Archival Report

Biological Psychiatry: CNNI

Brain Similarity as a Protective Factor in the Longitudinal Pathway Linking Household Chaos, Parenting, and Substance Use

Jungmeen Kim-Spoon, Tae-Ho Lee, Claudia Clinchard, Morgan Lindenmuth, Alexis Brieant, Laurence Steinberg, Kirby Deater-Deckard, and Brooks Casas

ABSTRACT

BACKGROUND: Socioecological factors such as family environment and parenting behaviors contribute to the development of substance use. While biobehavioral synchrony has been suggested as the foundation for resilience that can modulate environmental effects on development, the role of brain similarity that attenuates deleterious effects of environmental contexts has not been clearly understood. We tested whether parent-adolescent neural similarity—the level of pattern similarity between parent-adolescent functional brain connectivity representing the level of attunement within each dyad—moderates the longitudinal pathways in which household chaos (a stressor) predicts adolescent substance use directly and indirectly via parental monitoring.

METHODS: In a sample of 70 parent-adolescent dyads, similarity in resting-state brain activity was identified using multipattern connectivity similarity estimation. Adolescents and parents reported on household chaos and parental monitoring, and adolescent substance use was assessed at a 1-year follow-up.

RESULTS: The moderated mediation model indicated that for adolescents with low neural similarity, but not high neural similarity, greater household chaos predicted higher substance use over time directly and indirectly via lower parental monitoring. Our data also indicated differential susceptibility in the overall association between household chaos and substance use: Adolescents with low neural similarity exhibited high substance use under high household chaos but low substance use under low household chaos.

CONCLUSIONS: Neural similarity acts as a protective factor such that the detrimental effects of suboptimal family environment and parenting behaviors on the development of adolescent health risk behaviors may be attenuated by neural similarity within parent-adolescent bonds.

https://doi.org/10.1016/j.bpsc.2023.04.008

Adolescent substance use behaviors represent a pernicious—and potentially preventable—risk to mortality, health, and functioning that has long-term consequences of addiction and physical and mental health problems during adulthood (1–3). At present, however, little is known about neurobiological processes that may intervene in the development of substance use. Following the literature suggesting that biobehavioral synchrony, seen in humans' integration of physiology and behavior during social contact, serves as the foundation for resilience that modulates environmental effects on development (4,5), the present study investigated the buffering role of parent-adolescent neural dyadic similarity against stressful environments. Specifically, we examined whether parent-adolescent resting-state network connectome similarity moderated the detrimental effects of household chaos on adolescent health risk behaviors that operate directly as well as indirectly through parental monitoring.

Research indicates that socioecological factors such as family environment and parenting behaviors contribute to the development of substance use (6). There is an increasing body of evidence spanning childhood and adolescence documenting the detrimental effects of household chaos on parenting and adolescent adjustment, including risk taking. Specifically, household chaos is related to poorly regulated parenting behaviors, which is known to be a prominent predictor of delinquent behaviors (7–11). In the current study, we tested a developmental pathway through which household chaos as a stressful ecological context is associated with impaired parental monitoring, which in turn predicts greater adolescent substance use over time.

Social connections are critical for healthy development. Early in life, connectedness between a child and their parent lays the foundation for children to adapt to increasingly complex environments (12). Furthermore, throughout childhood and adolescence, parents provide psychological and social references upon which children can base and test their actions, thoughts, and feelings in ways that are more likely to be socially acceptable (13). Accordingly, parent-child behavioral dyadic similarity can promote social-emotional adjustment (14,15) and diminish psychopathology (16,17). Research also suggests that parent-child physiological synchrony indicates bonding between the parent and child and has long-term effects on developmental outcomes (e.g., self-regulation, behavioral problems) across childhood and up to adolescence (16). We note that these prior studies focused on parent-child synchrony, which is generally defined as the coordination of the temporal structure of physiological or behavioral processes (e.g., feelings, heart rates, hormonal levels) between interactive partners. The dyadic synchrony or concordance concept has been extended to neuroimaging research and suggests that the behavioral level of dyadic concordance in parent-adolescent dyads is based on their intrinsically attuned brain systems. Specifically, functional connectivity pattern similarity (as opposed to temporal structure similarity) has been found to predict not only parent-adolescent daily synchrony (e.g., one's emotion score predicting the other's emotion score) but also positive outcomes (e.g., higher emotional competence) in adolescents (18).

Parent-adolescent neural similarity may be beneficial to adolescent well-being because of adolescents' attunement to (i.e., being aware of and responsive to) parenting behaviors. Feldman (4) argued for the importance of attunement, or biobehavioral synchrony between parent and child, in providing a foundation for healthy behavioral, socioemotional, and cognitive development. Prior research suggests a link between greater mother-infant synchrony in affect states and more optimal attachment behavior in early life (16). In childhood, the role of parent-child synchrony seems to become more complex: Greater parent-child physiological synchrony is related to better parent-child interactions (19), but high parent-child physiological synchrony is not adaptive for children with parents who lack appropriate socialization skills (20). In adolescence, greater parent-adolescent neural similarity may serve as a foundation for fine-tuning the adolescent's brain to be better attuned to parents' actions and intentions so that positive effects of effective parental socialization would be facilitated. However, it is also possible that greater parent-adolescent neural similarity may amplify negative effects of ineffective parental socialization.

For effective socialization, parents must monitor their adolescents' behaviors, recognize risky behaviors when they occur, and prevent such behaviors (21). Adequate parental monitoring is critical for helping adolescents to avoid risky behaviors because it makes them more conscious of possible consequences of their actions. Indeed, adolescents with parents who are aware of and attempt to guide their behaviors are less likely to engage in health risk behaviors such as substance use (22,23), and higher parental monitoring is related to lower adolescent substance use through the promotion of self-regulation abilities (24). This important socialization process of parental monitoring may work best at minimizing adolescent maladjustment within dyads with greater parent-adolescent neural similarity because this similarity provides a foundation on which parental monitoring more effectively delays or otherwise reduces the development of adolescent risk-taking behaviors.

