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Evaluating Emotional and Biological Sensitivity to Maternal Behavior Among Self-Injuring and Depressed Adolescent Girls Using Nonlinear Dynamics

Sheila E. Crowell[1], Jonathan E. Butner[1], Travis J. Wiltshire[1], Ascher K. Munion[1], Mona Yaptangco[1], and Theodore P. Beauchaine[2]
[1]University of Utah and [2]The Ohio State University

Abstract
High sensitivity and reactivity to behaviors of family members characterize several forms of psychopathology, including self-inflicted injury (SII). We examined mother-daughter behavioral and psychophysiological reactivity during a conflict discussion using nonlinear dynamics to assess asymmetrical associations within time-series data. Depressed, SII, and control adolescents and their mothers participated ($N = 76$ dyads). We expected that (a) mothers' evocative behaviors would affect behavioral and psychophysiological reactivity among depressed and, especially, SII adolescents, (b) adolescents' behaviors would not evoke mothers' behavioral or physiological reactivity, and (c) control teens and mothers would be less reactive, with no dynamic associations in either direction. Convergent cross-mapping with dewdrop regression, which identifies directional associations, indicated that mothers' behaviors evoked behavioral responses among depressed and SII participants, but evoked psychophysiological reactivity for SII teens only. There were no effects of adolescents' behavior on mothers' reactivity. Results are interpreted based on sensitivity theories and directions for further research are outlined.

Keywords
depression, dynamical systems, self-inflicted injury, multispatial convergent cross-mapping

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According to several prominent theories, youth who are vulnerable to psychopathology are more sensitive than typical children (e.g., Belsky & Pluess, 2009; Cicchetti, Ackerman, & Izard, 1995; Linehan, 1993). There are many subtle variations on this theme. Proponents of the biological sensitivity to context theory (BSCT) posit that vulnerable youth are like orchids—thriving under optimal conditions yet wilting when environments are less than ideal (e.g., Boyce & Ellis, 2005). Research on gene-environment interactions indicates that certain allelic variants transmit vulnerability to neuroticism, negative affectivity, or "stress sensitivity," and when carriers of such vulnerabilities are exposed to adversity, psychopathology often ensues (e.g., Caspi, Hariri, Holmes, Uher, & Moffitt, 2010). Developmental theories of self-inflicted injury (SII) and borderline personality disorder suggest that vulnerability is highest when impulsive or emotionally sensitive youth experience invalidation and coercive conflict escalation in local family environments (Crowell, Beauchaine, & Linehan, 2009; Crowell et al., 2013).

Although many theories focus on child sensitivities, more contemporary developmental psychopathology models propose transactional, bidirectional, and evocative effects of family members on one another (e.g., Snyder, Schrepferman, & St Peter, 1997). According to this perspective, biologically sensitive, behaviorally reactive children are more challenging to raise because (a)

Corresponding Author:
Sheila E. Crowell, Department of Psychology, University of Utah, 380 South 1530 East, Rm. 502, Salt Lake City, UT 84112
E-mail: sheila.crowell@psych.utah.edu

they sometimes evoke compromised caregiving from parents who (b) are products of their own developmental histories in which their neurobiological sensitivities were shaped by challenging home environments, neighborhood risk, loss, abandonment, or any number of severe or chronic stressors (Cicchetti, 2016; Lytton, 1990). Thus, children's neurobiological and emotional sensitivities and reactivities *interact with and amplify* high-risk family environments.

According to BSCT, early neurobiological and emotional reactivities can also lead to adaptive outcomes in protective family environments (Obradović, Bush, Stamperdahl, Adler, & Boyce, 2010). In essence, BSCT suggests that emotional sensitivity broadens children's range of adaptive responding, which is "tuned" to untoward outcomes in contexts of adversity, but favorable outcomes in contexts of enrichment. Following from this logic, BSCT theorists hypothesize that ongoing calibration of emotional and physiological stress systems occurs within parent-child relationships (e.g., Del Giudice, Ellis, & Shirtcliff, 2011), which are often compromised in families of self-injuring youth (see, e.g., Crowell, 2016). To date, there are few tests of sensitivity theories among self-injuring samples. However, theoretical work suggests that self-injury is characterized by early emotional sensitivity, and that parent-child interaction patterns amplify and reinforce emotional, behavioral, and biological reactivity over time (Crowell et al., 2009). It is important that this transactional perspective does not place blame on parents or children. Indeed, when blame on individuals is minimized and problems are instead attributed to dynamic processes among vulnerable families, clinicians can gain considerable traction toward effecting behavior change.

Historically, studies of family-level predictors of psychopathology have focused predominantly on caregiver behavior. For example, low acceptance (Garber, Robinson, & Valentiner, 1997), low support with high conflict (Sheeber, Hops, Alpert, Davis, & Andrews, 1997), low nurturance and high overprotection (Betts, Gullone, & Allen, 2009), low positive engagement (Olino et al., 2015), and less secure attachment (Armsden, McCauley, Greenberg, Burke, & Mitchell, 1990) are all associated with adolescent depressive symptoms or suicidality. Recently, however, scholars have focused more attention on interactions between child vulnerabilities and parenting behavior (e.g., Crowell et al., 2014). In one such study, observed maternal aggression interacted with adolescents' hippocampal, amygdalar, and anterior cingulate volumes to predict adolescent depressive symptoms better than maternal aggression alone (Yap et al., 2008). In another study, the interaction between observed mother-daughter negativity/conflict and adolescent peripheral serotonin levels accounted for 64% of the variance in lifetime SII among vulnerable adolescent girls, even though main effects were small (Crowell et al., 2008).

More recently, studies have emerged that examine complex family dynamics of depressed and self-injuring adolescents (e.g., Crowell et al., 2013). For example, Crowell et al. (2014) evaluated dyadic concordance of behavioral and physiological measures of emotion regulation among depressed adolescents, some of whom self-injured, control adolescents, and their mothers. Typical controls evidenced concordant physiological regulation—assessed via respiratory sinus arrhythmia (RSA) increases—during minutes when they or their partner was more aversive. In contrast, clinical dyad members exhibited concordant physiological dysregulation—assessed via RSA decreases—during minutes when they or their partner was more aversive. In another study of cross-lagged associations between adolescent depression and combined adolescent/self-reports of parental hostility, reciprocal associations were observed over time between child depressive symptoms and parent hostility for mother-daughter dyads (Lewis, Collishaw, Thapar, & Harold, 2014). These studies suggest that some adolescents are vulnerable to aversive parent behavior and that aversive adolescent behaviors precede increases in problematic parent behaviors, such as hostility, among vulnerable dyads.

Developmental psychopathologists have used painstaking moment-to-moment sequential analyses to characterize the quality of parent-child interactions among externalizing samples. These studies reveal that escalatory family dynamics and associated physiological dysregulation/emotional lability are shaped slowly and maintained longitudinally across thousands of interactions between children and their caregivers (Beauchaine & Zalewski, 2016; Bronfenbrenner, 1979; Crowell et al., 2008; Snyder, Edwards, McGraw, & Kilgore, 1994; Snyder et al., 1997). However, almost no studies have examined the quality of moment-to-moment interactions between self-injuring or depressed adolescents and their parents with attention to biological sensitivity theories. Thus, it is unclear whether such youth are emotionally and physiologically reactive to their mothers (consistent with sensitivity theories), more likely to evoke their mother's emotional behavior and physiology, or whether both processes operate concurrently. This question motivated the current analyses, in which we use a recently developed statistical approach—multispatial convergent cross-mapping (Clark et al., 2015; Sugihara et al., 2012)—which, to our knowledge, has not been adopted by psychological scientists.

