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Parents' and Children's ADHD in a Family System

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Abstract ADHD symptoms "run in families". However, relatively little is known about the ways in which parents' symptoms might additively and interactively work with the parenting environment, to influence (and be influenced by) the developmental trajectory of symptoms in children and adolescents. In this commentary on the special section addressing this gap in knowledge, emphasis is placed on the importance of replicating and extending family-wide studies of ADHD symptoms and etiology. The current papers exemplify the leading-edge of such efforts, demonstrating the feasibility and rigor with which studies are being conducted, utilizing longitudinal and experimental designs. Families and parenting environments operate as a system in which individuals influence each other's symptoms and functioning. In so doing, parents produce tremendous variability within (as well as between) each family in individuals' ADHD symptoms from childhood through adulthood, via gene-environment transactions that may even begin during prenatal development.

Keywords Attention deficit hyperactivity disorder · Genetics · Parenting · Family systems · Intervention

ADHD "runs in families", is heritable, and is consistently associated with harsher and less positive parenting environments. This conclusion is not news, and is probably regarded as a foregone conclusion by most readers of the journal. However, major gaps in empirical evidence still exist. Nearly all of the evidence is based on small clinical studies or small-to-large behavioral genetic sibling (mostly twin) studies. There is a need for more research that empirically estimates the magnitude of effect sizes for the associations between parent ADHD and child ADHD, and that also examines associations with variability in parent-child relationship processes. The current special section represents a rigorous and very important step toward addressing several of these gaps. The authors and editors are to be commended for compiling an outstanding collection of review and empirical papers that examine connections (and some surprising disconnections) between parents' and children's ADHD symptoms, and parent-child relationship dynamics.

As a set, the special section is tackling some of the most basic and important questions and assumptions about familial transmission of ADHD and comorbid disorders, in an effort to better understand "what works" (and what does not work) when it comes to supporting parents in youth- and family-focused interventions. The special section situates the familial transmission and parenting topic in contemporary theories of family systems, genetic factors, and gene-environment interaction. That is, the papers implicitly or explicitly articulate hypotheses regarding: (a) the complex (potentially indistinguishable) causal influence that each family member has on other family members as part of systems of individuals and behaviors; and (b) parent-child genetic similarity operating alongside socialization influences via parenting behavior, reflecting complex gene-gene and gene-environment interaction processes.

Families and ADHD Symptoms as Systems

The collection of papers presents an important set of empirically-tested effects that reflect a potential causal role

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for both child and parent ADHD symptoms in the etiology of problematic parent-child relationship processes. Because it is neither feasible nor wise to attempt to summarize all of the key pertinent findings, I have provided a "bird's eye view" summary (Table 1) of the key constructs that were assessed and tested in the empirical papers in the special section. This table also highlights whether different results were found within a study, as a function of parent gender or ADHD dimension (i.e., inattention, hyperactivity/impulsivity).

Two patterns in Table 1 stand out. First, in every study in which mothers and fathers were assessed and compared (Moroney et al. 2017, and Auerbach et al. 2017, had samples that were all or nearly all mothers only), there were significant gender differences in associations in the models (noted with a "g" for gender, in Table 1). Second, in the two studies in which the two major dimensions of parental ADHD symptoms were examined separately, dimension differences were found (noted with a "d" for dimensions in Table 1). Relatedly, inclusion of parental comorbid conditions had an impact on results in the two studies that examined these. In spite of the moderate to substantial covariation between adults' dimensions of ADHD and comorbid conditions, inattentive and hyperactive-impulsive symptom scores showed distinct patterns of additive and interactive associations with the other variables being examined in those papers' predictive models. As Chronis-Tuscano et al. (2017) have noted in their review of the treatment literature, there is already good reason to expect distinct additive and interactive effects for mothers and fathers and for particular dimensions of symptoms or comorbid conditions, when predicting trajectories of child ADHD symptoms and parent-child relationship processes. The current set of empirical findings further reinforce the importance of considering both parenting partners and distinct dimensions and comorbid conditions in mothers and fathers. It remains to be seen whether the distinct predictive patterns reported in the special section are replicated for mothers versus fathers and inattentive versus hyperactive-impulsive symptoms. Still, there already is sufficient evidence that studying only one parenting partner or one dimension of ADHD typically will not yield findings that generalize to the other parenting partner or dimension of ADHD. The same reasoning applies to inclusion of multiple dimensions of ADHD and comorbid conditions for children and adolescents in future family studies.