In the current longitudinal study, we investigated functional connectome similarity in the intrinsic neural signal based on resting-state functional magnetic resonance imaging (fMRI) as an index of neural similarity. There are clear advantages of this approach. First, dyadic neural similarity was estimated based on multidimensional neural pattern similarity across networks. In response to the environment, the brain coordinates various operations simultaneously within and across different regions. Therefore, the brain's neural networks function as an orchestra instead of employing a single region (25). Using a network approach provides a more comprehensive description of dyadic neural similarity across the brain as compared with using overall signal magnitude for each region. Second, the intrinsic functional connectome built on the resting-state fMRI data provides an index of the individual's unique brain fingerprint that reflects the native neural configurations of the brain's system leading to individual differences in psychology and behavior (26,27). Further, the intrinsic connectome is modified and tuned gradually by accumulating socioemotional experiences (28). Therefore, the resting-state network connectome similarity can be seen as the indicator of the harmonized brain architecture between the parent and the adolescent, which is shaped by their shared lived experience over the life course. As such, higher neural pattern similarity observed in resting-state functional connectivity represents a more intrinsically attuned brain system within the dyad. We clarify that we examined resting-state network pattern configuration similarity, whereas most prior research on behavioral and physiological dyadic synchrony examined temporal structure similarity.

Based on theoretical work suggesting beneficial effects of parent-child similarity (4,12,14), we hypothesized that neural similarity between a parent and their adolescent would operate as a bioregulatory protective factor against the negative impact of stressful environments on adolescent outcomes. Specifically, neural similarity as a moderator was expected to attenuate the effects of a chaotic family environment (e.g., lack of structure and unpredictability in everyday activities) on adolescent substance use. We also hypothesized that neural similarity would buffer against adverse substance use outcomes in homes with greater levels of chaos by influencing parental monitoring. Specifically, we anticipated that higher household chaos would predict higher adolescent substance use directly and also indirectly via lower parental monitoring but that these effects would be reduced by higher levels of parent-adolescent neural similarity. Additionally, we tested the possibility that neural similarity would operate as a differential susceptibility factor (29,30) such that adolescents with high versus low neural similarity would be differentially affected by household contextual influences in both the positive and the negative conditions, i.e., low and high levels of chaos, respectively.

METHODS AND MATERIALS

Participants

The current study used a subset of a larger longitudinal study, including 70 parent-adolescent dyads whose resting-state fMRI data were available for analyses. Adolescents (43% female) were 13 to 17 years of age (mean = 15.07, SD = 1.28) at time 1 when fMRI data from both adolescents and their parents were collected along with household chaos and parental monitoring data from adolescents. Adolescent substance use data were collected at time 2, approximately 1 year later. Approximately 14% of adolescents identified as Black, 78% as

White, and 8% as other. Mean annual family income was in the range of $35,000 to $49,999, with varying levels of family economic status (ranging from "less than $1000" to "$200,000 or more"). The primary caregivers (referred to as parents hereafter: 58 mothers, 11 fathers, 1 grandmother) were 34 to 60 years of age (mean = 43.70, SD = 6.75). Inclusion criteria for adolescents included vision corrected to be able to see the computer display clearly, and exclusion criteria included claustrophobia, history of head injury resulting in loss of consciousness for >10 minutes, orthodontia impairing image acquisition, and other contraindications to MRI (e.g., pacemaker, aneurysm clips, neurostimulators, cochlear implants, metal in eyes, steel worker, or other implants). There were 74 parent-adolescent dyads whose resting-state fMRI data were collected together, but 4 dyads had to be excluded from the data analysis if any counterparts' data (either parent or adolescent) showed excessive head motion (mean framewise displacement > 0.25-mm movement).

Procedures

Adolescent participants and their parents were recruited from a southeastern state in the United States via email announcements, flyers, and snowball sampling (word-of-mouth). Data collection was administered at university offices where participants completed self-report questionnaires and behavioral and neuroimaging tasks and were interviewed by trained research assistants. Parent participants were scanned by the same scanner after the adolescent completed scanning on the same day. The study duration was 5 hours on average, and participants were compensated monetarily for their time. All procedures were approved by the institutional review board of the university, and written informed consent or assent was received from all participants.

Household Chaos (Time 1). Adolescents and parents reported on the level of household chaos (i.e., level of confusion and disorganization in the home) using the 6-item Confusion, Hubbub, and Order Scale (31). An example item is "You can't hear yourself think in our home." Response options range from "1 = Definitely untrue" to "5 = Definitely true." Mean scores were calculated, with higher scores indicating higher levels of household chaos (α = 0.62). The reliability was consistent with prior research, which has demonstrated robust predictive and construct validity of this scale (32).

Parental Monitoring (Time 1). Adolescents and parents reported on different aspects of parental monitoring, including parental knowledge, child disclosure, parent solicitation, and parental control, using the 25-item Parental Monitoring Scale (23) along a 5-point scale (1–5) that varied from question to question (e.g., "Do your parents normally know where you go and what you do after school?"). Higher scores indicated greater parental monitoring (α = 0.91).

Adolescent Substance Use (Time 2). Adolescents reported typical frequency of cigarette, alcohol, and marijuana use 1 year after the neural similarity assessment, using a substance use index adapted from Wills *et al.* (33,34). This index consists of 3 items (e.g., "Which is the most true for you about using alcohol?"), using a 6-point scale ranging from 1 (never used) to 6 (usually use every day). A polysubstance use composite score was computed using an average of all 3 items, with higher scores indicating greater use (α = 0.60).

