization with depressive and internalizing symptomatology across childhood and adolescence.

The Current Study

Despite good reason to believe that childhood vs. adolescent internalizing symptomatology may represent distinct developmental phenomena with unique etiological underpinnings, little research to date has evaluated this possibility directly (although see Sterba et al. 2007). As such, the objective of the present study was to provide the first large-sample, prospective examination of unique and overlapping correlates of internalizing problems that occur prior to and following pubertal onset by leveraging data from the NICHD Study of Early Child Care and Youth Development (SECCYD). Data from the SECCYD are particularly well-suited for this purpose as the study includes multi-informant data on children's internalizing symptoms, high quality assessments of the family, multi-informant data on children's peer experiences collected over an 18-year period, and measures of maternal depression. In addition, gold-standard measures of children's pubertal development, rated by trained health-care professionals, are available in the SECCYD dataset, thereby allowing for a more precise demarcation of childhood- and adolescent internalizing symptomatology (Susman et al. 2010).

Drawing in part on both the aforementioned work with major depressive disorder and depressive symptomatology (e.g., Harrington et al. 1996, 1997; Silberg et al. 1999) as well as the Duggal et al. (2001) study, we selected key early family experience variables from the SECCYD dataset including repeated measurements of observed maternal sensitivity, family income-to-needs ratio, father absence, the frequency of negative life events, and the perceived quality of the marital relationship by primary caregivers. Also consistent with Duggal et al. (2001), we used maternal reports of depression as a proxy variable reflecting, in part, genetic/familial loading for depressive symptomatology. Finally, we selected peer variables in the SECCYD (mother and teacher reports of peer victimization and child-reported friendship quality) that have been repeatedly identified in the literature as correlates of both depressive and internalizing symptomatology.

Based on the logic of the prior empirical work reviewed above (e.g., Harrington et al. 1997; Silberg et al. 1999) suggesting that there may be differences in the etiology of depressive symptomatology over development, we hypothesized that family environment influences would be associated more strongly with internalizing symptomatology occurring prior to pubertal onset in childhood whereas maternal depression, reflecting a genetic liability to internalizing symptomatology, would be associated more strongly with internalizing symptomatology occurring following pubertal onset in adolescence. Additionally, we were also interested in evaluating whether peer psychosocial influences would exert a unique influence on internalizing symptoms in childhood and adolescence after accounting for the effects of family experience factors and maternal depression. Based on the literature discussed above chronicling the reliable association between peer victimization and internalizing symptoms, we anticipated that peer victimization would be associated with higher levels of internalizing symptomatology, and in particular internalizing symptomatology occurring during adolescence. Moreover, we anticipated this association in adolescence *even after accounting for effects of family experiences and maternal depression*. Similarly, based on theoretical work articulating the importance of positive peer relationships in children and adolescent's development, we anticipated that friendship quality would be negatively associated with elevated internalizing symptomatology. Once again, because of the increasingly salient role that peer relationships play as children develop into adolescence, we anticipated this promotive effect to be more pronounced for internalizing symptomatology occurring during adolescence. That said, in some contrast to peer victimization, we were less certain how robust its association with internalizing symptoms might be in the context of our other predictors. In evaluating these questions, we address a limitation of prior research by controlling for the stability of internalizing symptomatology across childhood and adolescence.

Methods

Participants

Families were recruited for the NICHD SECCYD in 1991 from hospitals located in or near Little Rock, AR; Orange County, CA; Lawrence, KS; Boston, MA; Pittsburgh, PA; Philadelphia, PA; Charlottesville, VA; Seattle, WA; Morganton, NC; and Madison, WI. During selected 24-h sampling periods, 8986 women who gave birth were screened, 5416 of whom met the eligibility criteria for the study. Families were excluded if: (a) the mother was younger than 18 years of age, (b) the family planned to move, (c) there was a multiple birth, (d) the infant had a known disability or remained in the hospital more than 7 days, (e) the mother acknowledged substance abuse, (f) the mother did not speak English, (g) the mother lived more than an hour from the laboratory site or in an extremely unsafe neighborhood, as determined by local police. From that group, 1364 families became study participants upon completing a home interview when their infants were one month old. Additional details about recruitment and selection procedures are available in prior publications from the study (see NICHD Early Child Care Research Network [ECCRN 2005]) and from the study web site (https://www.nichd.nih.gov/research/supported/Pages/seccyd.aspx). Data were collected longitudinally on the SECCYD sample through age 15 years; and follow-up studies led by researchers at the University of California Irvine and the University of Washington provided age-18 data (see Booth-LaForce and Roisman 2014). Specifically, for the age-18 follow-up interviews, SECCYD youth assessment data was collected at the University of California Irvine and both youth and parent assessment data were collected at the University of Washington. Accordingly, in the current study, youth self-report internalizing data were pooled. Note that, while large, demographically diverse, and methodologically rich, the NICHD SECCYD was not designed to be a nationally representative study.

Analytic Sample

The analytic sample for the current report consists of 959 children who participated in any of the study's repeated physical assessments of pubertal status and for whom a categorical measure of the timing of pubertal onset could be estimated (see below for a detailed description of this variable). We conducted attrition analyses examining the

full sample and analytic sample on child sex, single-parent status in early childhood, total income after the birth of the child, and child race/ethnicity. The full and analytic sample did not differ on any of these demographic variables. In addition, the full and analytic samples did not differ on our index of the highest level of internalizing symptomatology (see below) across time points with the exception of our Kindergarten [$t(1073) = -2.02$, $p < 0.05$)], Grade 3 [($t(1079) = -3.55$, $p < 0.01$)], and Grade 6 [$t(1038) = -2.45$, $p < 0.05$)] measures. Note that effect sizes for these differences were all small by Cohen's standards (ds 0.17, 0.32, and 0.25, respectively). Lastly, the full and analytic samples did not differ on any of our composite substantive covariates of interest with the exception of negative life events [$t(1152) = -1.97$, $p < 0.05$] and teacher reports of peer victimization [$t(215.84) = -4.03$, $p < 0.01$]. Follow-up analyses indicated these differences were due to our Grade 3 measure of negative life events [$t(1026) = -2.14$, $p < 0.05$] and Grade 3 [$t(208.29) = -4.21$, $p < 0.01$] and Grade 6 [$t(119.87) = -2.38$, $p < 0.05$] measures of teacher-reported peer victimization. Similar to above, effect sizes for these differences were all generally small in magnitude (ds 0.21, 0.41, and 0.28 respectively). Note that for all differences, participants in the analytic sample demonstrated *higher* levels of internalizing symptomatology, negative life events, and teacher-reported peer victimization than those who were not. As these were the only observed differences between youth with pubertal timing data and those without, we assumed data were missing at random.

Measures

Measures are presented in four sets corresponding to their function and order of entry in the analyses discussed below: Variables used to create separate composite (dependent) measures of (1) child and adolescent internalizing symptomatology and variables used to composite measures of (2) maternal depression, (3) family experience, and (4) peer experience. In all cases we selected variables that were measured multiple times by multiple informants using standard assessment tools to maximize validity and reliability in our measurement.

Child and Adolescent Internalizing Symptomatology

Participant internalizing symptomatology from childhood to late adolescence was assessed using the internalizing scale of the Child Behavior Checklist obtained using the parent (CBCL) and teacher-report (TRF) versions (Achenbach 1991a; Achenbach and Edelbrock 1986; Achenbach et al. 1987). Participant self-reported internalizing symptomatology also was assessed in adolescence (ages 15 and 18 years) using the Youth Self Report (YSR) version of the Child Behavior Checklist (Achenbach 1991b). Following Duggal et al. (2001), for cases in which data were obtained concurrently from two different informants (e.g., mother and teacher), the highest internalizing symptomatology rating for a given informant at that assessment point was used as an index of the most severe level of internalizing symptomatology experienced. Because there are some differences in item content between the CBCL forms/versions for 2–3 year olds and that for 4–18 year olds, we used standardized *T* scores, which were averaged over time yielding mean internalizing symptomatology composites for the time periods prior to and after pubertal onset (see below for a description of demarcation of pubertal timing estimates) for each individual. Maternal reports on the CBCL were used from the following time points: 24, 36, and 54 months, Kindergarten, Grades 1, 3, 4, 5, and 6; and ages 15 and 18 years. Teacher reports were used from the following assessment points: Kindergarten and Grades 1, 2, 3, 4, 5, and 6. Youth self-reports from the age 15 and 18 assessment points were also used. The internalizing symptomatology scale demonstrated adequate reliability across time and had a standardized coefficient α averaging 0.84 for maternal reports, 0.86 for teacher reports, and 0.90 for youth self-reports across all assessments. Note that we also created average composite measures of childhood internalizing symptomatology within informant to use in sensitivity analyses (discussed below).