Multispatial Convergent Cross-Mapping

Multispatial convergent cross-mapping (MCCM) combines dewdrop regression (a form of bootstrapped

regression across multiple time series) with convergent cross-mapping (CCM). These combined methods arguably provide a mechanism for testing putative "causal" associations in nonlinear dynamical systems (Clark et al., 2015; Sugihara et al., 2012).[1] CCM relies on time-delayed reconstruction (Takens, 1981), in which lagged copies of observed variables are used to reconstruct properties of the system. This makes it possible to characterize how the system behaves over time. MCCM involves cross-comparisons between reconstructed state spaces (maps that predict where we would expect a person to go over time, given her current value) to determine the degree to which one variable can be used to reliably estimate the other across time (Sugihara et al., 2012).

Assessing driving factors during moment-to-moment conflict has historically been challenging because traditional statistical approaches cannot fully capture temporal patterns of behavior and physiology as they emerge over the course of an interaction. Given this, it has recently been noted that social interactions should be treated as dynamic systems and analyzed accordingly (e.g., Richardson, Dale, & Marsh, 2014). Adopting a dynamical systems approach provides both theoretical and analytic techniques to characterize temporal patterns of behavior and physiology on a moment-to-moment basis during mother-daughter social interactions. It is important that recent work argues for a need to study the dynamics of *both* behavior and physiology during parent-child interactions, particularly among vulnerable dyads (e.g., Beauchaine & Zalewski, 2016). In addition to findings outlined earlier, Giuliano, Skowron, and Berkman (2015) recently demonstrated that positive behavioral synchrony between mothers and daughters was associated with greater physiological changes.

During dyadic interactions, changes in behavior and physiology occur rapidly, sequentially, and at times simultaneously. It is therefore difficult—perhaps even futile—to establish temporal precedence (i.e., A must occur before B for A to "drive" B). When examining dynamic systems, however, patterns of behavior are a function of multidirectional interactions among variables. For this reason, we leverage MCCM given its ability to model driving relations in dynamic systems. Specifically, if we begin by assuming bidirectional associations, MCCM tests for asymmetry within these associations. When a daughter's behavioral state reliably estimates her mother's behavioral state, as determined from MCCM, we can infer a driver-driven association. Consistent with systems theory, we differentiate driver-driven associations from the traditional tenets of "causal" arguments (see Note 1). The systems literature assumes multidirectional causality through emergence and "driving" merely describes asymmetry in the observed forces.

Observational Method

Most studies of adolescent sensitivity and reactivity use self- or parent-reports to characterize adolescent vulnerabilities and environmental stressors. These methods undoubtedly capture some variability in vulnerability traits and contextual risk factors. However, such approaches neglect dynamic processes through which behavior and reactivity patterns are shaped during moment-to-moment interactions. This is a significant limitation given the centrality of transacting behavioral, biological, emotional, and cognitive processes to contemporary developmental psychopathology models (see Hinshaw, 2015). Studies that examine dyadic behavioral and psychophysiological reactivity can therefore make unique contributions to this literature. For example, behavioral coding methods often capture observed emotional behavior more objectively than self-reports of parenting or parent-child relationships. Similarly, psychophysiological responses occur rapidly and often precede conscious awareness of emotional states. Finally, observed behavior and psychophysiology capture different aspects of self-regulation, arousal, and reactivity.

RSA and electrodermal activity (EDA) are widely used psychophysiological measures in psychological research. Under appropriate stimulus conditions, RSA captures parasympathetic nervous system (PNS) influences on cardiac activity and reactivity (see Beauchaine, 2001; Beauchaine & Thayer, 2015). As detailed elsewhere (e.g., Porges, 2001), the PNS exerts inhibitory effects on peripheral target organs. High RSA reflects greater PNS regulation of HR and is associated with adaptive social and emotional functioning, positive mental health outcomes, and strong emotion regulation (Beauchaine, 2001; Beauchaine & Thayer, 2015). EDA is a measure of sympathetic nervous system (SNS) influences on eccrine sweat gland activity. The SNS has an activating influence on peripheral physiological systems, allowing organisms to respond quickly to stress. Higher EDA is an index of more SNS activity and correlates with behavioral inhibition and both state and trait anxiety (see Brenner, Beauchaine, & Sylvers, 2005). In contrast, low EDA is associated with fearlessness, externalizing symptoms, and aggression (see Crowell et al., 2012; Lorber, 2004). An advantage of measuring RSA and EDA is the ability to examine both PNS and SNS measures of physiological reactivity to environmental stressors. Because RSA and EDA index regulation and activation, respectively—which serve distinct evolutionary functions—we measured both.

The Current Study

Our objective was to better understand adolescent SII and depression by examining dynamic influences of

mothers' and daughters' behaviors, psychophysiological response patterns, and emotional reactivities on one another. We selected a conflict task that elicits strong emotions from both adolescents and mothers (see, e.g., Crowell et al., 2014). Psychophysiological measures were collected throughout the task from both dyad members. Concurrent assessment of behavioral, emotional, and physiological responding is consistent with a multiple levels of analysis approach to conceptualizing and understanding individuals as "full systems." Previous research supports a full-system approach, for example, finding that desynchrony across response systems is associated with emotion and behavior dysregulation (e.g., Marsh, Beauchaine, & Williams, 2008). No previous studies compare self-injuring and depressed adolescents using dynamic systems approaches. However, self-injuring adolescents are often described as uniquely sensitive to both neutral and aversive parenting behaviors (Miller, Rathus, & Linehan, 2007). Moreover, this sensitivity may contribute to emergence of self-harm and borderline personality traits, even within typical family environments (Crowell et al., 2009; Linehan, 1993). Thus, we seek to examine whether self-injuring youth are more emotionally and behaviorally reactive to maternal conflict behavior relative to depressed and typical youth.

Methodologically, prior research in this area has relied on lagged models, conditional probabilities, actor-partner interaction models, or correlational methods. These approaches capture general linear associations but are likely insensitive to nonlinear associations (Sugihara et al., 2012). Moreover, MCCM outperforms correlation-based measures in detecting bivariate directional associations, even in very short time series data (Clark et al., 2015; Sugihara et al., 2012). Thus, an additional objective is to apply this relatively new method to 10 min of mother-daughter conflict data with dyadic behavioral coding and psychophysiology. It is important that the goal of MCCM is not to evaluate increases or decreases in dependent variables (e.g., EDA decreases), but rather to evaluate three competing hypotheses within mother-daughter interactions: (a) Do mother's behaviors exhibit driving relations with adolescent's behaviors and physiology, consistent with biological sensitivity to context theories? (b) Do daughter's behaviors exhibit driving relations with mother's behaviors and physiology, consistent with theories that child psychopathology evokes aversive parental behavior? or (c) Are both processes operative? We hypothesized that the first hypothesis is most probable given the predominance of sensitivity theories in the literature. Thus, we expected that self-injuring and depressed adolescents would be more reactive to their mothers' behaviors than typical control adolescents across measures of observed behavior and physiology and that self-injuring adolescents may be even more reactive to mother behavior than depressed and typical control teens, consistent with limited research using other methods (see the discussion earlier).

Method

Participants

Participants included 76 female adolescents (n = 26 self-injuring, n = 24 depressed non-self-injuring, n = 24 control). Adolescents were 13 to 17 years old and participated with their biological mothers. Participants were recruited from outpatient clinics, psychiatric hospitals, newspaper and classified advertisements, and public school newsletters. A total of 84 mother-teen dyads were initially enrolled in the study. However, 11 dyads had insufficient data for these analyses due to failure of physiological or video recording equipment (n = 5), inability or refusal to return for the physiological assessment (n = 4), or arriving with a guardian other than the biological mother (n = 2). These 11 participants were not different demographically or diagnostically from the overall sample, all Fs ≤ 1.07, all ps ≥ .29. Demographic and diagnostic data comparing the three groups are reported elsewhere and are not a focus of this study (Crowell et al., 2012). Briefly, SII adolescents differed from typical adolescents across nearly every self- or parent-report of psychopathology. SII and depressed participants differed on some self-report scales of externalizing, delinquent behavior, substance use, and anxiety/depression, and on parent-report scales of substance use. However, SII and depressed adolescents did not differ on structured diagnostic interviews of depression or depressive symptoms. Thus, both SII and depressed adolescents reported high degrees of psychopathology and distress. There were no demographic differences between groups.

