J Autism Dev Disord (2015) 45:4140–4146
DOI 10.1007/s10803-015-2528-0

BRIEF REPORT

Brief Report: A Pilot Study of Parent–Child Biobehavioral Synchrony in Autism Spectrum Disorder

Jason K. Baker[1] · Rachel M. Fenning[1] · Mariann A. Howland[1] · Brian R. Baucom[2] · Jacquelyn Moffitt[1] · Stephen A. Erath[3]

Published online: 17 July 2015

Abstract The theory of biobehavioral synchrony proposes that the predictive power of parent–child attunement likely lies in the manner with which behaviors are aligned with relevant biological processes. Symptoms of autism spectrum disorder (ASD) may challenge the formation of behavioral and physiological synchrony, but maintenance of such parent–child attunement could prove beneficial. The present study is the first to examine parent–child physiological synchrony in ASD. Parent and child electrodermal activity (EDA) was measured continuously during naturalistic free play. Parent–child EDA synchrony (positive covariation) was positively correlated with observed parent–child emotional attunement. Hierarchical linear modeling revealed that child ASD symptoms moderated the association between parent EDA and child EDA, such that EDA synchrony was stronger for children with lower ASD symptom levels.

Keywords Autism spectrum disorder · Parent–child interaction · Synchrony · Electrodermal activity · Psychophysiology

Introduction

Certain historical perspectives regarding parents' ability to promote development in children with autism spectrum disorder (ASD) have highlighted the importance of parent–child synchronous exchange (Greenspan and Wieder 1999). To date, treatments associated with this approach have generally lacked the rigorous evidence of comprehensive child benefit demonstrated by behavioral interventions, which deemphasize the importance of synchrony in favor of a strong instructional agenda (Howlin et al. 2009; Lovaas 1987). However, interest in parent–child synchrony in ASD research has surged within the past decade, in large part due to two concurrent but distinct lines of inquiry. Family and language investigations have reported that certain forms of sensitive parenting predict language growth in children with or developing ASD (Baker et al. 2010; Siller and Sigman 2008), while developmental theory and intervention studies have begun to highlight the importance of parent–child engagement in the prevention and remediation of ASD symptoms (Dawson 2008; Dawson et al. 2010; Kasari et al. 2010).

Feldman (2012) has recently proposed the theory of *biobehavioral synchrony*, wherein it is argued that the marked predictive power of early parent–child synchrony to important cognitive and social-emotional outcomes in children with neurotypical development likely lies in the manner with which this behavioral synchrony is aligned with relevant biological processes in each interactive partner. Although considered particularly important in infancy, these processes are active throughout childhood (see Cole et al. 2004), and into adulthood (Schneiderman et al. 2014). Biobehavioral synchrony can be investigated in part through the examination of covariation in psychophysiological arousal levels between interactive

Portions of this manuscript were presented at the 2014 Gatlinburg Conference on Research and Theory in Intellectual and Developmental Disabilities.

✉ Jason K. Baker
jbaker@fullerton.edu

[1] Department of Child and Adolescent Studies, Center for Autism, California State University, Fullerton, 800 N. State College Blvd., Fullerton, CA 92831, USA

[2] University of Utah, Salt Lake City, UT, USA

[3] Auburn University, Auburn, AL, USA

partners (Feldman 2012). Indeed, studies have shown that the strength of the positive association between parents' and infants' heart rates is higher when certain behaviors representative of synchrony are observed (Feldman et al. 2011) and that parent–child dyads with maltreating mothers exhibit weaker positive covariation in heart rate than those in which maltreatment is absent (Creaven et al. 2014).

Children with ASD exhibit significant social, affective, and behavioral difficulties that may challenge the formation of biological synchrony, but maintenance of such physiological attunement might promote the development of these children (Feldman 2012). To our knowledge, no study has examined caregiver–child biological synchrony in ASD. In light of evidence that increasing dyadic (behavioral) engagement in children at-risk and/or diagnosed with ASD may be key to optimizing developmental outcomes (Baker et al. 2010; Dawson 2008; Kasari et al. 2010; Siller and Sigman 2008), this would seem to be an especially important area of investigation.