Maternal Depressive Symptomatology

Self-reported maternal depressive symptom average composites were created using every assessment point at which the Center for Epidemiologic Studies Depression Scale (CES-D; Radloff 1977) was acquired in the SECCYD: 1, 6, 15, 24, 36, 54 months; Grades 1, 3, 5, and 6; and ages 15 and 18 years. Although the CES-D was developed initially to assess the severity of depressive symptoms, it is now often used to estimate the prevalence of depression or screen for depressive symptomatology across many populations and settings (Santor and Kazdin 2000). The CES-D demonstrated adequate reliability across time with a standardized coefficient α averaging 0.90.

Family Experience Indicators

Maternal Sensitivity

Maternal sensitivity was assessed in the context of mother-child interactions that were videotaped during 15-min semi-structured situations at 6, 15, 24, 36 and 54 months; Grades 1, 3, and 5; and age 15. At each assessment point, the children were videotaped while engaging in tasks at the zone of proximal development while primary caregivers

provided assistance at the younger ages; at older ages (Grade 3 and older), joint tasks, including discussion tasks, were used. Tasks were designed to be developmentally appropriate. Psychometric properties for composite measures of observed maternal sensitivity at each assessment point were adequate (internal consistencies of the sensitivity composite measures for mothers averaged 0.79 [range 0.70–0.85] across assessments; for detailed information on the SECCYD sensitivity assessments see Belsky et al. 2007b; Haltigan et al. 2013; NICHD ECCRN 2001, 2004, 2008). Maternal sensitivity scores were standardized and averaged to create composite measures of observed maternal sensitivity.

Family Income-to-Needs Ratio

Family financial resources were operationalized in terms of an income-to-needs ratio, computed separately for every assessment point at which relevant data were acquired (1, 6, 15, 24, 36, 54 months; Grades 1, 3, 4, 5, 6; age 15). The income-to-needs ratio at each assessment point was calculated from US Census Bureau tables as the ratio of family income to the poverty threshold for each household size at that time point. Higher scores on this composite reflect greater income-to-needs. Scores were averaged across assessment points to create composite measures of family financial resources.

Father Absence

Primary caregivers indicated whether the study child's father was living in the home at each assessment wave through age 15. The scoring was reversed (0 = father in home, 1 = father not in home) and average composite measures of father absence (i.e., the father was *not* living in the household) were computed from 1 month of age to 15 years (1, 3, 6, 9, 12, 15, 24, 36, 42, 46, 50, 54, 60, and 66 months; Kindergarten-Fall [F], Kindergarten-Spring [S]; Grades 1F, 1S, 2F, 2S, 3, 4, 5, 6, 7; ages 14 and 15).

Negative Life Events

Mothers completed the Life Experiences Survey (LES; Sarason et al. 1978) at 54 months, Grades 3 and 5, and age 18. This 57-item questionnaire asks mothers to identify from a list those life events that have happened to them over the past year, and to rate, on a 7-point scale (from +3 = *very positive* to 0 = *neutral*, to –3 = *very negative*) the impact the event has had on their lives. Events include routine happenings (e.g., "child started school") to major events (e.g., "major change in financial status") to catastrophic events (e.g., "death of a parent"). This measure provides an overview of the stressful events that have befallen the child's family and may have an impact on the child's well-being, as well as on the quality of parenting. Composite (average) measures of negative life events were created from the assessment points noted above.

Marital Quality

Composite measures of marital quality were created using the 6-item intimacy subscale of the Personal Assessment of Intimacy in Relationships Inventory (Schaefer and Olson 1981) which was completed at the following assessment points: 1, 36, and 54 months; Grades 1, 3, 5, 6; and at the age 15 and 18-year assessment points. Samples items on this measure include *My partner listens when I need to talk* and *My partner understands me*. Subscale scores were computed as an average of the six item responses. Reliability was adequate across time with an average standardized coefficient $\alpha = 0.87$). Scores were standardized and averaged across assessment points with higher scores reflecting higher levels of emotional intimacy in the marital relationship.

Peer Psychosocial Indicators

Peer Victimization: Mother and Teacher Report

At Grades 3, 4, 5, and 6, mothers and teachers were asked to complete a questionnaire designed to measure the study child's peer-related behaviors. This questionnaire consisted of 43 items, which were adapted from the Child Behavior Scale (Ladd and Profilet 1996), the Peer Victimization Scale (Kochenderfer and Ladd 1996) and the Relational Aggression scale (Crick et al. 1996). Respondents were asked to rate the child's behavior with peers on a 3-point scale (0 = *Not True*, 1 = *Sometimes True*, 2 = *Often True*). For purposes of the current project, the peer victimization subscale (7 items) from this measure was used. Peer victimization scores were computed at each time point as the average of these 7 items. Both mother and teacher report measures of peer victimization demonstrated adequate reliability across time with a standardized coefficient α averaging 0.90 for maternal reports and 0.89 for teacher reports. Scores at each time point were then averaged across assessment points within informant to create separate composite measures of mother and teacher-reported peer victimization. Higher scores reflected higher levels of mother- and teacher-reported peer victimization.

Friendship Quality

Study children completed a modified version of the Friendship Quality Questionnaire (Parker and Asher 1993) designed to assess their perceptions of their friendship with

their very best friend at Grades 3, 4, 5, 6, age 15, and age 18 years. Children rated how true 20 statements (28 statements at grade 6, and ages 15 and 18) were of their relationship with their best friend on a 5-point scale, from 1 = *Not at all true* to 5 = *Really true*. These statements are grouped into six subscales including companionship and recreation, validating and caring, help and guidance, intimate disclosure, conflict and betrayal, and conflict resolution. In the current report, a friendship quality total score at each time point was computed as a weighted average of the items. Child reports of friendship quality demonstrated adequate reliability across time with a standardized coefficient α averaging 0.90. Friendship quality total scores at each assessment were standardized and averaged across assessment points to create friendship quality composites, with higher scores reflecting higher levels of friendship quality.

Pubertal Development

Starting at age 9½ years, all SECCYD children were asked to participate in an annual health and physical development assessment. A primary component of the assessment was a physical examination of the child. Pubertal status was assessed using Tanner staging. Tanner staging for girls was based on instructions from the American Academy of Pediatrics Manual, Assessment of Sexual Maturity Stages in Girls (Herman Giddens and Bourdony 1995), augmented with breast bud palpation. For boys, Tanner staging was based on Tanner's original criteria (adapted from Tanner 1962; Marshall and Tanner 1970). The majority of the exams were conducted by nurse practitioners; however, some were administered by pediatric endocrinologists, depending upon staff employed at each data collection site. All clinicians were experienced with Tanner staging of children in the evaluated age groups. Additional details regarding the measurement of pubertal development in the SECCYD can be found in Belsky et al. (2007a).

Results

Analytic Plan

Prior to conducting focal analyses, we first determined each individual's estimated onset of puberty using latent transition analysis applied to the Tanner staging assessments. Each individual's timing of pubertal onset was then used to create separate composite measures of internalizing symptomatology which began prior to and following pubertal onset. Next, zero-order intercorrelations among study variables were computed. We also tested whether the magnitude of family experience (i.e., maternal sensitivity, father absence, family income-to-needs, negative life events, and marital quality) and maternal depression associations with prepubertal vs. postpubertal internalizing symptomatology were significantly different.

Our primary substantive analyses consisted of two hierarchical stepwise regression models examining the predictive significance of family experience, maternal depression, and peer experience variables for child and adolescent internalizing symptomatology. In the first model, internalizing symptomatology in childhood was predicted from *childhood* measures of family experience, maternal depression, and peer experience predictor variables (Model 1). In a second model, *adolescent* internalizing symptomatology was predicted from *across-time* (i.e., childhood and adolescent) composites of family experience, maternal depression, and peer experience predictor variables (Model 2). Note that for Model 2, the use of across time predictor composites was designed to maximize the reliability and precision of predictor variable sets by leveraging all of the available data in the SECCYD in predicting adolescent internalizing symptomatology.