Self-injuring adolescents were included if they engaged in any self-injurious behaviors (suicidal or nonsuicidal) three or more times in the past 6 months, or five or more lifetime events with at least one occurring in the past 6 months. These criteria were selected to identify adolescents with a relatively severe and recent history of self-harm. Depressed adolescents were included if they met criteria of the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., text revision; *DSM-IV-TR*; American Psychiatric Association, 2000) for unipolar depression at least once in the past year. Exclusion criteria for all participants included mental retardation, schizophrenia spectrum disorders, medications that interfere with cardiac psychophysiology (e.g., stimulants, mood stabilizers, antihistamines),[2] and use of recreational drugs during the week of assessment. Depressed adolescents were also

excluded if they endorsed mania or lifetime SII. Typical controls were excluded if they reported any *DSM* diagnosis or lifetime SII.

Interested adolescents and their mothers were administered a telephone screening interview to determine whether inclusion/exclusion criteria were met. Dyads who met criteria were invited to participate in two separate lab visits lasting approximately 2 to 3 hr each. Adolescents were given $20 for completing Visit 1 and $40 for completing Visit 2, which were scheduled approximately 2 weeks apart. Study procedures were approved by the local Institutional Review Board and written informed consent/assent were obtained from mothers and daughters.

Procedure

Visit 1. The first visit consisted of diagnostic interviews and self-report measures to confirm study eligibility and characterize the sample. These data have been reported elsewhere and are not a focus of this study (see Crowell et al., 2014). After questionnaires and interviews were completed, mother-adolescent dyads were scheduled to return 2 weeks later to complete the physiological component of the study.

Visit 2. During the second visit, adolescents and their mothers independently completed the Issues Checklist (Prinz, Foster, Kent, & O'Leary, 1979), which was used to identify a frequent but moderate intensity discussion topic. This checklist includes a list of common areas of conflict between parent and child and respondents rate the frequency and intensity of each area of conflict. Frequency ratings range from 1 (*never*) to 5 (*very often*), and intensity is ranked from 0 (*calm*) to 40 (*intense*). Mother and daughter responses on this measure were collected and compared by a research assistant, who was trained to identify a topic that both participants rated as ≥4 on frequency and ≤20 on intensity. All dyads identified a topic with a score of 4 to 5 on frequency and a 20 on intensity. Thus, there were no group differences in self-reported intensities of topics selected. Moderate intensity topics were chosen specifically to minimize confounds between group status and topic intensity, and because of concerns about distress.

Next, participants were seated in a comfortable, sound-attenuated room with audiovisual monitoring to complete psychophysiological assessments. RSA and EDA were first collected during a 5-min resting baseline in which parents and adolescents were asked to sit quietly in the room together and look at their laps. Dyad members were informed that any movement or interaction would extend the 5-min resting period, which resulted in excellent compliance during the resting baseline. After the baseline, the RA reentered the room and read the following script:

> For the next 10 min I would like you to discuss a topic that you both rated as a frequent area of disagreement. I will tell you the topic and then I will exit the room to restart the recording equipment. I will knock on the wall when it is time for you to start the discussion and I will knock again in 10 min when the discussion is over. It is important for you to keep the conversation going for the full 10 min. Your discussion topic is [e.g., keeping the bedroom clean].

Measures

Psychophysiological assessments. Electrocardiographic (ECG) signals and electrodermal responding were obtained simultaneously from both participants using a BioPac MP100 system (Goleta, CA), sampled at 1 kHz. ECG electrodes were placed on participants' torsos using a standard spot electrode configuration (Qu et al., 1986). RSA was indexed by the high frequency component (>0.15 Hz) of the R-R time series using MindWare scoring software (MindWare Technologies, Ltd., Gahanna, OH). Given that 1 min is typically considered to be the minimum length necessary for spectral analysis of high frequency heart rate variability (Berntson et al., 1997), reactivity (i.e., change) scores were calculated for each min by subtracting baseline scores from discussion task scores. Electrodermal data were acquired with two standard 0.8-cm^2 Ag-AgCl electrodes attached to the thenar eminence of the nondominant hand with adhesive electrode washers and a 0.05 molar NaCl solution. Fluctuations exceeding 0.05 microseimens were considered nonspecific skin conductance responses. Change scores were also computed by subtracting baseline responses from each min of the discussion task.

Coding. The Family and Peer Process Code (FPPC; Stubbs, Crosby, Forgatch, & Capaldi, 1998) was used to score dyadic emotional behavior, moment to moment. Using this microanalytic behavioral coding system, raters assign a five-digit numerical code representing the speaker, content of utterance (next two numbers), listener, and affect. Codes change whenever there is a shift in speaker, listener, verbal content, or affect. The FPPC includes 25 content codes for verbal behavior and 6 affect codes (3 negative, 1 neutral, and 2 positive), which results in a possibility of 75 (25 × 3) combinations. Prior to coding tapes, two research assistants were trained on the FPPC using a multistep process. Coders received approximately 15 hr of training per week for 3 months to perform the following: (a) review the manual and become familiar with the codes, (b) code and discuss practice tapes, which were initially scored by the lead author, and (c) code three criterion tapes without assistance and discuss discrepancies. Once coders reached an acceptable level of reliability on practice tapes (see Crowell et al.,

2013), they scored three tapes per week including one shared tape, which was used to measure reliability, resolve discrepancies between coders, and prevent drift. Reliability was $\kappa = .76$ for content and $\kappa = .69$ for affect. Coders were also required to meet a minimum 10-key typing speed of 8,000 keystrokes per hour at 95% accuracy and were blind to group status and study hypotheses.

To examine observed behaviors on the same timescale as psychophysiology (e.g., 1 min epochs for stability of RSA measures), we followed a common empirically derived strategy for data reduction of moment-to-moment FPPC codes (see Crowell et al., 2013; Snyder et al., 1994). Each of the 75 (25 content × 3 affect) codes is collapsed into a single number on a 10-point scale ranging from 0, which includes highly positive and endearing behaviors, to 9, which includes highly aversive, angry, or violent behaviors. These codes were then averaged across each min, resulting in an average aversiveness score for both the mother and the adolescent for each of the 10 min.

Statistical method

MCCM is based on the logic that if one variable (A) precedes another variable (B), A will be found in the time delay reconstruction (TDR) of B (Takens, 1981). Thus, MCCM uses the TDR of B to build a predicted value of A (which we will call A′). The cross-correlation is then the correlation between the predicted value, A′, and A which asymptotes as more of the TDR is used to build predicted values. The nonparametric bootstrap extrapolates this logic to multiple time series simultaneously and tests for the difference between this cross-correlation and the observed bivariate correlation. In the present example of A preceding B and not B preceding A, when repeating the process of TDR on A and building B′, the cross-correlation of B′ and B is no different than the correlation of A and B. Through testing both directions, one is able to assess asymmetry in the AB relationship under the assumption that A and B form a complex nonlinear dynamic.

Analyses were conducted in R (R Core Team, 2015) using the multispatialCCM package (Clark, 2014). It requires several choices, each of which can influence results. First, we confirmed that the data were consistent with expectations for a nonlinear dynamic system. This involved examining the auto-prediction of how current values predict future values for mother and child separately. Using a nonparametric bootstrap from dewdrop regression in all cases, we observed an exponential loss in prediction further in time, consistent with chaotic systems. This does not prove that the time series conforms to a nonlinear dynamic, but it is a strong indicator of the possibility (Clark et al., 2015).