The current pilot study examined covariation in electrodermal activity (EDA), a measure of sympathetic nervous system arousal, between parents and their children with ASD during a naturalistic free play interaction. Traditional EDA measurement has utilized fairly intrusive equipment and has required little movement on the part of the child (Fowles 2008), leading to laboratory paradigms with limited ecological validity and an over-representation of children with lower ASD symptom levels in relevant studies (e.g., Kylliäinen and Hietanen 2006; Schoen et al. 2008). In the present investigation, we utilized new wireless wrist sensors that permitted complete mobility, and enabled the inclusion of children with substantial variation in developmental functioning and autism symptomatology.

Consistent with current theory and evidence suggesting that ASD symptoms may challenge the development of behavioral synchrony, and that such synchrony may be key to reducing these symptoms, we predicted that stronger positive parent–child EDA covariation would be associated with lower ASD symptom levels. In addition to our primary interest in physiological covariation and ASD symptoms, we also investigated the degree to which EDA covariation mapped on to the similar, but not identical, behavioral construct of observed *affective mutuality* (NICHD Early Child Care Research Network 2003). Affective mutuality refers to the degree of observed emotional attunement, comfort, intimacy, and positive responsiveness observed during parent–child interaction (see McElwain et al. 2008, for a detailed description). Convergence of these behavioral and biological measures would provide further support for the notion of biobehavioral synchrony. In addition to examining covariation in EDA within each dyad over time, we also considered the strength of covariation between individual differences in parent and child EDA profiles (mean and variability) across dyads for the overall free play task. We predicted that dyads that included more physiologically reactive parents (as compared to other parents) would also have more reactive children (as compared to other children), based upon evidence of covariation in EDA as a function of both heritability (Tuvblad et al. 2012) and social partnering (Guastello et al. 2006).

Methods

Participants

The sample included 28 children between the ages of 4 and 10 years and their primary caregivers (one father). See Table 1 for sample information. Participating children were required to have received a community diagnosis of ASD provided by a physician or a licensed clinical psychologist. Of note, the sample was relatively diverse with regard to race/ethnicity and socioeconomic status, with fewer than half of the participants identified as Caucasian, non-Hispanic, and 27 % of families reporting annual household incomes <$35,000 per year.

Procedures

All procedures were approved by our institutional review board. After obtaining consent from parents and assent from the children, wireless EDA sensors were placed on the dominant wrist of each child and parent. A short orientation period (approximately 5 min) then occurred wherein one experimenter discussed the visit with the mother while another gained rapport with the child. This was followed by a 4-min prohibition task that was not utilized in the current study. The dyads next engaged in a naturalistic 4-min free play that has been used in several studies of children with ASD and related developmental concerns (e.g., Baker et al. 2007, 2010). The room was large with blank walls, two large one-way mirrors, and two video cameras mounted high on the walls. A large clear box full of toys, including cars, common movie character figurines, blocks, a toy cash register, and cutable wooden fruit was placed at the front of the room. Dyads were told, "Please play together as you typically would at home" (Baker et al. 2007). The dyad was then left alone to explore the toys, and was observed through a one-way mirror. A series of additional laboratory tasks followed the free play; an abbreviated IQ assessment and a direct measure of autism symptoms were performed later in the visit by a licensed clinical psychologist with expertise in ASD.

Table 1 Descriptive statistics for sample characteristics and variables of interest

Variable	Value
Mean child age in years (*SD*)	6.70 (1.81)
Child is male (%)	79 %
Primary caregiver is married (%)	81 %
Mean annual family income	$50,000–$70,000
Race/ethnicity (% parent/child)	
Caucasian, non-Hispanic	36/43 %
Hispanic	29/25 %
Asian American	21/11 %
African American	4/11 %
"Other"	7/7 %
Mean child ABIQ (*SD*)	85.78 (24.16)
ASD symptom score mean (*SD*)	7.33 (2.40)
Uncorrected EDA score mean (*SD*)	
Parent	1.07 (2.20)
Child	1.30 (2.36)
Mean correlation between parent and child EDA (*SD*)	0.24 (.57)
Mean affective mutuality ratings (*SD*)	4.46 (1.48)

EDA electrodermal activity, *ABIQ* abbreviated battery IQ

Measures

Electrodermal Activity (EDA)