One of the methods for examining "system" concepts is to test higher-order interactive effects between both parents' ADHD symptoms, as well as with child symptoms. Two of the papers did this (Williamson et al. 2017; Wymbs et al. 2017) and one other conducted the preliminary analyses for future testing of an interaction effect (Breaux et al. 2017, who tested additive effects of mother, father and child ADHD symptoms predicting subsequent child ADHD symptoms). As a set, these three papers make clear that significant additive and interactive effects of mother and father ADHD symptoms can be expected, with the papers delving into some detail in inferences regarding "match/mismatch" and "compensatory" processes in couples' parenting as part of the family system. However, it is just as noteworthy that Wymbs et al. found little evidence for significant parent-by-parent-by-child ADHD three-way interaction effects. Still, when considered on the whole, the evidence from these tests of potential additive and interactive effects in the prediction of parenting processes and growth in child ADHD symptoms indicates the need for future studies to be sufficiently large for detecting multiple higher-order family member interaction effects.

The challenge that our field faces for tackling this complexity is that the results from post-hoc probing of statistical interaction effects are hard to replicate. This is especially true for three-way and higher-order interaction effects, and interaction effects that are detected using correlational designs. As most of the authors in the current section emphasize, such tests of parent-parent, parent-child, and family triad symptoms' additive and interactive effects require rigorous study designs and analytic methods. However, these standard approaches alone may not be sufficient for capturing *robust replicable patterns at the family level*, when it comes to identifying associations

Table 1 Special section papers' statistically significant additive or interactive effects

	Cross-sectional studies			Longitudinal studies		
	Williamson	Wymbs	Nikolas	Moroney	Auerbach	Breaux
Parent ADHD	✓g,d	✓g	✓	✓	✓d	✓g
Parent comorbidity	na	✓g	na	na	na	✓g
Family history or genetics	na	na	✓	na	✓	✓g
Family stress/adversity	na	na	na	na	✓	✓g
Parenting behavior	✓g,d	✓g	✓	✓	✓	✓g
Child ADHD	✓	✓	✓	✓	✓d	✓
Child comorbidity	na	✓	✓	✓	na	✓

g gender difference in effect, *d* ADHD dimension difference (inattentive vs. hyperactive/impulsive) in effect, *na* not assessed

between ADHD symptoms, parent-child relationship processes, and other constructs of interest. On this point, I offer two suggestions regarding methodology (a two-part idea involving variable-centered and family-centered analyses), and a third suggestion regarding a family system conceptual framework. These are suggested not only as alternatives to traditional tests of variable-by-variable interaction effects (e.g., mother ADHD by father ADHD, etc.) and family systems theory concepts, but as additional complementary approaches.

Regarding methods and a variable-centered approach, investigators can use family-level composite indices representing overall family history and symptom "load" for each family. This approach has been used widely in epidemiological studies, especially those striving to identify rare genetic variants of large effect size for discrete diseases and disorders (e.g., Hopper et al. 2005). The Nikolas and Momany (2017) paper in the current section provides a version of this kind of approach using examination of genotypes in multiple family members. As a general analytic approach, such measurement scaling and analytic methods might be useful for any family-based studies of dyads, triads or beyond. Within such a framework, competing hypotheses can be tested regarding dyad, triad, and family-level linear (i.e., additive) and nonlinear (i.e., interactive) effects. Researchers can compute (either separately for each dimension of ADHD and comorbid disorders, or for overall ADHD symptoms) an overall symptom load (e.g., sum or count score) for each dyad, a triad, or whole family.

The family-wide index of the continuum of ADHD symptoms can then be examined for linear and nonlinear associations with other constructs of interest, such as the parent-child relationship. An example is provided in Fig. 1, in which the potential statistical prediction of harsh, negative family relationship processes (inter-partner, parent-child, or a composite of these) from between-family variation in family-wide ADHD symptom load is estimated. Competing hypotheses can be tested for linear and nonlinear effects or functions. In this example, one would test for an "enhancing" pattern (accelerating effect at higher levels of family-wide symptom load) versus a "swamping" pattern (decelerating effect at higher levels of family-wide symptom load). Estimating and testing for such competing hypothesized functions in no way prevents researchers from also using traditional tests of interaction effects. However, the addition of such family-level function estimation might yield more replicable findings that also lead to insights about "threshold effects" in the family system that could be very useful for clinical prevention and intervention or social, medical and educational policy (e.g., May and Bigelow 2005). What may matter just as much (or even more) than a specific two-way or three-way interaction effect in family members' ADHD symptoms, is that a certain threshold is exceeded in family-wide ADHD symptoms.

Another complementary methodological toolbox involves identification of replicable and clinically meaningful qualitative sub-groups of dyads, triads or families based on symptoms. This identification can be done in a fully exploratory way (using cluster, latent class, or latent profile analysis methods; e.g., McCutcheon 1987), or confirmatory algorithms can be used to specify a priori anticipated sub-groups of families (e.g., comparing a group of families for which mother-father, parent-child, or triad ADHD symptom types and levels are very similar, to a group of families with average differences in symptom profiles and a group with notable symptom discrepancies). These groups can then be compared on a host of other constructs of interest. Such an approach could be used, for instance, to test for some of the proposed family-system compensatory and match/mismatch concepts highlighted in several of the papers in the special section.