MCCM offers a means of graphically representing data dynamics through time as a midstep, which conforms to requirements and restrictions of TDR. TDR (and therefore MCCM) requires a choice of a specific time delay (often labeled τ) and an embedding. Embedding corresponds to the number of unique dimensions needed to graphically depict the data trajectory through time to observe its dynamic properties (e.g., moves to a point, cycles, complex repetitions). In TDR, each of these dimensions is represented by a lead of the data into the future. τ corresponds to how many steps into the future one uses for creating this depiction. A τ of 1 corresponds to the immediate next measure in the time series. Ultimately, data at a given point from a given individual in time are represented by a vector of data points using the individual's current value and values into the future. The number of values in the vector is the embedding and the number of time steps between each data point that makes up the vector is τ. For example, a time delay of 1 with an embedding of 3 captures the current value, the value one step into the future, and the value two steps into the future.

To choose the proper embedding dimension we used a prediction method within dewdrop regression whereby each time delay embedding point is used to predict the next, across all of the time series. This form of autocorrelation is calculated for a range of embedding dimensions. In theory, the proper embedding value is identified when autocorrelation is maximized. We chose to use the first maximum after an embedding of 1. There are several possible approaches to choosing the ideal time delay (e.g., Fraser & Swinney, 1986). Given the relatively short length of our time series, we chose a delay of 1 to maximize the data used.

To summarize, MCCM was conducted using a time delay of 1 and an embedding value derived from examining autocorrelation plots. In each circumstance, we assessed both directions of relations (e.g., mother′ from daughter and daughter′ from mother). We restricted analyses only to those hypothesized, and conducted MCCMs for each of the three groups separately (self-injuring, depressed, control).[3]

Results

Primary results

Table 1 contains correlations and standard deviations for correlations across families, adjusting for the multilevel structure of the data (estimated from multilevel models of standardized variables in one predictor relationships, a random effect on the relationship, and no intercepts). Correlations in Table 1 represent the baseline comparison for MCCM (MCCM is examining significance from

Table 1. Correlations (Above Diagonal) and Variances of Correlations Between Families (Below Diagonal) by Group

Group	Child RSA	Child EDA	Child behavior	Mother RSA	Mother EDA	Mother behavior
Control						
Child RSA	1	–0.029	–0.186	0.178	0.250	0.139
Child EDA	0.421**	1	0.058	–0.032	0.124	–0.190
Child behavior	0.495**	0.450**	1	0.041	–0.273	0.274
Mother RSA	0.231*	0.290**	0.417**	1	–0.242	0.010
Mother EDA	0.468*	0.616*	0.613**	0.873**	1	–0.105
Mother behavior	0.638*	1.220**	1.230**	0.710*	0.561*	1
Depressed						
Child RSA	1	0.026	0.148	–0.002	0.053	0.278*
Child EDA	0.316*	1	0.141	0.162	0.358**	0.071
Child behavior	0.232*	0.124	1	–0.045	0.075	0.199
Mother RSA	0.145	0.323*	0.457*	1	–0.021	–0.253
Mother EDA	0.276*	0.271*	0.726**	0.319	1	0.266
Mother behavior	0.162	0.630**	0.970**	1.660**	0.351*	1
Self-injuring						
Child RSA	1	0.067	0.012	–0.005	0.011	0.065
Child EDA	0.225*	1	0.034	0.219	0.133	0.204
Child behavior	0.619**	0.515**	1	0.110	–0.062	0.302
Mother RSA	0.469**	0.599**	0.489**	1	0.243	0.138
Mother EDA	0.187*	0.165*	0.250*	0.390*	1	–0.011
Mother behavior	0.529**	0.487**	0.850**	0.716**	0.351**	1

Note: EDA = electrodermal activity; RSA = respiratory sinus arrhythmia.
*$p \leq .05$. **$p \leq .01$.

equivalent zero-order correlations). MCCM generates a bootstrapped test to compare values from observed correlations in Table 1 with predicted values generated by the time delay reconstructed state space. In other words, MCCM is testing associations beyond observed correlations. Table 2 contains MCCM results, by respective groups, and tests directional associations between variables. We report all hypothesized directional comparisons from teen/mother behavior to teen/mother behavioral and physiological responses.

Findings indicate that during conflict interactions, behaviors of mothers exerted a direct driving relationship on adolescent behavior within the self-injuring ($p < .01$) and depressed ($p < .05$) groups. However, these adolescents did not exhibit a driving effect on their mothers' behaviors (both ps $\geq .39$). In contrast and as hypothesized, control dyads showed no effect on one another's behaviors in either direction (both ps $\geq .40$). Thus driving relations between mothers' behaviors and their daughters' behaviors were observed for depressed and SII dyads, but not for control dyads. We tested whether these effects could be attributed solely to mean differences by restricting the embedding value to 1. An embedding of 1 includes only means and same-time correlations in the model, and yields the equivalent of a nonlinear correlation, eliminating effects of change. This model produced all nonsignificant parameter estimates, confirming that observed effects were not due to mean-level differences in mother aversiveness.

Mothers' behaviors also exerted driving effects on adolescents' EDA and RSA for the SII group (ps $< .05$). Thus, mother behaviors affected physiological activity of self-injuring adolescents, but not depressed or typical adolescents. As hypothesized, all other comparisons from adolescent behaviors to mothers' behavior, EDA, and RSA were nonsignificant. Thus, mothers' behaviors had a unidirectional effect on SII adolescents' behavioral and physiological responses during conflict, which was not the case for depressed or typical control adolescents.

Results are presented visually in Figure 1, which plots MCCM correlation values on the *y*-axis, and time series length (L), which is used to build predicted values in the MCCM procedure, on the *x*-axis. Each group has their own line, with confidence intervals derived from dewdrop regression. The two rows represent the group comparisons of adolescents' behavior driving the mothers' behavior (top panel) and vice versa (bottom panel). A strength of MCCM is that it can be used on short, noisy

Table 2. MCCM Analyses Showing Directional Effects From Mother to Adolescent or Adolescent to Mother Behavior or Physiology

Group	Variable	*p* value	Embedding dimension	Proportion of replication of *p*
	Behavior outcomes			
Control	TeenBeh → MomBeh	.41[a]	3	1.00
	MomBeh → TeenBeh	.54[a]	2	.86
Depressed	TeenBeh → MomBeh	.45[a]	3	.90
	MomBeh → TeenBeh	.04*	5	.17
Self-injuring	TeenBeh → MomBeh	.37[a]	2	.72
	MomBeh → TeenBeh	.00**	6	.42
	Electrodermal outcomes			
Control	TeenBeh → MomEDA	.43[a]	3	.80
	MomBeh → TeenEDA	.38[a]	2	.97
Depressed	TeenBeh → MomEDA	.36[a]	3	.76
	MomBeh → TeenEDA	.45	5	.86
Self-injuring	TeenBeh → MomEDA	.23[a]	2	1.00
	MomBeh → TeenEDA	.01**	6	.41
	Respiratory sinus arrhythmia outcomes			
Control	TeenBeh → MomRSA	.15[a]	3	.36
	MomBeh → TeenRSA	.16[a]	2	.83
Depressed	TeenBeh → MomRSA	.85[a]	3	.98
	MomBeh → TeenRSA	.18	5	.50
Self-injuring	TeenBeh → MomRSA	.08[a]	2	.79
	MomBeh → TeenRSA	.01**	6	.84

Note: Beh = observed behavior during conflict; EDA = electrodermal activity; Mom = mother; RSA = respiratory sinus arrhythmia; Teen = adolescent daughter.
[a]Hypothesized nonsignificant result.
*$p \leq .05$. **$p \leq .01$.

time series, although one of its assumptions is that larger correlation coefficients should be observed as L increases. Furthermore, as L increases, correlations should asymptote. This is most evident in the mother behavior drives adolescent behavior graph. When examining confidence intervals in the adolescent drives mother behavior graph (top), almost all of the lines overlap. However, in the mother drives adolescent behavior (bottom), correlation coefficients are higher for the SII and depressed groups than the control group at higher lengths. Plots of other comparisons (second and third columns) show similar patterns.