MRI Acquisition and Preprocessing

MRI data acquisition and preprocessing are described in the Supplement.

Connectome Similarity Analysis

To estimate the dyadic neural similarity in functional connectivity between parents and their adolescents (Figure 1), we followed the previous approach of multipattern connectivity similarity estimation [e.g., (18)]. To this end, we used 18 independent component networks (ICNs) that a previous meta-analysis identified (27): ICN 1 (limbic), ICN 2 (medial-temporal areas), ICN 3 (bilateral basal ganglia and thalamus), ICN 4 (bilateral anterior insula/frontal opercula and the anterior aspect of the body of the cingulate gyrus), ICN 5 (midbrain), ICN 6 (superior and middle frontal gyri), ICN 7 (middle frontal gyri and superior parietal lobules), ICN 8 (ventral precentral gyri, central sulci, postcentral gyri, superior and inferior cerebellum), ICN 9 (superior parietal lobule), ICN 10 (middle and inferior temporal gyri), ICNs 11 and 12 (lateral and medial posterior occipital cortices), ICN 13 (medial prefrontal and posterior cingulate/precuneus areas), ICN 14 (cerebellum), ICN

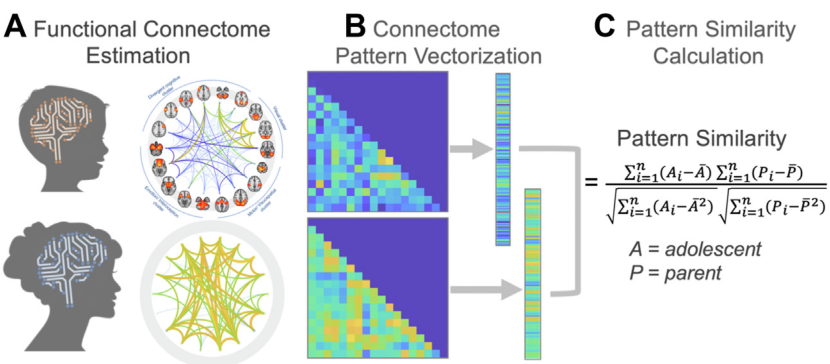

Figure 1. Functional connectome similarity calculation. **(A)** Functional connectome estimation using all possible pairs of 18 intrinsic network maps (27) for each adolescent-parent dyad. **(B)** Characterize individual-specific connectivity patterns and vectorization. Note that only the bottom half of the connectome matrix, excluding the diagonal, is shown in the figure. **(C)** Fisher's *z*–transformed connectome vectors were correlated between each pair of parent-adolescent dyads to calculate the connectome similarity.

15 (right-lateralized frontoparietal regions), ICN 16 (transverse temporal gyri), ICN 17 (dorsal precentral gyri, central sulci, postcentral gyri, superior and inferior cerebellum), and ICN 18 (left-lateralized frontoparietal regions). The extracted individual time courses for each ICN were then used to create a functional network connectivity matrix (i.e., connectome) by correlating (Pearson *r*) all possible pairs of time courses from 18 networks. Finally, the connectome similarity (i.e., connectivity pattern similarity) was calculated by vectorizing each individual's Fisher's *z*–transformed connectivity matrix and correlating between each pair of parent-adolescent dyads (Figure 1). This within-dyad Pearson *r* was used as the neural similarity matrix [e.g., (18,35)]. In addition to this grand level of connectivity pattern similarity across 18 ICNs, we also calculated the connectivity similarity within each intrinsic system. According to Laird *et al.* (27), those 18 ICNs can be clustered by 4 intrinsic systems: emotion (ICNs 1–5), motor/sensory (ICNs 6–9), visual (ICNs 10–12), and cognitive systems (ICNs 13–18). The connectivity similarity across subsets of ICNs within each intrinsic system allowed us to examine which intrinsic cluster drove the neural similarity effects (i.e., specificity of our findings).

Data Analytic Plan

For current study aims, we conducted a moderated mediation analysis using structural equation modeling following recommendations by Hayes (36). We used full information maximum likelihood estimation with robust standard errors (MLR) to estimate the parameters using all available observations rather than imputing missing scores (3 scores of substance use were missing) and to account for non-normal distributions better than maximum likelihood estimation, available in Mplus version 8.6 (37). To test indirect effects, we calculated bias-corrected bootstrap confidence intervals (CIs) with maximum likelihood estimation (bootstrapping is not available for MLR) using 10,000 bootstrapping samples (38). Model fit was assessed by χ^2 value, degrees of freedom, corresponding *p* value, root-mean-square error of approximation (RMSEA), and confirmatory fit index (CFI). RMSEA values <0.08 and CFI values >0.90 were taken to reflect acceptable fits (39). General linear modeling analyses including adolescent gender as a covariate indicated that adolescent gender was not a significant predictor of adolescent substance use ($p = .678$); thus, gender was not included in the model.

In the hypothesized moderated mediation model, we estimated the direct effect of household chaos on subsequent adolescent substance use 1 year later, as well as the indirect effect mediated through parental monitoring. We further tested whether the direct and indirect effects varied by the level of neural similarity (Figure 2).