EDA was recorded in microsiemens at 8 Hz using wireless *Affectiva* Q-Sensors (Picard et al. 2014; Poh et al. 2010; Sano et al. 2014) worn by each parent and child. The sensors utilized Ag/AgCl dry disc electrodes and data were recorded and stored within the wrist sensor itself, and downloaded for later analysis. Although measurement from the wrist is less standard and may result in decreased sensitivity to small changes in EDA as compared to certain other locations (Van Dooren et al. 2012), evidence suggests reliability of wrist data with traditional measurement locations, and that wrist measurement may actually be more sensitive to EDA under certain circumstances (Poh et al. 2010; Sano et al. 2014; Van Dooren et al. 2012). In addition to logging EDA, the sensors also recorded movement across three dimensions using a triaxis accelerometer. Movement data were summed across the three dimensions for each measurement frame and considered as a covariate. EDA scores during the free play were collapsed from 8 Hz (1920 frames per dyad) to 2 s (120 frames), to better approximate the temporal window for the processes of interest (Boucsein 2012; Skowron and Hastings 2014).

Autism Symptoms

Level of autism symptoms was assessed through direct testing with the Autism Diagnostic Observation Schedule-2 (ADOS-2; Lord et al. 2012). The ADOS-2 is a semi-structured assessment that facilitates observation and recording of child behaviors related to language, social communication and interaction, play, and repetitive behaviors and restricted interests. The ADOS-2 consists of five modules, one of which is selected for administration based upon an individual's age and expressive language use. The present study utilized the ADOS-2 comparison score, which allowed for examination of ASD symptomatology across different modules, with 1 indicative of minimal to no evidence of ASD-related symptoms, and 10 reflecting a high level of symptoms.

Affective Mutuality

Observation of affective mutuality during the free play was measured with the Affective Mutuality Scale of the NICHD Early Child Care Research Network Scales (e.g. 2003; McElwain et al. 2008). Dyads were rated from video on a seven-point scale (1 = *very low* to 7 = *very high*) for behaviors indicative of affective attunement, comfort of emotional exchange, and positive responsiveness to one another. Inter-rater reliability for the current study, as performed on 46 % of tapes, was high, $ICC = 0.93$.

Child IQ

An estimate of child IQ was obtained for descriptive purposes and for consideration as a control variable using the Abbreviated Battery of the Stanford-Binet Intelligence Scales, Fifth Edition (Roid 2003).

Results

Data Analysis Plan

EDA data often exhibit positive skew and require transformation prior to analysis to reduce non-normality (Boucsein 2012). We performed a standard log transformation to address positive skew in our EDA data. The secondary analyses examining overall EDA pattern covariation (mean and variability) between parents and their children were examined through basic correlation, as was our analysis of biological covariation and observed affective mutuality. Regarding the latter, more complex modeling was not used for this largely methodological examination due to sample size considerations.

Our main investigation of synchrony in relation to ASD symptoms was tested using hierarchical linear models (HLM; Raudenbush et al. 2011). HLMs can simultaneously model the effects of time-varying (EDA and in-time covariates) and non-time-varying (ASD symptoms) factors. HLMs allow for using a within-person, repeated measures design, and are currently recommended for the analysis of biological covariation between individuals (Skowron and Hastings 2014). Level 1 of the model involved the regression of child EDA scores (per 2-second interval) on to parent EDA scores (per 2-second interval), controlling for child movement (per 2-second interval) and time. The control for time prevented against inflated covariation estimates due to any growth in mean EDA levels across the task (i.e., the dyads tending to end higher in EDA than they began; Curran and Bauer 2011; Skowron and Hastings 2014).[1] The Level 1 equation was as follows:

$$\begin{aligned} Child\ EDA\_L_{ij} = {} & \beta_{0j} + \beta_{1j}\left(Child\ Movement_{ij}\right) \\ & + \beta_{2j}\left(Time_{ij}\right) + \beta_{3j}\left(Parent\ EDA_{ij}\right) + r_{ij} \end{aligned}$$

Level 2 included the main effect of the ADOS-2 comparison score as well as a cross-level interaction between the ADOS-2 comparison score and parent EDA. This cross-level interaction tested whether ASD symptoms moderated the association between parent EDA and child EDA. The cross-level interaction between ASD symptoms and time was also included, in order to control for variability in child EDA patterns over time as a function of ASD symptoms.

$$\beta_{0j} = \gamma_{00} + \gamma_{01}\left(ASD\ Symptoms_j\right) + u_{0j}$$

$$\beta_{1j} = \gamma_{10}$$

$$\beta_{2j} = \gamma_{20} + \gamma_{21}\left(ASD\ Symptoms_j\right)$$

$$\beta_{3j} = \gamma_{30} + \gamma_{31}\left(ASD\ Symptoms_j\right) + u_{3j}$$

[1] The models were also run using group-centered parent EDA rather than controlling for time. The findings were nearly identical in that the direction and significance of all effects were the same in both models and the magnitudes of effects were highly similar.