Sensitivity to time delay reconstruction parameters

Because MCCM is a relatively new method, it is important to consider the importance of decision processes for selecting time delays and embedding. As noted earlier, recommendations exist for choosing these, but little is known about effects of accepting vs. ignoring these. We therefore "*p*-hacked" the procedure by generating alternative solutions that fully ignore recommended decisions. Given our time series lengths, there were 3 reasonable time delays (τ) possible,[4] 7 different embedding dimensions for the teen, and 7 possible embedding dimensions for the mother (from 2 to 8). This yields a total of $3 \times 7 \times 7 = 147$ potential combinations for each condition. In the last column of Table 2, we report the proportion of the 147 combinations, which replicated our reported finding (rejection or nonrejection of the null hypothesis at $p = .05$). Although some parameters showed consistent results, many were contingent on time delays and embedding decisions. We discuss this in terms of *p*-hacking and the need for following established procedures for time delay and embedding in studies using MCCM.

Discussion

We hypothesized that self-injuring and depressed adolescents would be more reactive following their mothers' behaviors than typical adolescents and that self-injuring adolescents might show the highest degree of sensitivity. Based on BSCT, we also hypothesized that adolescents in the clinical groups (SII, depressed) would be more reactive than their mothers. We examined these hypotheses using observational methods (behavioral coding and

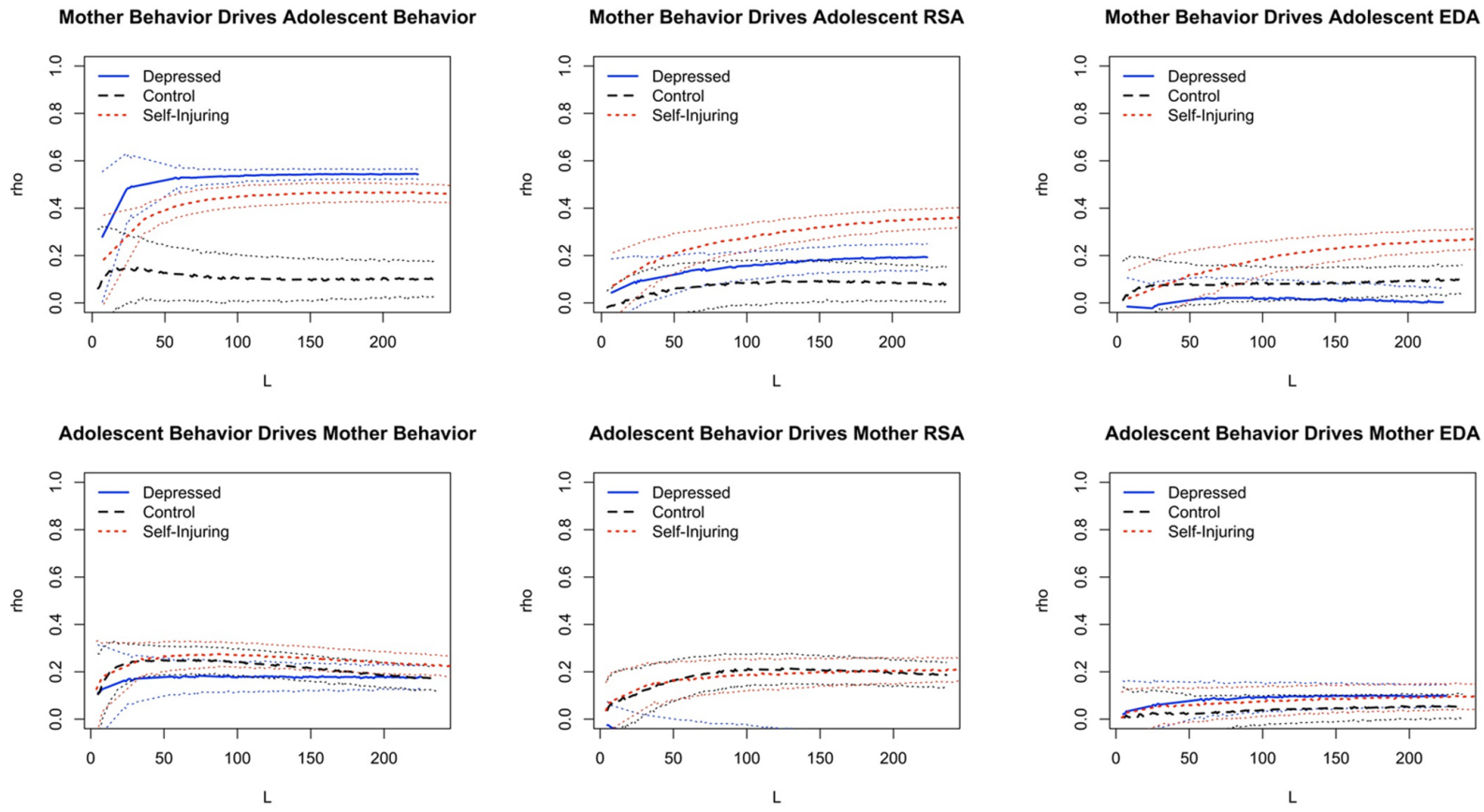

Fig. 1. MCCM graphs showing results from adolescent behaviors driving mother behaviors/physiology (top) and mother behaviors driving adolescent behaviors/physiology (bottom) with separate lines and confidence intervals for each group. The second row shows a significant driving effect of mother behavior on adolescent behavior for the depressed and self-injuring groups and on adolescent physiology for the self-injuring group.

psychophysiology) during a 10-min mother-daughter conflict discussion. To test our hypotheses we used a novel dynamical systems approach designed to test driving associations in time series data. Given the complexity of social systems and the nature of our data (i.e., a 10-min snapshot) we have used the more agnostic term *driven* rather than *causal* to describe observed associations in our data. As described in Note 1, myriad distal influences can affect proximal dyadic interaction patterns.

Consistent with biological sensitivity theories, mother behaviors had a driving effect on adolescents' behaviors and their psychophysiological responses for those in the SII group. In contrast, driving relations were not found from behaviors of SII adolescents to either behaviors of or psychophysiological responses of mothers. Thus, SII adolescents responded to their mothers' verbal behaviors across multiple levels of analysis, including both sympathetic and PNS measures. They therefore exhibited a "full-system" response during the conflict task. This is consistent with theories that self-injuring adolescents are particularly reactive and emotionally sensitive (Crowell et al., 2009)—a finding that should be replicated and extended using other statistical methods and clinical comparison groups.

As we also hypothesized, a driving relation was observed from mothers' behaviors to adolescents' behaviors for the depressed group whereas a converse effect was not observed. However, mother behaviors did not evoke psychophysiological responses from their depressed daughters. There are a number of possible explanations for this finding. One possibility is that mothers of SII adolescents behaved differently than mothers of depressed teens in ways that were not tested in this article (e.g., were louder) or that other unmeasured differences in family functioning drove patterns of results. It is also possible that depressed adolescents are less sensitive and less reactive than those who self-injure. Both of these theories are consistent with etiological models of SII (e.g., Crowell & Kaufman, 2016) and merit exploration in future research. Finally, no driving effects were observed between mothers and daughters in the typical control group. This is consistent with our hypothesis that control dyads would be less reactive during the conflict. Less reactivity among control dyads may therefore represent a valid nonresponse.

Clinically, these findings may have implications for understanding and intervening with self-injuring teens. Interpersonal conflict is a well-established precursor to adolescent suicide (Lowenstein, 2005; Pineda & Dadds, 2013), and our data suggest that reducing conflict or addressing adolescents' behavioral and physiological responses to conflict may be beneficial for treatment. Based on our findings, family systems-based approaches may be especially important for self-injuring adolescents. If replicated in future research, less costly individual-level treatments could continue as a first-line treatment for depressed adolescents whereas self-injuring youth may require interventions that target family conflict at the outset.