RESULTS

Descriptive statistics and correlations for all study variables are provided in Table 1. We tested the moderating effects of neural similarity on the direct and indirect effects of household chaos by estimating the interaction effects (i.e., multiplication of centered variables): the direct effect of the household chaos by neural similarity interaction on adolescent substance use and the indirect effect—the household chaos by neural similarity interaction on parental monitoring and the parental monitoring

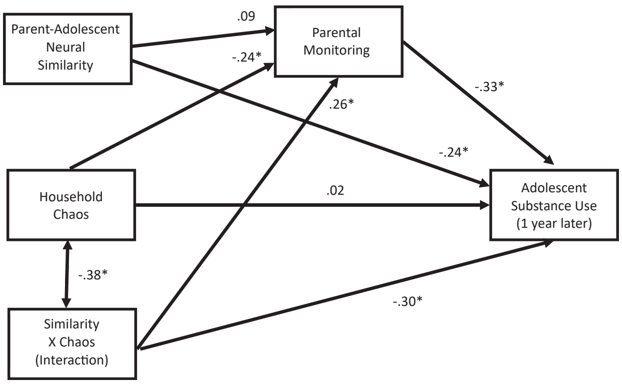

Figure 2. Longitudinal associations among household chaos, parental monitoring, and substance use moderated by parent-adolescent neural similarity. Standardized estimates are presented. *$p < .05$.

by neural similarity interaction on adolescent substance use (Figure 2). This full moderation model showed poor fit (χ^2 = 12.43, *df* = 6, *p* = .053, CFI = 0.84, RMSEA = 0.12) and indicated that the interaction effect between neural similarity and parental monitoring on substance use was not significant (*B* = −0.63, SE = 0.53, *p* = .234). Therefore, we eliminated the neural similarity by parental monitoring interaction term, and this trimmed model showed an acceptable fit with significant improvement of the fit (χ^2 = 0.16, *df* = 2, *p* = .923, CFI = 1.00, RMSEA = 0.00; $\Delta\chi^2$ = 12.27, Δdf = 4, *p* = .015). Furthermore, we tested our model with possible covariates, including parent age, parent substance use, and whether or not the dyads' gender matched, and these covariates were not significant and thus not kept. As reported in Table 2, in the final model, the interaction effect between household chaos and neural similarity was significant for adolescent substance use ($p < .001$). Additionally, there was an indication that the effect of household chaos on parental monitoring may vary depending on neural similarity (*p* = .051), and higher parental monitoring, in turn, was significantly predictive of lower adolescent substance use 1 year later ($p < .001$). The model explained 18% of variances in parental monitoring (mediator) and 34% of variances in adolescent substance use (outcome). Given the low power for detecting interaction effects in quasi-experimental designs, it has been recommended to use $p < .10$ as statistically significant for interaction terms (40,41); we probed the interaction effect between household chaos and neural similarity by inspecting simple effects.

The finding suggested that neural similarity moderated the direct path between household chaos and adolescent substance use as well as the first part of the indirect path between household chaos and parental monitoring. To interpret the moderating effects of neural similarity, we conducted simple slope analyses at low (1 SD below the mean) and high (1 SD above the mean) levels of neural similarity. First, as for the chaos effect on parenting (see Figure 3 for simple slopes and the Supplement for regions of significance), the effect of household chaos on parental monitoring was significant at low levels of neural similarity (*B* = −0.41, SE = 0.13, *p* = .002) but not at high levels of neural similarity (*B* = −0.01, SE = 0.16, *p* = .966). Second, as for the chaos effect on adolescent substance

Table 1. Descriptive Statistics and Bivariate Correlations of Household Chaos, Parental Monitoring, Neural Similarity, and Adolescent Substance Use

	1	2	3	Mean	SD	Minimum	Maximum
Household Chaos, Time 1	–	–	–	2.40	0.65	1.17	4.33
Parental Monitoring, Time 1	−0.35[a]	–	–	4.01	0.56	2.04	5.00
Neural Similarity, Time 1	−0.07	0.11	–	0.40	0.15	0.01	0.71
Substance Use, Time 2	0.29[a]	−0.45[a]	−0.25[a]	1.56	0.71	1.00	3.67

[a]$p < .05$.

use, the direct effect of household chaos on adolescent substance use was significant at low levels of neural similarity (B = 0.31, SE = 0.09, p = .001) but not at high levels of neural similarity (B = −0.27, SE = 0.18, p = .126). Similarly, the indirect effect of household chaos on adolescent substance use via parental monitoring was significant at lower levels of neural similarity (95% CI, 0.052–0.407) but not at higher levels of neural similarity (95% CI, −0.135 to 0.160).

Overall, higher household chaos at time 1 predicted higher adolescent substance use at time 2 directly and also indirectly via lower parental monitoring, but only among dyads with low parent-adolescent neural similarity. After probing the simple slopes in Figure 4 and regions of significance (see the Supplement), the pattern of interaction effects between chaos and neural similarity, considering both direct and indirect effects, on substance use was found to be consistent with differential susceptibility. The region of significance covered both low and high ends of the predictor, and a proportion affected index (i.e., the proportion that is differentially affected by the moderator) was greater than the recommended 16% (42). Our data revealed 26% proportion affected, indicating that 26% of adolescents reported household chaos lower than the crossover point (−0.59) of the interaction effect on substance use.

Sensitivity analyses using parent-reported household chaos and parent-reported monitoring clarified that the interaction effects were specific to adolescent-reported household chaos and adolescent-reported monitoring (see the Supplement). Testing specificity by comparing the results using the whole brain involving all intrinsic networks (18 ICNs) together with the results using each local intrinsic cluster (4 systems) in isolation revealed that the moderating effects of neural similarity for the chaos–substance use link emerged within each local intrinsic cluster (4 systems) in isolation, whereas the moderating role effects of neural similarity on the chaos-monitoring link emerged only when considering the overall patterns of the whole brain involving all intrinsic networks (18 ICNs) together (see the Supplement).