Due to sample size considerations, control variables (e.g., child age, IQ, race, gender, and family income) were tested separately as Level 2 main effects. None of these variables were significantly related to parent–child EDA covariation, thus only the ADOS-2 comparison score was included at Level 2 in the final model.

Missing Data and Sample Characteristics

All 56 participants (parents and children) tolerated the application of the EDA sensors for the entirety of the free play. The parent sensors malfunctioned for two of the free play tasks, however, and one family needed to leave early such that no measure of ASD symptoms was performed. These cases were omitted from the relevant analyses. Abbreviated Battery IQ scores for the children ranged from 47 to 139, indicating a wide range of estimated intellectual functioning. The average comparison score for level of autism-related symptoms on the ADOS-2 fell between "moderate" and "high" (see Table 1).

Parent–Child Covariation in Overall EDA Patterns

Results indicated a moderate, positive correlation between individual differences in parent and child EDA variability (standard deviation across the task), $r = .40$, $p < .05$, indicating that more physiologically variable parents tended to be in dyads with more variable children. Covariation of the mean levels of parent and child EDA for the overall free play was somewhat lower and not significant, $r = .31$, $p = .12$.

Parent–Child Biobehavioral Synchrony

Descriptive Data

Observed affective mutuality scores spanned the full 7-point scale, with the mean falling within the "moderate" to "moderately high" mutuality range (see Table 1). Physiological covariation exhibited similar variation, with one-third of dyads (as per bivariate correlation) associated at above $r = .66$ ($n = 8$), one-third falling between $r = -.05$ and .66 ($n = 9$), and the remaining third scoring further into the negative range ($n = 9$).

Correlation Between Biological Covariation and Affective Mutuality

A moderate positive correlation was found between the degree of positive association (correlation) of parent and child EDA scores (per 2-second interval) during the free play, and the observational ratings of dyadic affective

Table 2 Hierarchical linear model predicting child electrodermal activity (EDA)

Fixed effect	Coefficient	*SE*	*t* ratio	*p* value	CIs
Child EDA intercept, β_0					
Intercept, γ_{00}	0.51	0.18	2.86	.009	0.17 to 0.87
ASD symptoms, γ_{01}	0.11	0.072	1.57	.131	−0.028 to 0.25
Child movement, β_1					
Intercept, γ_{10}	−0.0053	0.0044	−1.19	.233	−0.014 to 0.0033
Time, β_2					
Intercept, γ_{20}	−0.00022	0.000032	−6.99	<.001	−0.00028 to −0.0016
ASD symptoms, γ_{21}	0.00013	0.000015	8.39	<.001	0.000096 to 0.00015
Parent EDA, β_3					
Intercept, γ_{30}	0.80	1.21	0.67	.510	−1.56 to 3.18
ASD symptoms, γ_{31}	−1.44	0.50	−2.86	.009	−2.42 to −0.46

The near-zero values above represent unstandardized coefficients and are close to zero due to the scaling of the variables involved

mutuality, $r = .49$, $p < .05$, indicating that dyads that were more highly positive in their EDA correlations across the free play also tended to be rated higher in observed affective mutuality.

Associations Between Biological Covariation and Autism Symptom Levels

After adjusting for time and movement, a significant cross-level interaction emerged between ASD symptom levels and parent EDA in predicting child EDA (Table 2). Decomposition of this interaction using simple slopes (Preacher et al. 2006) revealed that the slope for parent–child physiological covariation at the ASD symptom level one standard deviation below the sample mean was significant, slope = 4.38, $t = 2.49$, $p < .05$. The slopes were not significant at either the sample-mean level of ASD symptoms, slope = .81, $t = .67$, *ns*, or at the ASD symptom level one standard deviation above the mean, slope = −2.76, $t = -1.61$, *ns* (see Fig. 1). Evaluation of the regions of significance of the moderation effect estimated that covariation between parent and child EDA became significant at values of autism symptom levels <5.76 (moderate symptoms). Of note, the interaction between ASD symptom levels and time was significant, suggesting that children with lower ASD symptoms tended to exhibit greater decline in EDA levels over the task than did children with higher symptom levels (Table 2).