For each of the regression models, the order of entry of each of the three blocks of predictor variable sets was theoretically guided by the original logic of the aforementioned empirical work with depressive problems. Specifically, because prior research (e.g., Harrington et al. 1996, 1997; Murray and Sines 1996; Thapar and McGuffin 1996) suggests that family experience variables should be more strongly associated with internalizing symptomatology occurring prior to pubertal onset (i.e., in childhood) these variables were entered in the first block of Model 1, followed by maternal depression, and finally peer psychosocial influences. Similarly, because prior work (e.g., Duggal et al. 2001) suggests that maternal depression should be more strongly associated with internalizing symptomatology occurring following pubertal onset (i.e., in adolescence), this variable was entered in the first block of Model 2, followed by family experience, and finally peer psychosocial influences. In each model, we controlled for the stability of internalizing symptomatology in a final block. As such, we were able to ascertain the robustness of focal predictors for internalizing symptomatology in childhood and adolescence in relation to the addition of other predictors in each model.[1]

Finally, we conducted a series of sensitivity analyses to determine whether: (1) our substantive results from Models

[1] In light of the well documented finding that the initiation of puberty is associated with a rise in depressive and internalizing symptomatology among girls relative to boys (Nolen-Hoeksema, 2001; Zahn-Waxler et al., 2000) we also conducted interaction analyses to examine whether child sex moderated any of the focal associations between our predictor sets composited across time and internalizing symptomatology occurring in childhood and adolescence. None of the interaction effects were significant.

Table 1 *N*s and percentages of children starting puberty by age in the SECCYD

	Girls ($N = 476$)		Boys ($N = 483$)	
Age of pubertal onset	*N*	%	*N*	%
≤9.5	103	21.6	33	6.8
9.5–10.5	182	38.2	56	11.6
10.5–11.5	124	26.1	208	43.1
11.5–12.5	63	13.2	120	24.8
12.5–13.5	4	0.8	55	11.4
>13.5	–	–	11	2.3

1 and 2 differed as a function of whether internalizing symptomatology was operationalized by informant (i.e., mother, teacher, or youth self-report [adolescence only]) relative to our index of the highest level of internalizing symptomatology; and (2) substantive results for internalizing symptomatology in adolescence using *across time* composites of predictor variable sets differed when predictor sets were operationalized based on developmental timing (i.e., childhood predictor composites and adolescent predictor composites). Because few effects from these models were substantively different than our core models presented above, they are summarized in the electronic supplement to this manuscript.

Estimation of Pubertal Timing

As reported in previous work using this same dataset (Belsky et al. 2007a), a categorical version of the timing of pubertal onset was estimated using latent transition analysis (LTA; Collins and Flaherty 2002; Muthén and Muthén 1998–2006). Separate models were conducted for boys and girls. The data modeled indicated, at each age of measurement, whether the child exhibited any evidence of pubertal development (i.e., yes/no) on (1) physical exam of genitals (for boys) or breast (for girls) development and (2) physical exam of pubic hair development. LTA analyses were conducted using *M*Plus (version 4.1: Muthén and Muthén 1998–2006), which uses maximum likelihood (ML) estimation under the assumption of data missing at random (MAR). The weighting given to any particular indicator was equal across time points (i.e., measurement invariance) and children, once categorized as having initiated puberty, could not revert to a no-initiation state. That is, the LTA models constrained the weights of the measures listed above to be equal at each age and constrained children who had "started puberty" at one age to remain "in puberty" at later ages. Results from this analysis are presented in Table 1.

Table 2 Intercorrelations among childhood composites of family experience, peer, and maternal depression predictor variables and achenbach system measures of childhood and adolescent internalizing symptomatology

	1	2	3	4	5	6	7	8	9	10	11
1. Childhood internalizing	–										
2. Adolescent internalizing	0.48*	–									
3. Maternal sensitivity childhood	−0.28**	−0.10**	–								
4. Income-to-needs childhood	−0.21**	−0.11**	0.44**	–							
5. Father not in home childhood	0.20**	0.12**	−0.44**	−0.38**	–						
6. Negative life events childhood	0.15**	0.22**	0.09**	−0.07*	0.04	–					
7. Emotional intimacy childhood	−0.27**	−0.23**	0.14**	0.19**	−0.15**	−0.25**	–				
8. Peer victimization MR childhood	0.42**	0.33**	−0.29**	−0.18**	0.18**	0.16**	−0.21**	–			
9. Peer victimization TR childhood	0.30**	0.13**	−0.29**	−0.18**	0.19**	0.06	−0.06	0.49**	–		
10. Friendship quality score childhood	−0.09**	−0.10**	−0.03	−0.05	−0.03	−0.03	0.02	−0.09**	−0.08*	–	
11. Maternal depression childhood	0.45**	0.31**	−0.37**	−0.33**	0.33**	0.30**	−0.49**	0.31**	0.20**	0.02	–

Note All predictors are average childhood composites of the target variable. The maternal sensitivity and emotional intimacy composites are the average of the standardized scores for each assessment in childhood. For internalizing symptomatology in childhood and adolescence, composites are average *T* scores using the highest informant at each available time point

MR mother report, *TR* teacher report. *N*s range 902–959

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

Table 3 Sample descriptive statistics and intercorrelations among across time composites of family experience, peer, and maternal depression predictor variables and achenbach system measures of childhood and adolescent internalizing symptomatology

	1	2	3	4	5	6	7	8	9	10	11
1. Childhood internalizing	–										
2. Adolescent internalizing	0.48**	–									
3. Maternal sensitivity 6MO—age 15	−0.29**	−0.11**	–								
4. Income-to-needs 1MO—age 15	−0.22**	−0.11**	0.44**	–							
5. Father not in home 1MO—age 15	0.20**	0.13**	−0.43**	−0.37**	–						
6. Negative life events 54MO—age 18	0.17**	0.25**	0.09**	−0.06	0.04	–					
7. Emotional intimacy 1MO—X18	−0.25**	−0.25**	0.14**	0.18**	−0.13**	−0.28**	–				
8. Peer victimization MR G3–G6	0.44**	0.39**	−0.28**	−0.18**	0.20**	0.17**	−0.21**	–			
9. Peer victimization TR G3–G6	0.27**	0.19**	−0.32**	−0.20**	0.20**	0.05	−0.05	0.47**	–		
10. Friendship quality score G3—age 18	−0.13**	−0.16**	0.05	−0.01	−0.07*	−.02	0.04	−0.15**	−0.14**	–	
11. Maternal depression 1MO—age 18	0.46**	0.34**	−0.36**	−0.33**	0.32**	0.34**	−0.48**	0.32**	0.18**	−0.02	–
Mean	51.44	52.41	−0.02	3.85	0.25	3.37	−0.06	0.30	0.17	−0.01	9.61
Standard deviation	5.92	7.42	0.68	2.89	0.36	2.51	0.76	1.61	0.24	0.71	6.09
N	948	923	959	958	959	955	949	955	936	954	959

Note All predictors are average composites of the target variable. The maternal sensitivity and emotional intimacy composites are the average of the standardized scores for each assessment. For internalizing symptomatology in childhood and adolescence, composites are average *T* scores using the highest informant at each available time point

MR mother report, *TR* teacher report. *Ns* range 902–959

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

Intercorrelations among Study Variables

Zero-order correlations among prepubertal (*childhood*) composites of family experience, maternal depression, and peer experience predictor variables are reported in Table 2 and zero-order correlations of *across time* composites of family experience, maternal depression, and peer experience predictor variables and child and adolescent internalizing symptomatology, as well as summary descriptive statistics for study variables, are presented in Table 3. As is reflected in both correlation tables, there was moderate stability between internalizing symptoms in childhood and adolescence. Using equivalent transformations of Cohen's (1992) *d* effect size criteria to interpret *r* (small effect = 0.10, medium effect = 0.24, large effect = 0.37), intercorrelations among predictor variables, whether composited only in childhood or across time, were mostly small to medium in magnitude. Exceptions to this general pattern were correlations of larger magnitude between maternal sensitivity and income-to-needs, maternal sensitivity and father absence, income-to-needs and father absence, and mother and teacher reports of peer victimization. In addition, maternal depression was moderately correlated with each of the other predictors except teacher reports of peer victimization and child-reported friendship quality.