This study also makes a novel methodological contribution to psychological research. Dynamical systems approaches are still relatively underutilized in psychological studies of social systems, in spite of recent calls to do so (Giuliano et al., 2015). To our knowledge, MCCM has not yet been used in any other psychological study, making this an important contribution to the broader literature. Given this, it is important that other scholars replicate our findings in both clinical and nonclinical samples. For example, differences in reactivity are well established in other forms of psychopathology (e.g., aggression, conduct disorder; see Beauchaine & Zalewski, 2016). In addition, longitudinal data using MCCM before and after psychopathology emerges could provide for more sophisticated tests of etiological theories of SII and depression.

MCCM makes vastly different assumptions than models that are currently common in psychology. Granger (1969) proposed several criteria for assessing causality, including association, temporal precedence, and nonspuriousness. However, Granger noted that this model may not apply to situations such as nonlinear dynamic systems. In these circumstances, associations and temporal precedence can break down. As an example, deterministic chaos is known to have a drop-off in future prediction that requires exponentially more data to incrementally increase prediction (McSharry, 2005). Under such circumstances, classic modeling may be limited, and techniques such as MCCM may be more effective. This suggests clear utility of MCCM in psychology, given the complexity of phenomena we study.

Sugihara et al.'s (2012) work on CCM is based on the assumption that if one variable drives another in a nonlinear dynamic, we should be able to "find" the driving variable in the dynamics of the resultant. In essence, the driving relationship appears to reverse and is apparent in the predictive nature from the higher-order dynamic pattern rather than a simple association. Thus, CCM is a function of the predicted value of the driver generated from the TDR of the resultant, in correlation form. Its improvement over the same time correlation is consistent with a driver-driven relationship. The fact that our CCM findings (Table 2) reveal associations among variables that are not apparent in correlations (Table 1) is consistent with driver-driven relations in this sample. These would have gone undetected using typical linear analyses. Our choice to call these driving rather than causal relations follows recognition that behavioral and neurobiological

systems are multidetermined and multidirectional, with both proximal and distal causes.

Clark and colleagues' (2015) expansion of CCM to MCCM by integrating dewdrop regression, a form of nonparametric bootstrap, allows for application to time series of varying lengths, all believed to reside in the same dynamic system. In Sugihara et al.'s (2012) original work, the technique was applied to fish populations with long time series. Expansion to MCCM suggests that the technique can detect driver-driven associations with time series as short as five sequences, even in contexts of considerable noise and observation error (Clark et al., 2015). However, recent work shows that CCM, without a multispatial component, may produce inaccurate results if variables are strongly coupled and include noise (Mønster, Fusaroli, Tylén, Roepstorff, & Sherson, in press). The observed base cross-correlations in the present study were all relatively low (see Table 1). Thus, results were not affected negatively by strong coupling. Given due caution, MCCM is useful to psychology, where we tend to capitalize on short panel assessments from multiple individuals rather than single long time series designs.

Given the lack of prior psychological research using MCCM, we conducted additional tests to explore the extent to which our findings were contingent on rules used in TDR. In our initial analyses, we followed published guidelines for determining the appropriate embedding dimension and time delay. As we noted, however, this step involves some degree of choice, and lack of familiarity with the procedure and its sensitivity to qualities known to affect TDR generates a scenario ideal for "*p*-hacking." Therefore, we conducted a series of analyses in which we varied embedding dimension and time delay to reflect every possible choice for the data within a reasonable range of parameters. It is important that most of the possible solutions we generated violate criteria for producing reasonable TDR (Kantz & Schreiber, 2003) and therefore should also be problematic in MCCM. As presented in Table 2, the robustness of our findings varied. Thus, some findings were more invariant of analytic decisions than others. For example, in the SII group, the finding that mother behavior drove adolescent RSA reactivity was replicated more than 80% of the time at $p < .05$. This finding was therefore robust to analytic decisions. In contrast, the association between depressed adolescent behavior and mother behavior replicated only 17% of the time. For obvious mathematical reasons, nonsignificant findings were replicated more often (range = .36–1.00).

To better understand the importance of choosing time delay and embedding dimension, it is worth considering their effects. Choice of time delay is designed to distinguish autocorrelations due to temporal proximity of measurement from unfolding nonlinear dynamics. When not enough time has passed for values to change, an inherent increase in autocorrelation is observed, which can mask underlying dynamics. How this translates into problems with MCCM is less clear, as temporal autocorrelation would need to occur in both variables. However, we believe this is less of an issue in our study because our level of measurement (min) allowed for considerable variation within families across measures. Increasing time delay also reduces available data. At a higher τ, fewer data points have a time delay within the data window and therefore generate missing data. Thus, τs higher than we selected would lower statistical power.

The embedding dimension is designed to differentiate qualitatively different structures within data through time. When the embedding dimension is too low, we are unable to distinguish different types of temporal trajectories from one another. This affects MCCM directly, as predicted values used in calculations are contingent on embedding. Too high of an embedding dimension can also be problematic, though less so, as it sensitizes the analysis to noise in the system (Kantz & Schreiber, 2003). Notably, some techniques based on TDR intentionally pick too high of an embedding dimension because the cost of doing so is less than the cost of going too low (Webber & Zbilut, 2005). Higher embedding dimensions also generate a greater amount of missing data, as each embedding dimension requires going out τ steps in front of the current data point to represent that point. Thus, when τ is high and the embedding dimension is high, one quickly loses available data and, in turn, power. In all, our results suggest that careful decision making should be applied prior to examining MCCM results, and future publications should include important details describing how decisions were made, as done here.

Finally, it is important to acknowledge clinical and methodological limitations of this study. First, this is a relatively small sample of ~25 dyads/group. Statistically, limitations of small sample sizes are somewhat mitigated by the use of multiple measures within individuals (i.e., because analyses are conducted on observations rather than individuals, each group had ≥240 observations). Nonetheless, small samples can yield spurious findings, and limit generalizability, so results should be interpreted as preliminary. Second, the sample consisted entirely of girls and mothers. Future research should extend these findings to boys and fathers. Third, conflict topics of moderate intensity were chosen purposefully. Although this provides methodological and ethical advantages, findings may not generalize to more intense conflicts that often precede self-injury. Fourth, the method we used is designed to test directional associations between dyad members, but not positive or negative directions of effect within measures. Thus, we did not examine effects such

as whether increases in mother aversiveness predicted increases or decreases in adolescent RSA. Such associations have been studied with other analytic approaches and were not the focus of this study. Finally, some of our findings were less robust to our analytic choices. Although we followed a set of established rules for all analyses, making different decisions would have affected the pattern of results. Our objective in presenting these probabilities was to reveal the number of conditions in which *p*-hacking would yield the same result, a problem that appears to be rampant in the field (Nuzzo, 2014). The broader goal of conducting such analyses is to increase transparency and help reveal which results may be more difficult to replicate.

In conclusion, SII and depression are significant clinical problems that affect many adolescents. Family environments are important contexts for these youth, and it is important that family conflict is an established precursor to self-injury and suicide. Most research has examined family risk for psychopathology using self-report measures of parenting rather than observational methods. Moreover, few studies have used dynamical systems approaches to understand minute-to-minute dynamics within vulnerable families. Future research should extend these findings to better understand whether SII and depression are characterized by emotional and biological sensitivity to context across other measures and, if so, how parents could interact more effectively with depressed and self-injuring adolescents. Researchers should also extend MCCM to other clinical diagnoses and populations, with the goal of better understanding driving effects within time series data. Ultimately, such approaches have great potential to clarify the nature of complex psychological problems and elucidate key targets for intervention and prevention.

Author Contributions

The study was designed by S. E. Crowell and T. P. Beauchaine. Data were collected, processed, and entered by S. E. Crowell and M. Yaptangco. Statistical analyses were conducted by J. E. Butner, T. J. Wiltshire, and A. K. Munion. S. E. Crowell took the lead on writing this manuscript, and all authors contributed to additional writing, editing, and revising.

Declaration of Conflicting Interests

The authors declared that they had no conflicts of interest with respect to their authorship or the publication of this article.