DISCUSSION

Despite past research illustrating the importance of parent-offspring behavioral and physiological synchrony in fostering resilience and well-being (4,5), the role of parent-offspring neural-level similarity is not clearly understood. Most research on dyadic concordance has used physiological and behavioral data representing the synchrony in moment-to-moment states. By comparison, it is not known whether neural configuration pattern similarity is a neurobiological modulator that attenuates negative environmental effects on developmental outcomes over longer periods. The present study tested whether parent-adolescent dyadic similarity of neural activity protects adolescents against harmful effects of stressful home environments (i.e., chaos) by facilitating parental socialization processes (i.e., parental monitoring) and deterring adolescent risk taking (i.e., substance use). Specifically, we measured intrinsic neural pattern similarity between adolescents and their parents as an indicator of dyadic concordance. The brain connectome that is built upon resting-state fMRI data allows us to examine mind-to-mind attunement by providing insight into an individual's unique brain fingerprints that are expected to be modified as individuals interact with people (28). The results of the hypothesized moderated mediation model revealed that only for the parent-adolescent dyads with lower levels of neural similarity, higher household chaos predicted higher adolescent substance use directly and also mediated through lower levels of parental monitoring. Specifically, adolescents and parents with well-attuned brains were buffered against the potentially harmful outcomes (i.e., reduced parental monitoring and greater adolescent substance use) of a chaotic

Table 2. Parameter Estimates for Testing Moderation Effects of Neural Similarity on the Direct and Indirect Effects From Household Chaos to Adolescent Substance Use via Parental Monitoring

	B	SE	p	Beta
Main Effects				
Household chaos (T1) → parental monitoring (T1)	−0.21	0.10	.048	−0.24
Neural similarity (T1) → parental monitoring (T1)	0.34	0.47	.461	0.10
Household chaos (T1) → substance use (T2)	0.02	0.11	.856	0.02
Neural similarity (T1) → substance use (T2)	−1.12	0.46	.014	−0.24
Parental monitoring (T1) → substance use (T2)	−0.42	0.11	.000	−0.33
Interaction Effects				
Household chaos (T1) × neural similarity (T1) → parental monitoring (T1)	1.31	0.67	.051	0.26
Household chaos (T1) × neural similarity (T1) → substance use (T2)	−1.90	0.60	.001	−0.30

T, time.

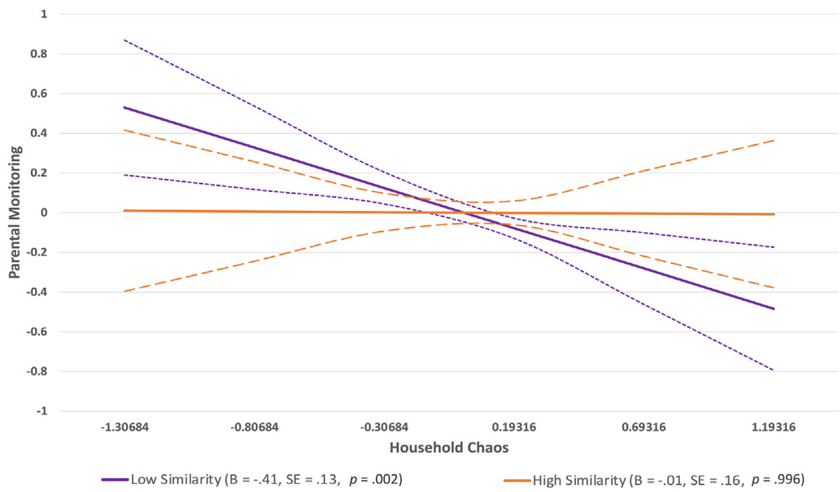

Figure 3. Simple slope analyses comparing associations between household chaos and parental monitoring for adolescents with high neural similarity (>1 SD above mean) and adolescents with low neural similarity (<1 SD below mean). Chaos is presented on a scale of 2 SD above (high chaos) and below (low chaos) the mean.

home environment, such that household chaos did not have significant influences on their substance use. In contrast, in dyads with low levels of brain attunement, living in a disorganized and unpredictable household predicted more substance use not only directly but also indirectly via lower levels of parental monitoring.

The protective effects of neural similarity were found in preserving beneficial parenting behaviors as well as preventing harmful risky behaviors by adolescents in the stressful context of a chaotic household. These beneficial effects of parent-adolescent neural similarity support the theoretical work that views neural synchrony as an adaptive neural mechanism. More in-tune neural states between 2 individuals make their mental connection more effortless and enjoyable, thus promoting positive social bonds (43), which can confer resilience to stress (44). Supporting this perspective, an empirical study demonstrated the link between higher interbrain synchrony (measured by electroencephalography) and more efficient interpersonal interactions (45).

When considering the overall effects of household chaos on adolescent substance use, our data suggested differential susceptibility of neural similarity in that adolescents with low levels of dyadic neural similarity showed high levels of substance use 1 year later in the context of high household chaos while also showing low levels of substance use in the context of low household chaos (30,46). This finding is in line with previous research suggesting physiological synchrony in respiratory sinus arrhythmia between parents and children as a differential susceptibility factor (47). In the previous study of respiratory sinus arrhythmia, children with higher dyadic synchrony were more susceptible to parenting, such that both positive and negative parenting behaviors had stronger effects on their behavioral problems compared with children with lower dyadic synchrony (47). In contrast, we did not find