Given their focal role in prior work examining differential correlates of child and adolescent major depressive disorder, using the *Psych* package (Revelle 2015) in the R environment for statistical computing (R Core Team 2016), we tested whether the magnitude of the associations of family experience variables and maternal depression with internalizing symptomatology occurring in childhood and adolescence differed significantly using methods recommended by Steiger (1980) for dependent, overlapping correlations (Case A, see Steiger 1980). Paralleling our analytic approach described previously for core regression analyses (see below), in these preliminary analyses we leveraged the rich longitudinal data in the SECCYD to maximize the precision of our covariates by using the *across-time* composites of family experience and maternal depression variables. Note that the methods recommended by Steiger (1980) require a single sample size for computation. As such, listwise deletion was implemented when producing a correlation matrix among family experience variables, maternal depression, and internalizing symptomatology composites ($n = 911$). Of the family experience variables, maternal sensitivity ($r = -0.29$ vs. $r = -0.11$), family income-to-needs ($r = -0.22$ vs. $r = -0.11$), and father absence ($r = 0.21$ vs. $r = 0.12$) were all significantly ($p < 0.01$) more strongly associated with internalizing symptomatology occurring in childhood compared to adolescence. In contrast, the frequency of negative life experiences was more strongly associated with internalizing symptomatology in adolescence relative to

Table 4 Hierarchical regression analysis predicting internalizing symptomatology in childhood from childhood family experience, maternal depression, and peer psychosocial variables (model 1)

Step	Independent variables	R^2 change	*B* (SE)	β	Overall		
					R^2	*F*	df
1.	Maternal sensitivity	0.16**	−2.01 (0.30)	−0.25**	0.16	31.82	5, 848
	Income-to-needs		−0.06 (0.08)	−0.03			
	Father not at home		0.60 (0.59)	0.04			
	Negative life events		0.21 (0.07)	0.10**			
	Emotional intimacy		−1.49 (0.24)	−0.21**			
2.	Maternal sensitivity	0.07**	−1.29 (0.30)	−0.16**	0.23	41.94	6, 847
	Income-to-needs		0.01 (0.07)	0.00			
	Father not at home		−0.08 (0.57)	−0.01			
	Negative life events		0.05 (0.07)	0.02			
	Emotional intimacy		−0.59 (0.25)	−0.08*			
	Maternal depression		0.32 (0.04)	0.35**			
3.	Maternal sensitivity	0.08**	−0.67 (0.29)	−0.08*	0.31	42.80	9, 844
	Income-to-needs		0.01 (0.07)	0.00			
	Father not at home		−0.19 (0.55)	−0.01			
	Negative life events		−0.03 (0.07)	−0.01			
	Emotional intimacy		−0.42 (0.24)	−0.06			
	Maternal depression		0.29 (0.04)	0.31**			
	Peer victimization mother report		4.56 (0.63)	0.25**			
	Peer victimization teacher report		1.88 (0.74)	0.08*			
	Friendship quality		−0.42 (0.20)	−0.06*			
4.	Maternal sensitivity	0.08**	−0.79 (0.28)	−0.10**	0.39	54.69	10, 843
	Income-to-needs		−0.01 (0.07)	−0.00			
	Father not at home		−0.23 (0.51)	−0.01			
	Negative life events		−0.09 (0.06)	−0.04			
	Emotional intimacy		−0.27 (0.23)	−0.04			
	Maternal depression		0.23 (0.03)	0.25**			
	Peer victimization mother report		3.04 (0.61)	0.17**			
	Peer victimization teacher report		2.21 (0.70)	0.10**			
	Friendship quality		−0.26 (0.19)	−0.04			
	Adolescent internalizing		0.24 (0.02)	0.31**			

Note $N = 854$

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

childhood internalizing symptomatology ($r = 0.25$ vs. $r = 0.16$, $p < 0.01$). Importantly, maternal depression was also significantly more strongly associated with internalizing symptomatology in childhood relative to internalizing symptomatology in adolescence ($r = 0.45$ vs. $r = 0.34$, $p < 0.01$).[2]

[2] Steiger (1980) recommended tests of separate, non overlapping (Case B; see Steiger 1980) dependent correlations between covariates composited within childhood and childhood internalizing symptomatology versus correlations of covariates composited within adolescence and adolescent internalizing symptomatology were not materially different than tests of dependent correlations noted above with the exception of associations between father absence and internalizing symptomatology ($r = 0.20$ [childhood] vs. $r = 0.13$ [adolescence], $p = 0.14$).

Unique Predictors of Childhood Internalizing Symptomatology

Results of the hierarchical multiple regression analysis predicting childhood internalizing symptomatology from childhood family experience, maternal depression, and peer experience variables (Model 1) are presented in Table 4. Maternal sensitivity, negative life events, and emotional intimacy in the marriage were all significant predictors of childhood internalizing symptomatology at entry in the first block, accounting for 16 % of the variance. Higher levels of maternal sensitivity and emotional intimacy in the marital relationship predicted lower levels, and negative life events higher levels, of internalizing symptomatology occurring in

Table 5 Hierarchical regression analysis predicting internalizing symptomatology in adolescence from across time maternal depression, family experience, and peer psychosocial variables (model 2)

Step	Independent variables	R^2 change	*B* (SE)	β	Overall		
					R^2	*F*	*df*
1.	Maternal depression	0.12**	0.43 (0.04)	0.35**	0.12	120.43	1, 886
2.	Maternal depression	0.03**	0.30 (0.05)	0.25**	0.15	25.12	6, 881
	Maternal sensitivity		−0.17 (0.42)	−0.02			
	Income-to-needs		0.04 (0.09)	0.02			
	Father not at home		0.38 (0.74)	0.02			
	Negative life events		0.40 (0.10)	0.14**			
	Emotional intimacy		−0.96 (0.35)	−0.10**			
3.	Maternal depression	0.09**	0.25 (0.05)	0.20**	0.24	30.11	9, 878
	Maternal sensitivity		0.57 (0.41)	0.05			
	Income-to-needs		0.03 (0.09)	0.01			
	Father not at home		−0.00 (0.71)	0.00			
	Negative life events		0.29 (0.10)	0.10**			
	Emotional intimacy		−0.72 (0.33)	−0.07*			
	Peer victimization mother report		6.90(0.88)	0.28**			
	Peer victimization teacher report		0.78 (1.07)	0.03			
	Friendship quality		−1.10 (0.32)	−0.10**			
4.	Maternal depression	0.07**	0.13 (0.05)	0.11**	0.31	39.30	10, 877
	Maternal sensitivity		0.87 (0.39)	0.08*			
	Income-to-needs		0.04 (0.08)	0.02			
	Father not at home		0.03 (0.67)	0.00			
	Negative life events		0.29 (0.09)	0.10**			
	Emotional intimacy		−0.64 (0.32)	−0.07*			
	Peer victimization mother report		4.53 (0.87)	0.18**			
	Peer victimization teacher report		0.44 (1.02)	0.01			
	Friendship quality		−0.90 (0.30)	−0.08**			
	Childhood internalizing		0.42 (0.04)	0.33**			

Note $N = 888$

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

childhood. When maternal depression was entered in the 2nd block, only maternal sensitivity remained a significant predictor. Higher levels of maternal depression significantly predicted higher levels of childhood internalizing symptomatology and accounted for an additional 7 % of the variance. When peer psychosocial influences were added on the third block, both maternal sensitivity and maternal depression remained significant predictors of childhood internalizing symptomatology, along with mother and teacher-reports of peer victimization (which predicted higher) and child-reported friendship quality (which predicted lower) levels of childhood internalizing symptomatology. Together, the peer psychosocial variables accounted for an additional 8 % of the variance. Lastly, when we controlled for internalizing symptomatology in adolescence in the 4th block, maternal sensitivity, maternal depression, and mother and teacher-reports of peer victimization remained significant predictors of childhood internalizing symptomatology. Not surprisingly, internalizing symptomatology in adolescence was significantly and modestly associated with internalizing symptomatology in childhood. Altogether, the final model accounted for 39 % of the variance in childhood internalizing symptomatology (31 % prior to the inclusion of adolescent internalizing symptomatology).

Unique Predictors of Internalizing Symptomatology in Adolescence

Results of the hierarchical multiple regression analysis predicting internalizing symptomatology in adolescence from *across time* family experience, maternal depression, and peer experience variables (Model 2) are reported in Table 5. On the first step, higher levels of maternal depression predicted higher levels of adolescent internalizing symptomatology, accounting for 12 % of the variance.

When family experience variables were added on the 2nd step, only negative life events and emotional intimacy in the marital relationship were significant predictors. Experiencing a greater number of negative life events predicted higher levels of internalizing symptomatology in adolescence whereas greater emotional intimacy in the marital relationship predicted lower levels of internalizing symptomatology in adolescence. Maternal depression remained significant in the 2nd step and the combined variables accounted for an additional 3 % of the variance. Mother reports of peer victimization and the child-reported friendship quality were both significant at entry on the 3rd step. Higher levels of mother-reported peer victimization predicted elevated levels of internalizing symptomatology in adolescence whereas higher friendship quality predicted lower levels of internalizing symptomatology in adolescence. Negative life events and emotional intimacy in the marital relationship remained significant predictors, as did maternal depression. Taken together, the peer psychosocial predictors accounted for an additional 9 % of the variance in internalizing symptomatology occurring in adolescence in the third step. Finally, when we controlled for childhood internalizing symptomatology in the 4th step, all of the family experience and peer psychosocial variables that were significant at step 3 remained significant. In addition, a small counterintuitive effect emerged with higher levels of maternal sensitivity predicting elevated levels of internalizing symptomatology in adolescence. As was the case in our analysis predicting internalizing symptomatology in childhood, there was a moderate and significant positive association between internalizing symptomatology occurring in adolescence with that occurring in childhood. The final model accounted for 31 % of the variance in internalizing symptomatology occurring in adolescence. (24 % prior to the inclusion of childhood internalizing symptomatology).