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Notes

1. The history of psychological science is replete with examples of new statistical methods being touted as resolvers of causal mechanisms in nonexperimental data. Many readers likely recall, for example, structural equation modeling initially being referred to as "causal modeling." It is important to note that no statistical method can be used to unambiguously identify casual mechanisms of any phenomenon without (a) random assignment to experimental and control conditions, (b) manipulation of the primary independent variable, and (c) control of extraneous influences. Data presented herein are nonexperimental—without random assignment or control of extraneous influences. Thus, causality cannot be inferred. In fact, it is likely that unmeasured distal variables (e.g., operant reinforcement histories, personality traits, histories of abuse) influenced observed patterns of interaction. Nevertheless, MCCM does identify proximal patterns of directionality in time series data. From this point forward, we refer to "drivers" *within these interactions* to avoid confusion regarding proximal versus distal causes. The term *drivers* is consistent with dynamical systems theory and is intended, here, in the most agnostic sense of the word.

2. Selective serotonin reuptake inhibitors (SSRIs) were allowed. SSRIs are used routinely for treatment of adolescent depression, so excluding participants based on their use would have created a biased sample. It is important that SSRIs exert more modest effects on cardiovascular function than other types of antidepressants (see Kemp et al., 2010; Udupa et al., 2011).

3. Given that MCCM is new to the social sciences, interested readers are directed to https://sites.google.com/site/dynamicsystem-satutah/home/cross-convergent-mapping for further discussion.

4. In our simulations, we used the same τ for both variables.

References

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text revision). Washington, DC: Author.

Armsden, G. C., McCauley, E., Greenberg, M. T., Burke, P. M., & Mitchell, J. R. (1990). Parent and peer attachment in early adolescent depression. *Journal of Abnormal Child Psychology*, *18*, 683–697. doi:10.1007/BF01342754

Beauchaine, T. P. (2001). Vagal tone, development, and Gray's motivational theory: Toward an integrated model of autonomic nervous system functioning in psychopathology. *Development and Psychopathology*, *13*, 183–214. doi:10.1017/S0954579401002012

Beauchaine, T. P., & Thayer, J. F. (2015). Heart rate variability as a transdiagnostic biomarker of psychopathology. *International Journal of Psychophysiology*, *98*, 338–350. doi:10.1016/j.ijpsycho.2015.08.004

Beauchaine, T. P., & Zalewski, M. (2016). Physiological and developmental mechanisms of emotional lability in coercive relationships. In T. J. Dishion & J. J. Snyder (Eds.), *The Oxford handbook of coercive relationship dynamics* (pp. 39–52). New York, NY: Oxford University Press.

Belsky, J., & Pluess, M. (2009). Beyond diathesis stress: Differential susceptibility to environmental influences. *Psychological Bulletin*, *135*, 885–908. doi:10.1037/a0017376

Berntson, G. G., Thomas Bigger, J., Eckberg, D. L., Grossman, P., Kaufmann, P. G., Malik, M., . . . Der Molen, M. W. (1997). Heart rate variability: Origins, methods, and interpretive caveats. *Psychophysiology, 34,* 623-648. doi:10.1111/j.1469-8986.1997.tb02140.x

Betts, J., Gullone, E., & Allen, J. S. (2009). An examination of emotion regulation, temperament, and parenting style as potential predictors of adolescent depression risk status: A correlational study. *British Journal of Developmental Psychology*, *27*, 473–485. doi:10.1348/026151008X314900

Boyce, W. T., & Ellis, B. J. (2005). Biological sensitivity to context: I. An evolutionary-developmental theory of the origins and functions of stress reactivity. *Development and Psychopathology*, *17*, 271–301. doi:10.1017/S0954579405050145

Brenner, S. L., Beauchaine, T. P., & Sylvers, P. D. (2005). A comparison of psychophysiological and self-report measures of BAS and BIS activation. *Psychophysiology*, *42*, 108–115. doi:10.1111/j.1469-8986.2005.00261.x

Bronfenbrenner, U. (1979). Contexts of child rearing: Problems and prospects. *American Psychologist*, *34*, 844–850. doi:10.1037/0003-066X.34.10.844

Caspi, A., Hariri, A. R., Holmes, A., Uher, R., & Moffitt, T. E. (2010). Genetic sensitivity to the environment: The case of the serotonin transporter gene and its implications for studying complex diseases and traits. *American Journal of Psychiatry*, *167*, 509–527. doi:10.1176/appi.ajp.2010.09101452

Cicchetti, D. (2016). Socioemotional, personality, and biological development: Illustrations from a multilevel developmental psychopathology perspective on child maltreatment. *Annual Review of Psychology*, *67*, 187–211. doi:10.1146/annurev-psych-122414-033259

Cicchetti, D., Ackerman, B. P., & Izard, C. E. (1995). Emotions and emotion regulation in developmental psychopathology. *Development and Psychopathology*, *7*, 1–10. doi:10.1017/S0954579400006301

Clark, A. (2014). *Multispatial CCM: Multispatial convergent cross mapping* (R package version 1.0). Retrieved from http://CRAN.R-project.org/package=multispatialCCM

Clark, A. T., Ye, H., Isbell, F., Deyle, E. R., Cowles, J., Tilman, G. D., & Sugihara, G. (2015). Spatial convergent cross mapping to detect causal relationships from short time series. *Ecology*, *96*, 1174–1181. doi:10.1890/14-1479.1

Crowell, S. E. (2016). Biting the hand that feeds: Current opinion on the interpersonal causes, correlates, and consequences of borderline personality disorder. *F1000Research, 5*. doi:10.12688/f1000research.9392.1

Crowell, S. E., Baucom, B. R., McCauley, E., Potapova, N. V., Fitelson, M., Barth, H., . . . Beauchaine, T. P. (2013). Mechanisms of contextual risk for adolescent self-injury: Invalidation and conflict escalation in mother-child interactions. *Journal of Clinical Child and Adolescent Psychology*, *42*, 467–480. doi:10.1080/15374416.2013.785360

Crowell, S. E., Baucom, B. R., Yaptangco, M., Bride, D., Hsiao, R., McCauley, E., & Beauchaine, T. P. (2014). Emotion dysregulation and dyadic conflict in depressed and typical adolescents: Evaluating concordance across psychophysiological and observational measures. *Biological Psychology*, *98*, 50–58. doi:10.1016/j.biopsycho.2014.02.009

Crowell, S., Beauchaine, T., Hsiao, R., Vasilev, C., Yaptangco, M., Linehan, M., & McCauley, E. (2012). Differentiating adolescent self-injury from adolescent depression: Possible implications for borderline personality development. *Journal of Abnormal Child Psychology*, *40*, 45–57. doi:10.1007/s10802-011-9578-3

Crowell, S. E., Beauchaine, T. P., & Linehan, M. M. (2009). A biosocial developmental model of borderline personality: Elaborating and extending Linehan's theory. *Psychological Bulletin*, *135*, 495–510. doi:10.1037/a0015616

Crowell, S. E., Beauchaine, T. P., McCauley, E., Smith, C., Vasilev, C., & Stevens, A. L. (2008). Parent-child interactions, peripheral serotonin, and intentional self-injury in adolescents. *Journal of Consulting and Clinical Psychology*, *76*, 15–21. doi:10.1037/0022-006X.76.1.15

Crowell, S. E., & Kaufman, E. A. (2016). Development of self-inflicted injury: Comorbidities and continuities with borderline and antisocial personality traits. *Development and Psychopathology, 28,* 1071–1088. doi:10.1017/S0954579416000705

Del Giudice, M., Ellis, B. J., & Shirtcliff, E. A. (2011). The adaptive calibration model of stress responsivity. *Neuroscience and Biobehavioral Reviews, 35,* 1562–1592. doi:10.1016/j.neubiorev.2010.11.007

Fraser, A. M., & Swinney, H. L. (1986). Independent coordinates for strange attractors from mutual information. *Physical Review A*, *33*, 1134–1140. doi:10.1103/PhysRevA.33.1134

Garber, J., Robinson, N. S., & Valentiner, D. (1997). The relation between parenting and adolescent depression: Self-worth as a mediator. *Journal of Adolescent Research*, *12*, 12–33. doi:10.1177/0743554897121003

Giuliano, R. J., Skowron, E. A., & Berkman, E. T. (2015). Growth models of dyadic synchrony and mother-child vagal tone in the context of parenting at-risk. *Biological Psychology*, *105*, 29–36. doi:10.1016/j.biopsycho.2014.12.009

Granger, C. W. J. (1969). Investigating causal relations by econometric models and cross-spectral methods. *Econometrica*, *37*, 424–438. doi.org/10.2307/1912791

Hinshaw, S. P. (2015). Developmental psychopathology, ontogenic process models, gene-environment interplay, and brain development: An emerging synthesis. *Journal of Abnormal Psychology*, *124*, 771–775. doi:10.1037/abn0000110

Kantz, H., & Schreiber, T. (2003). *Nonlinear time series analysis*. New York, NY: Cambridge University Press.