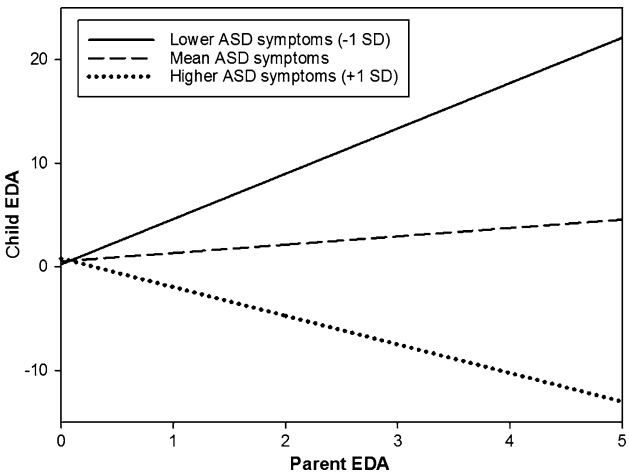

Fig. 1 Parent–child covariation of electrodermal activity (EDA) as a function of ASD symptoms

Discussion

This is the first study to apply the theory of biobehavioral synchrony (Feldman 2012) to children with ASD. Results suggest that stronger positive covariation in sympathetic nervous system arousal between parents and their children with ASD may be related to lower levels of child ASD symptoms. As with most preliminary data, these findings require further study. It is possible that ASD symptoms interfere with the development of synchrony. Future investigations will need to determine the degree to which this effect is mediated by symptoms exhibited during the measurement period, or whether asynchrony in certain dyads may be the result of a history of challenging interaction. Consistent with our conceptualization of these effects as bidirectional, this finding also supports the intriguing notion that the maintenance of caregiver–child physiological synchrony might contribute to reduced ASD symptom levels over time (Dawson 2008). Sequential micro-coding of in-time symptom manifestation and the use of a longitudinal design would extend the current findings.

Although interesting, our finding that more physiologically variable parents were from dyads with more variable

children requires further attention. It will be important to discriminate the degree to which this correlation might reflect heritability versus social partnering (Guastello et al. 2006). Furthermore, although parent–child, in-time covariation in these processes appears important, it is not yet established that these individual indices of functioning are of consequence for children with ASD. Investigations suggest that EDA reactivity may play a role in the development of problems in children with neurotypical development (Beauchaine 2001); however, this association is not yet established for children with ASD (McCormick et al. 2014), and studies of EDA in general have not tended to focus on the type of long-term, naturalistic patterns of arousal examined in the present investigation.

We hope that the current findings promote interest in examining biobehavioral synchrony in children with ASD, particularly as a potential explanatory mechanism for the effects of interventions focused on the maintenance of parent–child engagement (e.g., Dawson et al. 2010). It remains to be seen, however, whether covariation in sympathetic nervous system arousal is meaningful in-and-of-itself, or whether it is a prerequisite or even just a correlate of other causal processes. Studies with larger samples may also consider that certain forms of parent–child physiological *asynchrony* may be beneficial (e.g., co-regulatory; Cole et al. 2004), and that covariation of sympathetic nervous system arousal may not always be helpful (e.g., if child distress is met with parent distress).

Findings are preliminary given our relatively modest sample size. Nonetheless, HLMs have been used effectively with smaller samples in prior ASD research (e.g., Lerner et al. 2011). The present sample was considerably more diverse with regard to developmental functioning and level of ASD symptomatology than most studies of physiology in this population, and no study to our knowledge has examined EDA during naturalistic parent–child interaction. In this study, the free play always followed a delay of gratification task, thus potential carryover effects might be active and could be considered in future investigations. Incorporating a comparison group of children with neurotypical development might be useful for future research, to consider potential diagnostic-group differences in synchrony; however, we were primarily interested in understanding individual differences among children with ASD. Ultimately, the intent of this pilot project was to encourage a new framework of study in ASD that emphasizes the intersection between interactive behavior and physiological functioning as a potential environmental support for these children.

Acknowledgments This project was funded by an intramural faculty award given to the first author from the California State University, Fullerton.

Author contributions JB and RF conceived of the study, participated in its design and coordination, performed certain statistical analyses, and drafted much of the manuscript; MH and BB participated in the design and interpretation of the data, performed certain statistical analyses, and drafted portions of the manuscript; MH and JM participated in the design and coordination of the study, performed aspects of the measurement, and contributed text to the manuscript; SE participated in the design and interpretation of the data and contributed to the manuscript. All authors read and approved the final manuscript.