Discussion

In light of the well-established rise in internalizing symptomatology during adolescence, and inspired in part by work suggesting that major depressive disorders that begin in childhood and adolescence may represent unique developmental phenomena (e.g., Harrington et al. 1996, 1997; Silberg et al. 1999), the objective of the present inquiry was to provide the first large-sample examination of unique and overlapping correlates of internalizing symptomatology occurring in childhood and adolescence. Using multi-method, multi-informant data from the SECCYD, we tested whether family experiences (i.e., maternal sensitivity, family income-to-needs, presence of father in the home, quality of the marital relationship) were associated more strongly with childhood internalizing symptomatology (compared to internalizing symptomatology in adolescence) and whether maternal depression was associated more strongly with internalizing symptomatology in adolescence (compared to internalizing symptomatology occurring in childhood). Altogether, our comprehensive set of results demonstrated that both family influences and maternal depression tracked more strongly with internalizing symptomatology in childhood while the influence of peer relationship variables on internalizing symptomatology occurring in childhood and adolescence was relatively nonspecific. Thus, we did not find support for distinctive developmental correlates of childhood and adolescent internalizing symptomatology.

Despite finding that maternal sensitivity, family income-to-needs, and father absence more strongly tracked with childhood internalizing symptomatology (relative to that occurring in adolescence), the degree to which these variables alone accounted for variation in childhood internalizing symptomatology was modest. Moreover, zero-order associations between maternal depression—whether aggregated across time or within childhood and adolescence—and childhood and adolescent internalizing symptomatology suggested that maternal depression tracked more strongly with internalizing symptomatology occurring in childhood—a finding not in keeping with prior work on major depressive disorder suggesting that familial loading for depression is associated with pubertal rather than prepubertal depression (Harrington et al. 1997; Silberg et al. 1999). Additionally, unlike the results of Duggal et al. (2001), we found that maternal depression continued to remain a significant predictor of the most severe level of internalizing problems occurring in childhood even when family experience variables were taken into account.

A novel aspect and strength of the current study was the inclusion of *peer* relationship variables in predicting internalizing symptomatology occurring in childhood and adolescence while also controlling for stability in internalizing symptomatology itself. Inclusion of peer relationship variables along with family influences and maternal depression in predictive models of internalizing symptomatology is seldom seen in the literature and allows for a more comprehensive understanding of the relative predictive significance of these factors. The current findings demonstrate that peer psychosocial influences, especially maternal-reports of peer victimization, represent unique predictors of internalizing symptomatology, even after controlling for family influences and maternal depression (as well as stability in internalizing symptomatology itself).

Given the present results, it remains ambiguous as to whether a familial loading for internalizing symptomatology has any unique predictive significance for its timing of occurrence (i.e., prior to vs. following pubertal onset) or is

simply a more general marker of vulnerability that is distributed over childhood *and* adolescence. Future longitudinal work, including research informed by behavior-genetic designs (Thapar and McGuffin 1996), is needed to answer this question more definitively. Another possibility is that biological and/or genetic loading for depression could be associated with a distinct subclass of childhood-onset internalizing symptomatology. Additional person-centered methodological work with internalizing symptomatology (e.g., Sterba et al. 2007) could provide insight into this possibility.

There are limitations to the current study that delimit the reach of our findings. As has been noted in other reports using the SECCYD dataset, although it is a large national study, its sample is not nationally representative and is a normative-risk cohort. It remains possible that stronger associations between family experience variables, maternal depression, and internalizing symptomatology occurring in childhood and adolescence would have been uncovered had a high-risk cohort been studied in which clinically elevated levels of internalizing symptomatology might have been more commonly observed. A second limitation concerns the maternal depression variable that served as a proxy for genetic-loading (i.e., heritability) in the context of prior work examining differential correlates of childhood and adolescence-onset depression. We acknowledge that maternal depression reflects an imprecise marker of genetic loading for depression in offspring. Third, shared method variance between mother and teacher reports of peer victimization experiences and these same informants' reports of child internalizing symptomatology might partially explain the robust associations observed between maternal and teacher reports of peer victimization and internalizing symptomatology both in childhood and adolescence. Fourth, we exclusively relied on mother and teacher-reports of peer victimization. Other measurement approaches used to operationalize peer victimization, such as peer nomination procedures, are well-validated and their inclusion would have broadened our assessment battery for this construct. Fifth, it should be noted that the current study focused on internalizing symptomatology and it is conceivable that the current results may have been different if we had considered individual subscales (e.g., anxiety/depression) that comprise the broad-band internalizing dimension. That said, we believe this possibility is unlikely given the typically large associations observed between individual subscales of the internalizing dimension and the internalizing dimension itself.[3] Finally, the focus of the current study was on mean levels of internalizing symptomatology within childhood and adolescence as demarcated by pubertal status. The current analyses therefore do not address questions concerning growth in internalizing symptomatology across time nor were intended to directly address questions concerning 'onset' of clinically significant levels of problematic internalizing symptomatology.

[3] In the current study, we observed correlations in excess of 0.80 between the anxious/depressed and internalizing raw scale scores for teacher-reports and in excess of 0.90 for parent-reports. The correlation between these scales for youth self-reports at age 15 was 0.93 and at age 18 it was 0.92 (all *ps* < 0.01).

Conclusion

The current study contributes to the literature on internalizing symptomatology in at least two novel ways. First, it is the first large-scale prospective, multi-domain investigation to raise and address the possibility that there may be distinct developmental correlates of childhood and adolescent internalizing symptomatology. Using a variety of predictors from conceptually distinct domains (e.g., family, peer) and operationalized in multiple ways (e.g., observational, parent, teacher, and self-report), we were able to explain a good deal of the total variation in internalizing symptomatology occurring both in childhood and adolescence. In particular, we found relatively robust effects for peer victimization on internalizing symptomatology in both childhood and adolescence. Second, the current study provides a basic substantive contribution by explicitly testing a model of differential prediction of child and adolescent internalizing symptomatology that is based on earlier work with major depressive disorder (e.g., Harrington et al. 1997). We found little evidence indicating that specific conceptual subsets of predictors were uniquely associated with internalizing symptomatology in childhood or adolescence, as might have been expected based on this earlier work and subsequent work inspired by it (e.g., Duggal et al. 2001). This is not to suggest that that identifiable and meaningful patterns of distinct internalizing symptomatology in childhood and adolescence do not exist. Rather, in encouraging future research on this issue, we echo Cicchetti and Natsuaki (2014) and Shanahan et al. (2014), who point out that future work examining internalizing symptomatology will need to invoke an integrative perspective that investigates multiple co-active and interactive factors at different levels (e.g., family, school) of dynamic developmental systems in order to better understand the etiological underpinnings and developmental course of internalizing symptomatology. Such work, we believe, will yield potentially important implications for basic research into the nature of internalizing symptomatology as well as applied prevention and intervention efforts for youth with internalizing problems.

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Authors' Contributions JDH contributed to the conceptualization of the study, performed the statistical analyses, and drafted the manuscript; GIR contributed to the conceptualization of the study, interpretation of the data, and drafting of the manuscript; EC and CBLF also assisted in interpretation of the analyses and in the drafting of the manuscript. All authors read and approved the final manuscript.

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Compliance with Ethical Standards

Conflict of Interest The authors declare that they have no competing interests.

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COVID-19 PANDEMIC AND CHILD MENTAL HEALTH: AN INVITED DISCUSSION SECTION

Introduction: The influence of COVID-19 pandemic policy on child and adolescent mental health: strong signal or mostly noise?

With the exception of the vigorous and intense debate surrounding SARS-CoV-2 vaccination efficacy and COVID-19 policy mandates, arguably no discourse surrounding the pandemic has been more contentious and polarized in both Canada and the United States than the proposed and/or claimed effects of social restriction measures on mental health (1–6). In particular, the effects of school closures (5–7) and other lock down measures on child and adolescent mental health have been vigorously debated on social media—especially among credentialed academics, medical doctors, and scientists on Twitter—often drawing from various academic studies or review articles that appear to support one of two 'prototype' conclusions:

1. that school closures and lock downs had much more modest overall negative effects on child and adolescent mental health than portrayed in media and other narratives (or possibly had salubrious effects on some mental health indices, such as reducing the incidence of youth suicide (7)) and media and public official proclamations to the contrary reflected sensationalism that gave rise to a moral panic; or
2. that school closures and lock downs had meaningful cumulative adverse impacts of varying magnitudes on child and adolescent mental health.