Kemp, A. H., Quintana, D. S., Gray, M. A., Felmingham, K. L., Brown, K., & Gatt, J. M. (2010). Impact of depression and antidepressant treatment on heart rate variability: A review and meta-analysis. *Biological Psychiatry*, *67*, 1067–1074. doi:10.1016/j.biopsych.2009.12.012

Lewis, G., Collishaw, S., Thapar, A., & Harold, G. T. (2014). Parent-child hostility and child and adolescent depression symptoms: The direction of effects, role of genetic factors and gender. *European Child and Adolescent Psychiatry*, *23*, 317–327. doi:10.1007/s00787-013-0460-4

Linehan, M. M. (1993). *Cognitive-behavioral treatment of borderline personality disorder*. New York, NY: Guilford.

Lorber, F. (2004). Psychophysiology of aggression, psychopathy, and conduct problems: A meta-analysis. *Psychological Bulletin*, *130*, 531–552. doi:10.1037/0033-2909.130.4.531

Lowenstein, L. F. (2005). Youths who intentionally practise self-harm: Review of the recent research 2001–2004. *International Journal of Adolescent Medicine and Health*, *7*, 225–230. doi:10.1515/IJAMH.2005.17.3.225

Lytton, H. (1990). Child and parent effects in boys' conduct disorder: A reinterpretation. *Developmental Psychology*, *26*, 683–697. doi:10.1037/0012-1649.26.5.683

Marsh, P., Beauchaine, T. P., & Williams, B. (2008). Dissociation of sad facial expressions and autonomic nervous system responding in boys with disruptive behavior disorders. *Psychophysiology*, *45*, 100–110. doi:10.1111/j.1469-8986.2007.00603.x

McSharry, P. E. (2005). The danger of wishing for chaos. *Nonlinear Dynamics in Psychological Life Sciences*, *9*, 375–397.

Miller, A. L., Rathus, J. H., & Linehan, M. M. (2006). *Dialectical behavior therapy with suicidal adolescents*. New York, NY: Guilford Press.

Mønster, D., Fusaroli, R., Tylén, K., Roepstorff, A., & Sherson, J. F. (in press). Causal inference from noisy time-series data—Testing the convergent cross-mapping algorithm in the presence of noise and external influence. *Future Generation Computer Systems*. doi:10.1016/j.future.2016.12.009

Nuzzo, R. (2014). Scientific method: Statistical errors. *Nature*, *506*, 150–152. doi:10.1038/506150a

Obradović, J., Bush, N. R., Stamperdahl, J., Adler, N. E., & Boyce, W. T. (2010). Biological sensitivity to context: The interactive effects of stress reactivity and family adversity on socioemotional behavior and school readiness. *Child Development*, *81*, 270–289. doi:10.1111/j.1467-8624.2009.01394.x

Olino, T. M., McMakin, D. L., Nicely, T. A., Forbes, E. E., Dahl, R. E., & Silk, J. S. (2015). Maternal depression, parenting, and youth depressive symptoms: Mediation and moderation in a short-term longitudinal study. *Journal of Clinical Child and Adolescent Psychology*. Advance online publication. doi:10.1080/15374416.2014.971456

Pineda, J., & Dadds, M. R. (2013). Family intervention for adolescents with suicidal behavior: A randomized controlled trial and mediation analysis. *Journal of the American Academy of Child and Adolescent Psychiatry*, *52*, 851–862. doi:10.1016/j.jaac.2013.05.015

Porges, S. W. (2001). The polyvagal theory: Phylogenetic substrates of a social nervous system. *International Journal of Psychophysiology*, *42*, 123–146. doi:10.1016/S0031-9384(03)00156-2

Prinz, R. J., Foster, S., Kent, R. N., & O'Leary, K. D. (1979). Multivariate assessment of conflict in distressed and nondistressed mother-adolescent dyads. *Journal of Applied Behavior Analysis, 12,* 691–700.

R Core Team. (2015). *R: A language and environment for statistical computing*. Vienna, Austria: R Foundation for Statistical Computing. Retrieved from https://www.R-project.org/

Richardson, M. J., Dale, R., & Marsh, K. L. (2014). Complex dynamical systems in social and personality psychology. In H. T. Reis & C. M. Judd (Eds.), *Handbook of research methods in social and personality psychology* (pp. 253–282). New York, NY: Cambridge University Press.

Sheeber, L., Hops, H., Alpert, A., Davis, B., & Andrews, J. (1997). Family support and conflict: Prospective relations to adolescent depression. *Journal of Abnormal Child Psychology*, *25*, 333–344.

Snyder, J., Edwards, P., McGraw, K., & Kilgore, K. (1994). Escalation and reinforcement in mother-child conflict: Social processes associated with the development of physical aggression. *Development and Psychopathology*, *6*, 305–321. doi:10.1017/S0954579400004600

Snyder, J., Schrepferman, L., & St Peter, C. (1997). Origins of antisocial behavior. Negative reinforcement and affect dysregulation of behavior as socialization mechanisms in family interaction. *Behavior Modification*, *21*, 187–215. doi:10.1177/01454455970212004

Stubbs, J., Crosby, L., Forgatch, M. S., & Capaldi, D. M. (1998). *Family and peer process code: A synthesis of three Oregon Social Learning Center behavior codes* (Training manual.). Eugene, OR: Oregon Social Learning Center.

Sugihara, G., May, R., Ye, H., Hsieh, C. H., Deyle, E., Fogarty, M., & Munch, S. (2012). Detecting causality in complex ecosystems. S*cience*, *338*, 496–500. doi:10.1126/science.1227079

Takens, F. (1981). Detecting strange attractors in turbulence. In D. Rand & L.-S. Young (Eds.), *Dynamical systems and turbulence, Warwick 1980* (pp. 366–381). Berlin, Germany: Springer.

Udupa, K., Thirthalli, J., Sathyaprabha, T. N., Kishore, K. R., Raju, T. R., & Gangadhar, B. N. (2011). Differential actions of antidepressant treatments on cardiac autonomic alterations in depression: A prospective comparison. *Asian Journal of Psychiatry*, *4*, 100–106. doi:10.1016=j.ajp.2011.02.006

Webber, C. L., Jr., & Zbilut, J. P. (2005). Recurrence quantification analysis of nonlinear dynamical systems. In M. A. Riley & G. C. Van Orden (Eds.), *Tutorials in contemporary nonlinear methods for the behavioral sciences* (pp. 26–94). Arlington, VA: National Science Foundation.

Yap, M. H., Whittle, S., Yücel, M., Sheeber, L., Pantelis, C., Simmons, J. G., & Allen, N. B. (2008). Interaction of parenting experiences and brain structure in the prediction of depressive symptoms in adolescents. *Archives of General Psychiatry*, *65*, 1377–1385. doi:10.1001/archpsyc.65.12.1377