Turning to conceptualizations of the family system, the special section papers also inspire us as readers to continually reconsider the value of applying longstanding theorized family transactional models (e.g., Minuchin et al. 1975; Olson 2000) as heuristics for understanding ADHD symptoms, parenting, and intergenerational transmission. There are multiple examples of this already in the family ADHD literature, as demonstrated in most of the special section empirical papers and summarized in the review paper by Chronis-Tuscano et al. (2017). As another example of a family systems lens, my colleagues and I recently described a dyadic transactional model of each partner's

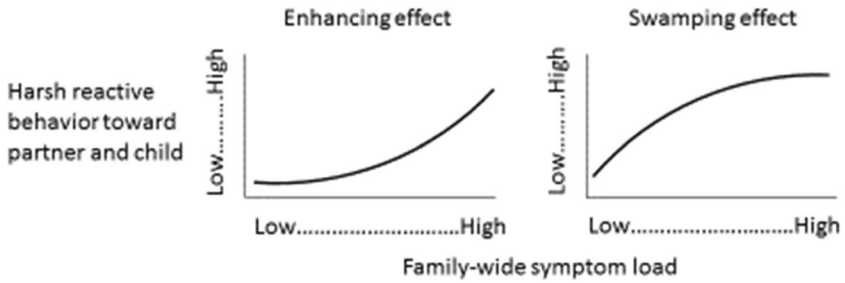

Fig. 1 ADHD symptoms and harsh reactive family relationship processes. This figure illustrates examples of two competing hypotheses regarding the family-wide level of ADHD (and possibly comorbid disorder) symptoms and distinct associations with variance in distressed, conflicted parent-parent, parent-child, and family triad relationships

noxious behavior as an elicitor of stress reactivity and self-regulation (including cognitive-affective, behavioral, and physiological) in the other. These effects work together to serve an ongoing relationship process that reinforces adaptive or maladaptive functioning (including symptoms of psychopathology) in both relationship partners (Deater-Deckard et al. 2016). Accordingly, ADHD symptoms themselves and their underlying endophenotypes (e.g., executive function deficits, physiological hypo- or hyper-reactivity) *in parents and children alike* play crucial roles in the etiology of chronic harsh reactive and insensitive parenting behavior, and growth over time in both dyad partners' symptoms. This kind of family process theorizing is evident in several of the current special section papers. Also of relevance regarding ADHD endophenotypes, a coinciding complementary two-issue special section at *Journal of Family Psychology* (2017, Volume 31, Issues 1–2) includes 11 studies of parental neurobiological and neurocognitive factors (e.g., inhibitory control, working memory, effortful control, heart rate reactivity as well as variability [vagal tone]) and their role in family processes and children's functioning. Although none examined ADHD symptoms specifically, most of those studies' results align with the studies in the current special section. Just like the child, the parent's ADHD symptoms and underlying deficits in executive function and physiological self-regulation influence each other in the parent-child dyad (and probably the parenting partner dyad as well). It is apparent and welcomed, that a "new norm" for family systems studies of ADHD and key neurobiological vulnerability factors, is for studies that place equal emphasis on individual and dyadic parent and youth symptoms, functioning, and relationships. Yet there is a caveat. The findings from multi-level family-wide empirical approaches present new challenges for translation in clinical contexts. It remains to be seen whether such approaches can be applied to improve the efficacy of existing individual and family-level intervention and prevention approaches.

Family Diversification of ADHD Symptoms: Gene-Environment Processes

Although it may not have been a stated goal of the authors in all of the papers, the empirical papers as a group (and a few of the papers quite intentionally) provide critically important data on the effect sizes pertaining to familial transmission in ADHD symptoms. Families are generators of diversity in ADHD and related symptoms—like most aspects of human variation, which arises from within (as well as between) each family. What is the evidence for this diversification? To examine this question, correlations between family members' ADHD symptoms from the largest relevant study to date (see Table 4 of Boomsma et al. 2010) along with relevant correlations from the current special section set of papers, are summarized in Fig. 2. The figure shows unweighted averages of correlations reported in each paper that I computed (often, averaged across dimensions of parent or child ADHD symptoms, across both parents, and across negativity and positivity scales in the parent-child relationship). Because they are distinct in various ways, twin-pair correlations (in Boomsma et al.) were not included in the estimates. The computed average correlations in Fig. 2 can be thought of as effect sizes, with larger positive or negative correlations approaching 1.0 indicating stronger effects, and correlations approaching 0.00 indicating weaker effects.