Of course, as is so often the case in matters of human psychosocial functioning, determinative, robust single-variable 'mechanistic' effects are seldom found and nuance in drawing conclusions is the rule rather than the exception. In today's social media environment, however, nuance is rarely encountered. Rather, partisans ascribing to one or the other prototype conclusions described above seek to discredit the other, often in divisive, theatrical ways enabled by social media platforms (8). Indeed, one of the motivations for developing this special section of the *Journal of the Canadian Academy of Child and Adolescent Psychiatry* (JCACAP) arose as a consequence of observing such contentious debate around COVID-19 pandemic policy occurring on Twitter between academics, doctors, and researchers. Given their academic and medical credentials, and in many cases large number of social media followers, these individuals likely influenced public perception of regional, local, and federal pandemic policy in both the United States and Canada. As such, this special section of the journal reflects an effort to provide an organized, academic forum for researchers and scientists from both Canada and the United States an opportunity to engage in a more robust, civil, and principled discourse concerning whether—and how much—COVID-19 related lock downs, and in particular school closures, may have had on child and adolescent mental health functioning.

The focal commentary by Black et al. (9) makes the case that the portrayal of pandemic restrictions by the media and professional organizations, especially those concerning school closure and remote learning, were depicted as having more pernicious consequences and harm for child and adolescent mental health than the data addressing these questions warranted. The rejoinders from Vaillancourt et al. (10) and Vidal et al. (11) push back against this narrative frame with data and analyses of their own, suggesting that the effects of pandemic restrictions on youth mental health functioning were deleterious and non-negligible in magnitude, and likely had disproportionate impact on the most vulnerable youth.

Finally, the rejoinder by Ray (12) stitches the pieces together and highlights that critical methodological issues, including empirical formalizations of endpoint constructs, data

reduction approaches, the time period studied, and other methodological, statistical, and conceptual considerations influence conclusions researchers and research groups draw as they relate to the 'net effect' impact of pandemic policy on child and adolescent mental health. Nonetheless, investigating the influence of pandemic policy on child and adolescent mental health with precision and nuance is crucial to better understand how to address current negative mental health impacts, prevent future undue influence of ineffective or iatrogenic policy, and to communicate openly and honestly with the public about the need for given social restrictions during future pandemic circumstances.

Collectively, the investigations and synopses in this special section also underscore the need for moving beyond a 'net effect determination' toward a focus on understanding why some youth deteriorated while others did not to advance understanding of mechanisms of risk and resilience that plausibly impacted the outcomes of young people to pandemic-related interventions. Such work will also inform the study of multifinality and equifinality (13,14) as they relate to mental health pandemic science, while also providing a natural integration point with the broader discipline of developmental psychopathology which brings together scientists and practitioners from across the psychological and medical fields (15).

Such questions will require high-quality longitudinal data and analyses, underscoring the need for patience and equipoise from researchers and journalists when discussing and interpreting what the data reveal, especially as findings may inform potential implications for pandemic policy (16). Answering such questions also underscores the inherent issue of 'levels' in psychopathology research (17), and the need for investigators to be clear when communicating to the public about the results of their investigation whether they primarily inform policy implementation and practical useability/actionability at the public health level, or basic mechanistic understanding of disease process.

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My Diversity, Equity, & Inclusion (DEI) Statement for a Recent Academic Job Posting

A Principled Stand Against Mandatory DEI Statements in Academic Job Hiring

J.D. HALTIGAN

FEB 23, 2023

Note to readers: As many of my readers are aware, the use of mandatory diversity, equity, and inclusion (DEI) statements in the academic setting for hiring, promotion, tenure, and other forms of advancement or public acknowledgement are pervasive. Many public intellectuals, academics, legislators, and investigative journalists have raised alarms about the use of the DEI rubric on several grounds including civil rights, discrimination, and more generally the degradation of academic research and teaching in the university setting. I share these views and believe the DEI rubric in the Academy has also contributed to creating a corrosive and hostile environment that is intolerant of viewpoint diversity and is anathema to high-quality research and teaching. When a recent university job posting for a position I believe I am well-suited for came to my attention, I decided to apply for the position and penned the below statement for my DEI element of the application. I am posting it here, in its entirety with only university-identifying information redacted, as I strongly believe taking a principled stand against the use of the DEI rubric in the Academy is crucial for the continued survival of our institutions of higher learning as they were intended: bastions of the unfettered pursuit of knowledge and truth and the immersion of its students into the principles of liberal discourse and the development of critical thought.

John D. Haltigan, Ph.D

University of *** Faculty Position Diversity, Equity, & Inclusion (DEI) Statement**

Against the use of DEI Statements in Faculty Job Searches

Throughout the course of my academic trajectory, I have had several research and teaching experiences which have enabled me to contribute to diversity through research, teaching, and service. In addition to these experiences as part of my educational training, my experience in the mental health field working with adolescents from a variety of ethnic and sociocultural backgrounds has provided me with a deep appreciation of the importance of a sensitivity to the social and cultural factors that shape human development. I am committed to colorblind inclusivity, viewpoint diversity, merit-based evaluation, and value outreach to underrepresented groups in higher education. Across all of my teaching and mentorship, I have endeavored to treat students and mentees equally, without regard to identity-based characteristics. Taken together, the above experiences position me well to carry out the mission of the University of ***** which is described as:

*(a) providing high-quality undergraduate programs in the arts and sciences and professional fields, with emphasis upon those of special benefit to the citizens of *****; (b) offering superior graduate programs in the arts and sciences and the professions that respond to the needs of *****, as well as to the broader needs of the nation and the world; (c) engaging in research, artistic, and scholarly activities that advance learning through the extension of the frontiers of knowledge and creative endeavor; (d) cooperating with industrial and governmental institutions to transfer knowledge in science, technology, and health care; (e) offering continuing education programs adapted to the personal enrichment, professional upgrading, and career advancement interests and needs of adult *****; and (f) making available to local communities and public agencies the expertise of the University in ways that are consistent with the primary teaching and research functions and contribute to social, intellectual, and economic development in the Commonwealth, the nation, and the world.*

However, I believe that the use of diversity, equity, and inclusion (DEI) statements in evaluating candidates for positions in higher education and academia are anathema to the ideals and principles of rigorous scholarship, and the sound practice of science and teaching—all of which public universities were created to uphold. DEI statements have become a political litmus test for political orientation and activism that has created an

untenable situation in higher academia where diversity of thought—the bedrock of liberal education—is neither promoted nor tolerated. Public trust in our universities has been severely diminished as a consequence. As the noted American sociologist and sociocultural scholar Philip Rieff noted decades ago in relation to the vogue for politically engaged teaching and scholarship "inactivism is the ticket."

Several recent investigative journalism efforts have documented how DEI statements have been used to screen and penalize applicants for not possessing 'correct' political ideas or endorsing activist ideologies, such as the 'anti-racist' strand of 'scholarship' developed and promoted by Ibram Kendi as well as concepts such as "intersectionality", a term coined by one of the architects of critical race theory, Kimberlé Crenshaw. Most crucially, what is meant by "equity" is inconsistent with the principle of 'equal opportunity' and is used to denote equal outcomes irrespective of inherent capability or merit; disparities in outcome are ipso facto taken as indicating social oppression or injustice; other factors—including biological or genetic ones—are dismissed out of hand. These exposés have lead administrators at several institutions to harshly criticize DEI-based research and scholarship and to suspend the use of mandatory DEI statements in hiring and promotion.

Moreover, there is a growing recognition among scholars, public intellectuals, and elected legislators that mandatory DEI statements are not only unethical, but also serve to preclude the very attributes they presume to enhance, instead creating censorious, divisive, polarizing, and otherwise inhospitable workplace cultures that are at odds with the core principles upon which public universities have been founded. In short, the institutionalization of DEI has become an iatrogenic force, making the university ill-suited to producing reliable knowledge. Indeed, the Academic Freedom Alliance called for an end to mandatory diversity statements. Renowned social psychologist and liberal public intellectual Jonathan Haidt publicly announced his resignation from the Society for Personality and Social Psychology over required DEI statements. Several other scholars have called for the abolition of DEI statements citing their violations of civil rights law, use as political screeners, and more generally creating a divisive and dysfunctional workplace culture. Most recently, the Foundation for Individual Rights and Expression (FIRE) has introduced model legislation that aims to address DEI bureaucracy's "chilling effect on campus".

My Vision to Encourage Diversity, Inclusion, & High-Quality Learning

I look forward to continuing to engage in mentoring activities, research activities, and community outreach that enhance viewpoint diversity and encourage attention to high quality, rigorous scholarship, research, and teaching at the University of *****. I value outreach that encourages diversity and inclusion of underrepresented minorities in higher education and welcome collaborative opportunities with others who do not necessarily share the same ideological standpoint as my own. As can be gleaned from my curriculum vitae, I am a member of Heterodox Academy, a non-partisan collaborative of educators who believe open inquiry, diverse viewpoints, and constructive disagreement are critical to research and education. I have provided mentorship to several students from underrepresented minority groups. Many of these students explicitly sought out my mentorship due to my clear position, communicated on social media, that I reject activist 'scholarship' that is neither conceptually coherent nor methodologically sound.