References

Baker, J. K., Fenning, R. M., Crnic, K. A., Baker, B. L., & Blacher, J. (2007). Prediction of social skills in 6-year-old children with and without developmental delays: Contributions of early regulation and maternal scaffolding. *American Journal on Mental Retardation, 112*, 375–391. doi:10.1352/0895-8017(2007)112[0375:POSSIY]2.0.CO;2.

Baker, J. K., Messinger, D. S., Lyons, K. K., & Grantz, C. J. (2010). A pilot study of maternal sensitivity in the context of emergent autism. *Journal of Autism and Developmental Disorders, 40*, 988–999. doi:10.1007/s10803-010-0948-4.

Beauchaine, T. (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.

Boucsein, W. (2012). *Electrodermal activity* (2nd ed.). New York, NY: Springer.

Cole, P. M., Martin, S. E., & Dennis, T. A. (2004). Emotion regulation as a scientific construct: Methodological challenges and directions for child development research. *Child Development, 75*, 317–333. doi:10.1111/j.1467-8624.2004.00673.x.

Creaven, A., Skowron, E. A., Hughes, B. M., Howard, S., & Loken, E. (2014). Dyadic concordance in mother and preschooler resting cardiovascular function varies by risk status. *Developmental Psychobiology, 56*, 142–152. doi:10.1002/dev.21098.

Curran, P. J., & Bauer, D. J. (2011). The disaggregation of within-person and between-person effects in longitudinal models of change. *Annual Review of Psychology, 62*, 583–619. doi:10.1146/annurev.psych.093008.100356.

Dawson, G. (2008). Early behavioral intervention, brain plasticity, and the prevention of autism spectrum disorder. *Development and Psychopathology, 20*, 775–803. doi:10.1017/S0954579408000370.

Dawson, G., Rogers, S., Munson, J., Smith, M., Winter, J., Greenson, J., et al. (2010). Randomized, controlled trial of an intervention for toddlers with autism: The Early Start Denver Model. *Pediatrics, 125*, 17–23. doi:10.1542/peds.2009-0958.

Feldman, R. (2012). Bio-behavioral synchrony: A model for integrating biological and microsocial behavioral processes in the study of parenting. *Parenting: Science and Practice, 12*, 154–164. doi:10.1080/15295192.2012.683342.

Feldman, R., Magori-Cohen, R., Galili, G., Singer, M., & Louzoun, Y. (2011). Mother and infant coordinate heart rhythms through episodes of interaction synchrony. *Infant Behavior & Development, 34*, 569–577. doi:10.1016/j.infbeh.2011.06.008.

Fowles, D. C. (2008). The measurement of electrodermal activity in children. In L. A. Schmidt & S. J. Segalowitz (Eds.), *Developmental psychophysiology: Theory, systems, and methods* (pp. 286–316). New York, NY: Cambridge University Press.

Greenspan, S. I., & Wieder, S. (1999). A functional developmental approach to autism spectrum disorders. *Journal of the*

Association for Persons with Severe Handicaps, 24, 147–161. doi:10.2511/rpsd.24.3.147.

Guastello, S. J., Pincus, D., & Gunderson, P. R. (2006). Electrodermal arousal between participants in a conversation: Nonlinear dynamics and linkage effects. *Nonlinear Dynamics, Psychology, and Life Sciences, 10*, 365–399.

Howlin, P., Magiati, I., & Charman, T. (2009). Systematic review of early intensive behavioral interventions for children with autism. *American Journal on Intellectual and Developmental Disabilities, 114*, 23–41.

Kasari, C., Gulsrud, A. C., Wong, C., Kwon, S., & Locke, J. (2010). Randomized controlled caregiver mediated joint engagement intervention for toddlers with autism. *Journal of Autism and Developmental Disorders, 40*, 1045–1056. doi:10.1007/s10803-010-0955-5.

Kylliäinen, A., & Hietanen, J. K. (2006). Skin conductance responses to another person's gaze in children with autism. *Journal of Autism and Developmental Disorders, 36*(4), 517–525. doi:10.1007/s10803-006-0091-4.

Lerner, M. D., Mikami, A. Y., & Levine, K. (2011). Socio-dramatic affective-relational intervention for adolescents with Asperger syndrome & high functioning autism: Pilot study. *Autism, 15*, 21–42. doi:10.1177/1362361309353613.