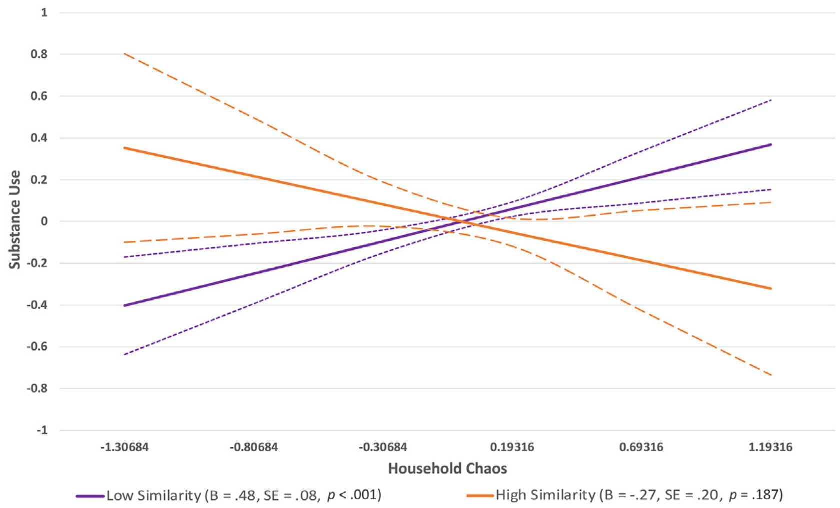

Figure 4. Simple slope analyses depicting total (direct and indirect) effects of household chaos on substance use for adolescents with high neural similarity (>1 SD above the mean) and adolescents with low neural similarity (<1 SD below the mean). Chaos is presented on a scale of 2 SD above (high chaos) and below (low chaos) the mean.

evidence of such amplifying effects of neural similarity with respect to parenting, as indicated by nonsignificant moderation effects of neural similarity on the link between parental monitoring and adolescent substance use. In our study, adolescents with low neural similarity were more susceptible to the effects of a contextual risk factor (i.e., household chaos). Our findings point to the importance of considering the environmental contexts in which parent-offspring interactions are embedded as parenting-offspring biological similarity interfaces with those contextual factors to contribute to adjustment outcomes. Future research may investigate whether high parent-offspring similarity amplifies susceptibility to variations of parenting behaviors due to the source of risk residing in parenting, whereas low parent-offspring similarity amplifies susceptibility to variations of contextual factors (other than parenting per se).

Our findings revealed that parent-adolescent attunement shown by neural similarity exerts protective effects on adolescent substance use in part by attenuating potential detrimental effects of household chaos on parental monitoring. The significant link between greater household chaos and lower parental monitoring extends past research demonstrating the association between household chaos and adolescent behavioral maladjustment mediated by impaired parenting behaviors such as maternal hostility (8). While previous studies primarily focused on parental warmth or hostility to explain the effects of household chaos on adjustment outcomes, our prospective analysis presents preliminary yet novel evidence clarifying that parent-adolescent dyads with higher neural similarity may be better connected, as shown by perceived parental monitoring differing across levels of chaotic household environments depending on the levels of neural similarity.

Our sensitivity tests revealed that moderating effects of parent-adolescent neural similarity between household chaos and adolescent substance use did not hold when using parents' reports of household chaos and monitoring behaviors. This finding demonstrates that adolescents' perception of household and parenting environments plays an important role in the development of risky behaviors. We found that differences between adolescents' and parents' perceptions are consistent with past research indicating a significant discrepancy between parents' and adolescents' reports of family and parenting environments and adolescent adjustment (48,49) and that adolescents' perceptions of their parental monitoring are more predictive of adolescents' risky behaviors (50,51).

Findings from the present study need to be interpreted in light of its limitations. First, although we used a longitudinal design, our correlational data do not allow us to infer causality in the identified associations. Second, given the low numbers of father-adolescent dyads, we were unable to test whether the hypothesized model would hold similarly between the mother-adolescent dyads and the father-adolescent dyads. Furthermore, the majority (94%) of parents were biologically related to the adolescent, and there is a possibility that genetics may influence neural similarity. Future work investigating whether brain similarity effects vary between mothers and fathers and between biologically related versus unrelated parents would be beneficial. Finally, we focused on household chaos, parental monitoring, and neural similarity in predicting adolescent substance use but acknowledge that there are other important biological and socioecological factors (e.g., genetic factors, peer influences) that have been shown to contribute to adolescent substance use behaviors. For instance, the psychobiological model of intergenerational transmission of self-regulation, which plays an important role in substance use development, suggests that individual differences in self-regulation are transmitted across generations within parent-child relationships through biosocial processes (52). Importantly, future work should consider how these predictors of substance use as well as substance use per se may influence parent-child neural similarity across development.

Despite the theoretical importance of neurobiological processes involved in promoting health and well-being (5,53,54), empirical research on these brain processes is lacking. We believe that parent-child neural similarity may provide a window into understanding of the shared life experiences that parents and children have established through their close ties to each other, i.e., linked lives (55). Our longitudinal moderated mediation model results suggest high neural similarity as a protective factor, such that the expected detrimental effects of stressful environments on the development of health risk behaviors may be attenuated by resilience-promoting brain-to-brain similarity within affiliative bonds.

ACKNOWLEDGMENTS AND DISCLOSURES

This work was supported by the National Institute on Drug Abuse (Grant No. R01 DA036017 [to JK-S and BC]).

JK-S, BC, and T-HL designed research; JK-S, T-HL, and CC performed research; JK-S, T-HL, CC, ML, and AB analyzed data; and JK-S, T-HL, CC, and KD-D wrote the paper with help from LS.

We thank the former and current JK Lifespan Development Lab members for their help with data collection. We are grateful to the adolescents and parents who participated in our study.

The authors report no biomedical financial interests or potential conflicts of interest.

ARTICLE INFORMATION

From the Department of Psychology, Virginia Tech, Blacksburg, Virginia (JK-S, T-HL, CC, ML, BC); School of Neuroscience, Virginia Tech, Blacksburg, Virginia (JK-S, T-HL, BC); Department of Psychology, Yale University, New Haven, Connecticut (AB); Department of Psychology and Neuroscience, Temple University, Philadelphia, Pennsylvania (LS); Department of Psychology, King Abdulaziz University, Jeddah, Saudi Arabia (LS); Department of Psychological and Brain Sciences, University of Massachusetts, Amherst, Massachusetts (KD-D); and Fralin Biomedical Research Institute at Virginia Tech Carilion, Roanoke, Virginia (BC).