I will continue my biologically and genetically informed research in developmental psychopathology that addresses questions relevant to disparate outcomes across different ethnic and cultural groups. Findings from my previous work, published in *Child Development,* provided insight into how the legacy of economic hardship may, in conjunction with biological and genetic factors, contribute to different stylistic ways of talking about early life attachment experiences among African American pregnant women. I subsequently extended this work (Haltigan et al., 2019, *Journal of Child Psychology and Psychiatry*), and aim to programmatically advance work aimed at improving our understanding of how underrepresented minority groups and other immigrant populations talk about their early life and attachment experiences. Such work provides an opportunity to identify aspects of resilience in diverse underrepresented populations that can inform prevention and intervention science, and promote child, family, and community health amongst these populations (Beal Spencer et al., 2006).

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Psychology: Developmental Psychology - Assistant Professor (Initial Review 09/19/22)

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Job #JPF01346

- Psychology / Social Sciences Division / UC Santa Cruz

POSITION OVERVIEW

Position title: Assistant Professor of Developmental Psychology
Salary range: Commensurate with qualifications and experience; academic year (nine-month basis).
Anticipated start: July 1, 2023, with the academic year beginning in September 2023 and is contingent upon budgetary approval. Degree requirements must be met by September 22, 2023 for employment effective beyond that date.

APPLICATION WINDOW

Open date: July 21, 2022

Most recent review date: Monday, Sep 19, 2022 at 11:59pm (Pacific Time)
Applications received after this date will be reviewed by the search committee if the position has not yet been filled.

Final date: Friday, Jun 30, 2023 at 11:59pm (Pacific Time)
Applications will continue to be accepted until this date, but those received after the review date will only be considered if the position has not yet been filled.

POSITION DESCRIPTION

The Department of Psychology at the University of California, Santa Cruz (UCSC) invites applications for an Assistant Professor (tenure-track) in Developmental Psychology. We seek to enhance our Developmental Psychology program's long-established strengths in studying the lived experiences of children and youth from diverse backgrounds, bridging traditionally separate areas of developmental research, and integrating cultural, interpersonal, and individual aspects of human development, especially as they relate to global and community health.

We are particularly interested in developmental psychology scholars whose research addresses diversity in human development. In addition, we seek a scholar whose research addresses the intersection of developmental psychology and global and/or community health. Health here is broadly construed to include psychological, mental or physical health with a focus on the well-being of children and youth in their families, peer relations, schools, and/or cultural communities. Some examples include (but are not limited to) candidates whose research examines:
● Cultural assets that promote healthy development in the contexts of inequities related to gender, ethnicity/race, social class, and/or sexuality.
● Conditions and practices that leverage the psychological strengths of children from historically under-served backgrounds in the U.S. or other countries.
● Familial, peer, educational, political, cultural, technological, and/or economic systems related to healthy psychological development.
● Health systems and community health services as contexts for children's and youth's psychological health, well-being, and resilience.

Ideal candidates will be able to teach for both Psychology and the new UCSC Global and Community Health Program (https://transform.ucsc.edu/work/gch/ (https://web.archive.org/web/20230529151248/https://transform.ucsc.edu/work/gch/)), an interdisciplinary program with research foci spanning Psychology and the social determinants of health and health policy (with health broadly construed).

Applicants should be actively engaged in research with the promise of continued research productivity. In addition, they should be capable of teaching courses at both graduate and undergraduate levels, including core courses in the new BA in global and community health. We are looking for candidates who will contribute to the diversity and excellence of our academic community through their research, teaching, and service, including the mentoring of doctoral students. The successful candidate must work well with students, faculty, and staff from a wide range of social and cultural backgrounds. UC Santa Cruz is a Hispanic-Serving Institution (HSI) and an Asian American and Native American Pacific Islander Serving Institution (AANAPISI) with a high proportion of first-generation undergraduate students and a growing number of first-generation college students entering our doctoral program.

We welcome candidates who understand the barriers facing women and other minoritized people who are underrepresented in higher education careers (as evidenced by life experiences and educational background), and who can clearly articulate their contributions to equity, diversity and justice with respect to teaching, mentoring, research, life experiences, or service towards building an equitable, inclusive, and diverse scholarly environment.

The chosen candidate will be expected to sign a statement representing that they are not the subject of any ongoing investigation or disciplinary proceeding at their current academic institution or place of employment, nor have they in the past ten years been formally disciplined at any academic institution/place of employment. In the event the candidate cannot make this representation, they will be expected to disclose in writing to the hiring Dean the circumstances surrounding any formal discipline that they have received, as well as any current or ongoing investigation or disciplinary process of which they are the subject. (Note that discipline includes a negotiated settlement agreement to resolve a matter related to substantiated misconduct.)
Department: https://psychology.ucsc.edu/ (https://web.archive.org/web/20230529151248/https://psychology.ucsc.edu/)

QUALIFICATIONS

Basic qualifications (required at time of application)

Applicants must have a Ph.D. (or equivalent foreign degree) in Psychology or related field. The successful candidate must have both a record of empirical research and a record of teaching. It is expected that the degree requirement will be completed by September 22, 2023.

APPLICATION REQUIREMENTS

Document requirements

- Cover Letter - Letter of application that briefly summarizes your qualifications and interest in the position as described in this add.
- Curriculum Vitae - Your most recently updated C.V.
- Statement of Research** - Research statement describing your program of research and possible future directions .
- Statement of Contributions to Diversity, Equity, and Inclusion** - Statement addressing your understanding of the barriers facing traditionally underrepresented groups and your past and/or future contributions to diversity, equity, and inclusion through teaching and professional or public service. Candidates are urged to review guidelines on statements (see (https://web.archive.org/web/20230529151248/https://apo.ucsc.edu/diversity.html)https://apo.ucsc.edu/diversity.html (https://web.archive.org/web/20230529151248/https://apo.ucsc.edu/diversity.html)) before preparing their application.

  ** Initial screening of applicants will be based *only* on the research statement and the statement on contributions to diversity, equity, and inclusion**
- Statement of Teaching - Teaching statement describing your teaching experience and overall teaching interests; also, briefly address how you can contribute teaching for the new UCSC Global and Community Health Program ((https://web.archive.org/web/20230529151248/https://transform.ucsc.edu/work/gch/)https://transform.ucsc.edu/work/gch/ (https://web.archive.org/web/20230529151248/https://transform.ucsc.edu/work/gch/))
- Most significant reprints or preprints (1 of 2 required)
- Most significant reprints or preprints (2 of 2 required)
- Most significant reprints or preprints (1 of 3 optional) (Optional)
- Most significant reprints or preprints (2 of 3 optional) (Optional)
- Most significant reprints or preprints (3 of 3 optional) (Optional)

Reference requirements

- 3-5 letters of reference required

Applications must include confidential letters of recommendation* (a minimum of 3 are required and a maximum of 5 will be accepted). Please note that your references, or dossier service, will submit their confidential letters directly to the UC Recruit System.

*All letters will be treated as confidential per University of California policy and California state law. For any reference letter provided via a third party (i.e., dossier service, career center), direct the author to UCSC’s confidentiality statement at http://apo.ucsc.edu/confstm.htm (https://web.archive.org/web/20230529151248/http://apo.ucsc.edu/confstm.htm).

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Help contact: psycdept@ucsc.edu (https://web.archive.org/web/20230529151248/mailto:psycdept@ucsc.edu)

CAMPUS INFORMATION

The University of California is an Equal Opportunity/Affirmative Action Employer. All qualified applicants will receive consideration for employment without regard to race, color, religion, sex, sexual orientation, gender identity, national origin, disability, age, or protected veteran status. UC Santa Cruz is committed to excellence through diversity and strives to establish a climate that welcomes, celebrates, and promotes respect for the contributions of all students and employees. Inquiries regarding the University's equal employment opportunity policies may be directed to the Office for Diversity, Equity, and Inclusion at the University of California, Santa Cruz, CA 95064 or by phone at (831) 459-2686.

Under Federal law, the University of California may employ only individuals who are legally able to work in the United States as established by providing documents as specified in the Immigration Reform and Control Act of 1986. Certain UCSC positions funded by federal contracts or sub-contracts require the selected candidate to pass an E-Verify check (see https://www.uscis.gov/e-verify (https://web.archive.org/web/20230529151248/https://www.uscis.gov/e-verify)). The university sponsors employment-based visas for nonresidents who are offered academic appointments at UC Santa Cruz (see https://apo.ucsc.edu/policy/capm/102.530.html (https://web.archive.org/web/20230529151248/https://apo.ucsc.edu/policy/capm/102.530.html)).