Lord, C., Rutter, M., DiLavore, P. C., Risi, S., Gotham, K., & Bishop, S. L. (2012). *Autism diagnostic observation schedule, second edition (ADOS-2) Manual*. Torrance, CA: Western Psychological Services.

Lovaas, O. I. (1987). Behavioral treatment and normal educational and intellectual functioning in young autistic children. *Journal of Consulting and Clinical Psychology, 55*, 3–9. doi:10.1037/0022-006X.55.1.3.

McCormick, C., Hessl, D., Macari, S. L., Ozonoff, S., Green, C., & Rogers, S. J. (2014). Electrodermal and behavioral responses of children with autism spectrum disorders to sensory and repetitive stimuli. *Autism Research, 7*, 468–480. doi:10.1002/aur.1382.

McElwain, N. L., Booth-LaForce, C., Lansford, J. E., Wu, X. Y., & Dyer, W. J. (2008). A process model of attachment-friend linkages: Hostile attribution biases, language ability, and mother–child affective mutuality as intervening mechanisms. *Child Development, 79*, 1891–1906. doi:10.1111/j.1467-8624.2008.01232.x.

NICHD Early Child Care Research Network. (2003). Early child care and mother-child interaction from 36 months through first grade. *Infant Behavior and Development, 26*, 345–370. doi:10.1016/S0163-6383(03)00035-3.

Picard, R. W., Fedor, S., & Ayzenberg Y. (2014). Multiple arousal theory and daily-life electrodermal activity asymmetry. *Emotion Review*. http://affect.media.mit.edu/publications.php

Poh, M. Z., Swenson, N. C., & Picard, R. W. (2010). A wearable sensor for unobtrusive, long-term assessment of electrodermal activity. *IEEE Transactions on Biomedical Engineering, 57*, 1243–1252. doi:10.1109/TBME.2009.2038487.

Preacher, K. J., Curran, P. J., & Bauer, D. J. (2006). Computational tools for probing interaction effects in multiple linear regression, multilevel modeling, and latent curve analysis. *Educational and Behavioral Statistics, 31*, 437–448.

Raudenbush, S. W., Bryk, A. S., Cheong, A. S., Fai, Y. F., Congdon, R. T., & du Toit, M. (2011). *HLM 7: Hierarchical linear and nonlinear modeling*. Lincolnwood, IL: Scientific Software International.

Roid, G. H. (2003). *Stanford-Binet intelligence scales* (5th ed.). Itasca, IL: Riverside Publishing.

Sano, A., Picard, R. W., & Stickgold, R. (2014). Quantitative analysis of wrist electrodermal activity during sleep. *International Journal of Psychophysiology, 94*, 382–389. doi:10.1016/j.ijpsycho.2014.09.011.

Schneiderman, I., Kanat-Maymon, Y., Zagoory-Sharon, O., & Feldman, R. (2014). Mutual influences between partners' hormones shape conflict dialog and relationship duration at the initiation of romantic love. *Social Neuroscience, 9*, 337–351. doi:10.1080/17470919.2014.893925.

Schoen, S. A., Miller, L., Brett-Green, B., & Hepburn, S. L. (2008). Psychophysiology of children with autism spectrum disorder. *Research in Autism Spectrum Disorders, 2*, 417–429. doi:10.1016/j.rasd.2007.09.002.

Siller, M., & Sigman, M. (2008). Modeling longitudinal change in the language abilities of children with autism: Parent behaviors and child characteristics as predictors of change. *Developmental Psychology, 44*, 1691–1704. doi:10.1037/a0013771.

Skowron, E. & Hastings, P. (2014). Examining the importance of physiological self-regulation for parenting across the spectrum of risk. Symposium presented at the Society for Research in Child Development Special Topic Meeting on New Conceptualizations in the Study of Parenting-At-Risk. San Diego, CA.

Tuvblad, C., Gao, Y., Isen, J., Botwick, T., Raine, A., & Baker, L. A. (2012). The heritability of the skin conductance orienting response: A longitudinal twin study. *Biological Psychology, 89*, 47–53. doi:10.1016/j.biopsycho.2011.09.003.

van Dooren, M., de Vries, J., & Janssen, J. H. (2012). Emotional sweating across the body: Comparing 16 different skin conductance measurement locations. *Physiology & Behavior, 106*, 298–304. doi:10.1016/j.physbeh.2012.01.020.