JK-S and T-HL contributed equally to this work.

Address correspondence to Jungmeen Kim-Spoon, Ph.D., at jungmeen@vt.edu, or Tae-Ho Lee, Ph.D., at taehol@vt.edu.

Received Nov 9, 2022; revised Apr 10, 2023; accepted Apr 17, 2023.

Supplementary material cited in this article is available online at https://doi.org/10.1016/j.bpsc.2023.04.008.

REFERENCES

1. Blum RW, Nelson-Mmari K (2004): The health of young people in a global context. J Adolesc Health 35:402–418.
2. Hammond CJ, Mayes LC, Potenza MN (2014): Neurobiology of adolescent substance use and addictive behaviors: Treatment implications. Adolesc Med State Art Rev 25:15–32.
3. Kessler RC, Berglund P, Demler O, Jin R, Merikangas KR, Walters EE (2005): Lifetime prevalence and age-of-onset distributions of DSM-IV

disorders in the national comorbidity survey replication. Arch Gen Psychiatry 62:593–602.
4. Feldman R (2021): Social behavior as a transdiagnostic marker of resilience. Annu Rev Clin Psychol 17:153–180.
5. Gunnar MR (2021): Forty years of research on stress and development: What have we learned and future directions. Am Psychol 76: 1372–1384.
6. Li M, Lindenmuth M, Tarnai K, Lee J, King-Casas B, Kim-Spoon J, Deater-Deckard K (2022): Development of cognitive control during adolescence: The integrative effects of family socioeconomic status and parenting behaviors. Dev Cogn Neurosci 57:101139.
7. Coldwell J, Pike A, Dunn J (2006): Household chaos–Links with parenting and child behaviour. J Child Psychol Psychiatry 47: 1116–1122.
8. Deater-Deckard K, Godwin J, Lansford JE, Tirado LMU, Yotanyamaneewong S, Alampay LP, *et al.* (2019): Chaos, danger, and maternal parenting in families: Links with adolescent adjustment in low- and middle-income countries. Dev Sci 22:e12855.
9. Marsh S, Dobson R, Maddison R (2020): The relationship between household chaos and child, parent, and family outcomes: A systematic scoping review. BMC Public Health 20:513.
10. Mills-Koonce WR, Willoughby MT, Garrett-Peters P, Wagner N, Vernon-Feagans L, Family Life Project Key Investigators (2016): The interplay among socioeconomic status, household chaos, and parenting in the prediction of child conduct problems and callous-unemotional behaviors. Dev Psychopathol 28:757–771.
11. Lauharatanahirun N, Maciejewski D, Holmes C, Deater-Deckard K, Kim-Spoon J, King-Casas B (2018): Neural correlates of risk processing among adolescents: Influences of parental monitoring and household chaos. Child Dev 89:784–796.
12. Ainsworth MDS, Blehar M, Waters E, Wall S (1978): Patterns of Attachment: A Psychological Study of the Strange Situation. Hillsdale, NJ: Erlbaum.
13. Hostinar CE, Gunnar MR (2015): Social support can buffer against stress and shape brain activity. AJOB Neurosci 6:34–42.
14. Gunnar MR, Hostinar CE, Sanchez MM, Tottenham N, Sullivan RM (2015): Parental buffering of fear and stress neurobiology: Reviewing parallels across rodent, monkey, and human models. Soc Neurosci 10:474–478.
15. Morris AS, Silk JS, Steinberg L, Myers SS, Robinson LR (2007): The Role of the family context in the development of emotion regulation. Soc Dev 16:361–388.
16. Feldman R (2007): Parent–infant synchrony and the construction of shared timing; physiological precursors, developmental outcomes, and risk conditions. J Child Psychol Psychiatry 48:329–354.
17. Suveg C, Braunstein West K, Davis M, Caughy M, Smith EP, Oshri A (2019): Symptoms and synchrony: Mother and child internalizing problems moderate respiratory sinus arrhythmia concordance in mother-preadolescent dyads. Dev Psychol 55:366–376.
18. Lee TH, Miernicki ME, Telzer EH (2017): Families that fire together smile together: Resting state connectome similarity and daily emotional synchrony in parent–child dyads. NeuroImage 152:31–37.
19. DePasquale CE (2020): A systematic review of caregiver–child physiological synchrony across systems: Associations with behavior and child functioning. Dev Psychopathol 32:1754–1777.
20. Creavy KL, Gatzke-Kopp LM, Zhang X, Fishbein D, Kiser LJ (2020): When you go low, I go high: Negative coordination of physiological synchrony among parents and children. Dev Psychobiol 62:310–323.
21. Gottfredson MR, Hirschi T (1990): A General Theory of Crime. Palo Alto: Stanford University Press.
22. Crouter AC, Bumpus MF, Davis KD, McHale SM (2005): How do parents learn about adolescents' experiences? Implications for parental knowledge and adolescent risky behavior. Child Dev 76:869–882.
23. Stattin H, Kerr M (2000): Parental monitoring: A reinterpretation. Child Dev 71:1072–1085.
24. Kim-Spoon J, Farley JP, Holmes C, Longo GS, McCullough ME (2014): Processes linking parents' and adolescents' religiousness and adolescent substance use: Monitoring and self-control. J Youth Adolesc 43:745–756.
25. Buzsáki G, Draguhn A (2004): Neuronal oscillations in cortical networks. Science 304:1926–1929.
26. Finn ES, Shen X, Scheinost D, Rosenberg MD, Huang J, Chun MM, *et al.* (2015): Functional connectome fingerprinting: Identifying individuals using patterns of brain connectivity. Nat Neurosci 18:1664–1671.
27. Laird AR, Fox PM, Eickhoff SB, Turner JA, Ray KL, McKay DR, *et al.* (2011): Behavioral interpretations of intrinsic connectivity networks. J Cogn Neurosci 23:4022–4037.
28. Gabard-Durnam LJ, Gee DG, Goff B, Flannery J, Telzer E, Humphreys KL, *et al.* (2016): Stimulus-elicited connectivity influences resting-state connectivity years later in human development: A prospective study. J Neurosci 36:4771–4784.
29. Belsky J (1997): Theory testing, effect-size evaluation, and differential susceptibility to rearing influence: The case of mothering and attachment. Child Dev 68:598–600.
30. Belsky J, Pluess M (2009): Beyond diathesis stress: Differential susceptibility to environmental influences. Psychol Bull 135:885–908.
31. Matheny AP, Wachs TD, Ludwig JL, Phillips K (1995): Bringing order out of chaos: Psychometric characteristics of the confusion, hubbub, and order scale. J Appl Dev Psychol 16:429–444.
32. Asbury K, Dunn JF, Pike A, Plomin R (2003): Nonshared environmental influences on individual differences in early behavioral development: A monozygotic twin differences study. Child Dev 74:933–943.
33. Kann L, Kinchen S, Shanklin SL, Flint KH, Kawkins J, Harris WA, *et al.* (2014): Youth risk behavior surveillance – United States, 2013. MMWR Suppl 63:1–168.
34. Wills TA, Yaeger AM, Sandy JM (2003): Buffering effect of religiosity for adolescent substance use. Psychol Addict Behav 17:24–31.
35. Lee TH, Qu Y, Telzer EH (2018): Dyadic neural similarity during stress in mother–child dyads. J Res Adolesc 28:121–133.
36. Hayes AF (2022): Introduction to Mediation, Moderation, and Conditional Process Analysis: A Regression-Based Approach, 3rd ed. New York: Guilford Press.
37. Muthén LK, Muthén BO (1998–2021): Mplus User's Guide, 8th ed. Los Angeles: Muthén & Muthén.
38. Preacher KJ, Hayes AF (2008): Asymptotic and resampling strategies for assessing and comparing indirect effects in multiple mediator models. Behav Res Methods 40:879–891.
39. Little TD (2013): Longitudinal Structural Equation Modeling. New York: Guilford press.
40. McClelland GH, Judd CM (1993): Statistical difficulties of detecting interactions and moderator effects. Psychol Bull 114:376–390.
41. Whisman MA, McClelland GH (2005): Designing, testing, and interpreting interactions and moderator effects in family research. J Fam Psychol 19:111–120.
42. Roisman GI, Newman DA, Fraley RC, Haltigan JD, Groh AM, Haydon KC (2012): Distinguishing differential susceptibility from diathesis-stress: Recommendations for evaluating interaction effects. Dev Psychopathol 24:389–409.
43. Wheatley T, Kang O, Parkinson C, Looser CE (2012): From mind perception to mental connection: Synchrony as a mechanism for social understanding. Soc Pers Psychol Compass 6:589–606.
44. Ozbay F, Johnson DC, Dimoulas E, Morgan CA, Charney D, Southwick S (2007): Social support and resilience to stress: From neurobiology to clinical practice. Psychiatry (Edgmont) 4:35–40.
45. Djalovski A, Dumas G, Kinreich S, Feldman R (2021): Human attachments shape interbrain synchrony toward efficient performance of social goals. NeuroImage 226:117600.
46. Ellis BJ, Boyce WT, Belsky J, Bakermans-Kranenburg MJ, Van Ijzendoorn MH (2011): Differential susceptibility to the environment: An evolutionary-neurodevelopmental theory. Dev Psychopathol 23:7–28.
47. Oshri A, Liu S, Suveg CM, Caughy MO, Goodgame Huffman L (2023): Biological sensitivity to context as a dyadic construct: An investigation of child-parent RSA synchrony among low-SES youth. Dev Psychopathol 35:95–108.
48. De Los Reyes A, Kazdin AE (2005): Informant discrepancies in the assessment of childhood psychopathology: A critical review, theoretical framework, and recommendations for further study. Psychol Bull 131:483–509.