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If you need accommodation due to a disability, please contact Disability Management Services at roberts@ucsc.edu (https://web.archive.org/web/20230529151248/mailto:roberts@ucsc.edu) (831) 459-4602.

UCSC is committed to addressing the spousal and partner employment needs of our candidates and employees. As part of this commitment, our institution is a member of the Northern California Higher Education Recruitment Consortium (NorCal HERC). Visit the NorCal HERC website at https://www.hercjobs.org/regions/higher-ed-careers-northern-california/ (https://web.archive.org/web/20230529151248/https://www.hercjobs.org/regions/higher-ed-careers-northern-california/) to search for open positions within a commutable distance of our institution.

The University of California offers a competitive benefits package and a number of programs to support employee work/life balance. For information about employee benefits please visit https://ucnet.universityofcalifornia.edu/compensation-and-benefits/index.html (https://web.archive.org/web/20230529151248/https://ucnet.universityofcalifornia.edu/compensation-and-benefits/index.html)

As a condition of employment, you will be required to comply with the University of California Policy on Vaccination Programs – With Updated Interim Amendments (https://web.archive.org/web/20230529151248/https://policy.ucop.edu/doc/5000695/VaccinationProgramsPolicy). All Covered Individuals under the policy must provide proof of receiving the COVID-19 Vaccine Primary Series or, if applicable, submit a request for Exception (based on Medical Exemption, Disability, Religious Objection, and/or Deferral based on pregnancy or recent COVID-19 diagnosis and/or treatment) no later than the applicable deadline. All Covered Individuals must also provide proof of receiving the most recent CDC-recommended COVID-19 booster or properly decline such booster no later than the applicable deadline. New University of California employees should refer to Exhibit 2 (https://web.archive.org/web/20230529151248/https://policy.ucop.edu/doc/5000695/VaccinationProgramsPolicy#page=44), Section II.C. of the SARS-CoV-2 (COVID-19) Vaccination Program Attachment

(https://web.archive.org/web/20230529151248/https://policy.ucop.edu/doc/5000695/VaccinationProgramsPolicy#page=13) for applicable deadlines. All Covered Individuals must also provide proof of being Up-To-Date on seasonal influenza vaccination or properly decline such vaccination no later than the applicable deadline. Please refer to the Seasonal Influenza Vaccination Program Attachment (https://web.archive.org/web/20230529151248/https://policy.ucop.edu/doc/5000695/VaccinationProgramsPolicy#page=51). (Capitalized terms in this paragraph are defined in the policy.) Federal, state, or local public health directives may impose additional requirements. Additional information and resources for compliance are located on the campus Health Center site (https://web.archive.org/web/20230529151248/https://healthcenter.ucsc.edu/services)

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The University of California, Santa Cruz is committed to providing reasonable accommodations to applicants with disabilities (/web/20230529151248/https://recruit.ucsc.edu/accessibility).

See our Jeanne Clery Disclosure of Campus Security Policy and Campus Crime Statistics Act Annual Security Reports (https://web.archive.org/web/20230529151248/http://www.ucop.edu/ethics-compliance-audit-services/compliance/clery-act/clery-act-details.html)

(/)

Psychology: Assistant or Associate Professor, Quantitative Psychology (initial review Oct. 31, 2023)

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Job #JPF01612

- Psychology / Social Sciences Division / UC Santa Cruz

POSITION OVERVIEW

Position title: Assistant or Associate Professor, Quantitative Psychology
Salary range: The salary range for this position; academic year (nine-month basis):

Assistant Professor $98,000 - $110,000.
Associate Professor $120,000 - $150,000
The posted UC salary scales set the minimum pay determined by rank and/or step at appointment. “Off-scale salaries” and other components of pay, i.e., a salary that is higher than the published system-wide salary at the designated rank and step, are offered when necessary to meet competitive conditions.
See salary scales titled See FACULTY--LADDER RANKS--PROFESSOR SERIES (https://apo.ucsc.edu/docs/scales-crnt.pdf)
Percent time: Full-time, 100%
Anticipated start: July 1, 2024, with the academic year beginning in September 2024. Degree requirements must be met by September 16, 2024 for employment beyond that date.

APPLICATION WINDOW

Open date: September 1, 2023

Most recent review date: Tuesday, Oct 31, 2023 at 11:59pm (Pacific Time)
Applications received after this date will be reviewed by the search committee if the position has not yet been filled.

Final date: Sunday, Jun 30, 2024 at 11:59pm (Pacific Time)
Applications will continue to be accepted until this date, but those received after the review date will only be considered if the position has not yet been filled.

POSITION DESCRIPTION

The Psychology Department (https://psychology.ucsc.edu/ (https://psychology.ucsc.edu/)) at the University of California, Santa Cruz (UCSC) invites applications for an Assistant (tenure-track) or Associate (tenured) Professor in Quantitative Psychology. The chosen candidate could have a PhD in Quantitative Psychology or in another field of Psychology as long as they have expertise in quantitative methods and demonstrated excellence in teaching statistics at the graduate and undergraduate levels. The chosen candidate will be expected to lead the graduate program's quantitative curriculum. This includes teaching the program's first-year statistics series, which is a 2-quarter sequence on univariate and multivariate methods. We seek a colleague who is an expert at statistics and quantitative methodology but who also understands the value of qualitative methods. The candidate should have an interest in collaborating with researchers in cognitive, developmental, and/or social psychology, which are the three graduate areas in the department. We are especially interested in candidates who can contribute to the diversity and excellence of our academic community through their research, teaching, and service, and who will add to the department's contributions to campus initiatives in data science and critical data studies.

UC Santa Cruz values diversity, equity, and inclusion and is committed to hiring faculty who will work to advance these values. UC Santa Cruz is a Hispanic-Serving Institution (HSI) and an Asian American and Native American Pacific Islander Serving Institution (AANAPISI) with a high proportion of first-generation students. We welcome candidates who understand the barriers facing historically oppressed groups in higher education (as evidenced by life experiences and educational background) and who can clearly articulate participation in equity and diversity advancement efforts with respect to teaching, mentoring, research, and service towards building an equitable and scholarly environment. Activities promoting equity and inclusion at UC Santa Cruz will be recognized as important university service during the faculty promotion process. More information can be found: https://apo.ucsc.edu/diversity.html (https://apo.ucsc.edu/diversity.html)

The chosen candidate will be expected to sign a statement representing that they are not the subject of any ongoing investigation or disciplinary proceeding at their current academic institution or place of employment, nor have they in the past ten years been formally disciplined at any academic institution/place of employment. In the event the candidate cannot make this representation, they will be expected to disclose in writing to the hiring Dean the circumstances surrounding any formal discipline that they have received, as well as any current or ongoing investigation or disciplinary process of which they are the subject. (Note that discipline includes a negotiated settlement agreement to resolve a matter related to substantiated misconduct.)

Psychology Department: https://psychology.ucsc.edu/ (https://psychology.ucsc.edu/)

QUALIFICATIONS

Basic qualifications (required at time of application)
Applicants must have a Ph.D. (or equivalent foreign degree) in psychology or related field. The successful candidate must have both a record of empirical research and a record of teaching. It is expected that the degree requirement will be completed by September 16, 2024.

APPLICATION REQUIREMENTS

Document requirements

- Curriculum Vitae - Your most recently updated C.V.
- Cover Letter - Letter of application that briefly summarizes your qualifications and interest in the position.

- Teaching Statement**
- Research Statement**
- Statement of Contributions to Diversity, Equity, and Inclusion** - Statement on your contributions to diversity, equity, and inclusion, including information about your understanding of these topics, your record of activities to date, and your specific plans and goals for advancing equity and inclusion if hired at UC Santa Cruz. Candidates are urged to review guidelines on statements (see (https://apo.ucsc.edu/diversity.html)https://apo.ucsc.edu/diversity.html (https://apo.ucsc.edu/diversity.html)) before preparing their application.

  ** **Initial screening of applicants will be based *only* on the research statement, teaching statement, and the statement on contributions to diversity, equity, and inclusion**
- Most significant reprints or preprints (1 of 2 required)
- Most significant reprints or preprints (2 of 2 required)
- Most significant reprints or preprints (1 of 3 optional) (Optional)
- Most significant reprints or preprints (2 of 3 optional) (Optional)
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Help contact: psycdept@ucsc.edu (mailto:psycdept@ucsc.edu)

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As a University employee, you will be required to comply with all applicable University policies and/or collective bargaining agreements, as may be amended from time to time. Federal, state, or local government directives may impose additional requirements.

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