49. Human LJ, Dirks MA, DeLongis A, Chen E (2016): Congruence and incongruence in adolescents' and parents' perceptions of the family: Using response surface analysis to examine links with adolescents' psychological adjustment. J Youth Adolesc 45:2022–2035.
50. Abar CC, Jackson KM, Colby SM, Barnett NP (2015): Parent–child discrepancies in reports of parental monitoring and their relationship to adolescent alcohol-related behaviors. J Youth Adolesc 44: 1688–1701.
51. Cottrell L, Li X, Harris C, D'Alessandri D, Atkins M, Richardson B, Stanton B (2003): Parent and adolescent perceptions of parental monitoring and adolescent risk involvement. Parenting 3:179–195.
52. Deater-Deckard K (2014): Family matters: Intergenerational and interpersonal processes of executive function and attentive behavior. Curr Dir Psychol Sci 23:230–236.
53. Feder A, Fred-Torres S, Southwick SM, Charney DS (2019): The biology of human resilience: Opportunities for enhancing resilience across the life span. Biol Psychiatry 86:443–453.
54. Masten AS, Lucke CM, Nelson KM, Stallworthy IC (2021): Resilience in development and psychopathology: Multisystem perspectives. Annu Rev Clin Psychol 17:521–549.
55. Elder GH (1998): The life course as developmental theory. Child Dev 69:1–12.