First, consider path "A" in Fig. 2—parenting partner similarity, estimated as the correlation between symptoms in mothers and fathers; a higher positive correlation would indicate greater partner similarity in symptoms, and a negative correlation would indicate partner differentiation. These data suggest that there is only modest selective pairing (i.e., assortative mating) of partners based on ADHD symptoms—either selecting someone more alike or more different from oneself. These potential selection effects must be taken into account when evaluating evidence of intergenerational transmission of symptoms, because selection on symptoms can systematically bias estimates of effect sizes in family studies (e.g., Loehlin et al. 2009). Fortunately, with an overall averaged effect size of 0.10, this does not appear to be a major concern for studies of family processes and family member ADHD symptoms. As many of the papers in the special section make clear, it is critically important to take into consideration *both* parents'

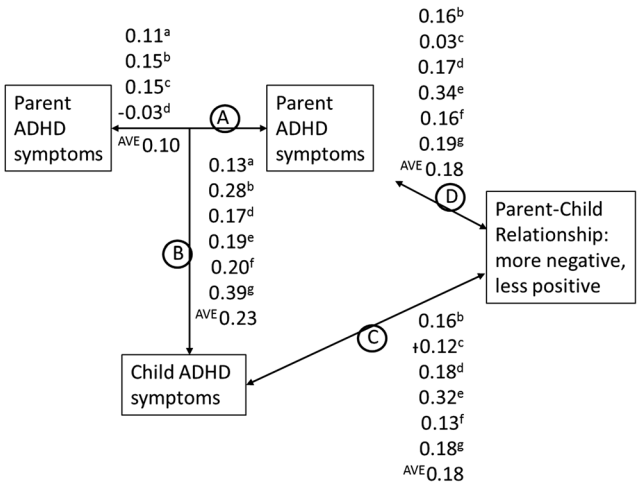

Fig. 2 Effect sizes for links between parent-parent-child ADHD symptoms and family relationship processes. This figure illustrates effect sizes (correlations or beta weights [†]) from [a] Boomsma et al. (2010) and current special section papers (all 2017): [b] Breaux et al., [c] Wymbs et al., [d] Williamson et al., [e] Auerbach et al., [f] Moroney et al., [g] Nikolas & Momany, and [AVE] unweighted average effect size. A = mother-father symptom similarity; B = parent-child symptom similarity; C = link between parenting environment and child symptoms; D = link between parenting environment and parent symptoms

ADHD symptoms, because on average, one parent's symptoms tell us little about the other's—and both contribute as part of a family system. I return to this crucial point later.

That partner selection is probably negligible for ADHD symptoms is not too surprising. Studies of related temperament and personality facets (e.g., activity, impulsivity, distractibility) tend to show similarly modest partner correlations (Watson et al. 2004). Perhaps far more surprising is the modest effect size found for parent-child similarity in ADHD symptoms—shown as path "B" in Fig. 2. Like path "A" (partner similarity in symptoms), a higher positive correlation would indicate greater parent-child similarity in symptoms, and a negative correlation would point to greater parent-child differentiation. These studies had an overall averaged correlation of 0.23. This effect size fits with existing studies of full siblings. For example, Boomsma et al. (2010) reported sibling similarity (excluding twin pairs) of 0.09 averaged across gender combinations. In Nikolas and Burt (2010), a meta-analysis of twin and adoptive sibling studies of ADHD reported modal sibling-similarity correlations in the 0.20 range for fraternal twins (with the range of similarity correlations in some studies including 0.00 and even slightly below 0.00). Indeed, some of the smallest (and sometimes *negative*) correlations for fraternal twin and full-sibling similarity are seen in parents' ratings of ADHD-related facets of temperament including attention, impulsivity, and activity level (Mullineaux et al. 2009).

How can this be? Heritability estimates for ADHD are not small—but this is because identical twin similarity is much more substantial. Importantly, this heritability effect includes additive and interactive effects of many genes and environmental inputs, reflecting gene-by-gene interaction effects and possibly unidentified gene-environment interaction effects as well (Mullineaux et al. 2009; Nikolas and Burt 2010). As several papers in the current section make clear, unique genotypes and unique experiences within the same family can contribute to very substantial differences between family members. This is also apparent when symptoms in extended-family members (e.g., brothers and sisters of parents, cousins of children) are considered; this information on extended family networks adds critical information for examining how the disorder and its comorbid conditions may "move through" and operate within family systems (Breaux et al. 2017). The emerging picture points to families as "differentiation generators"—through additive and interactive genetic and environmental mechanisms, including robust effects of differential parenting behavior toward sibling children that reinforces existing differences in behavior and functioning (Dunn and Plomin 1990).

Another major contribution of the special section is its examination of the links between parent and child ADHD, and the parenting environment. In the commentary, I refer globally to "parent-child relationship processes" indicated as higher levels of negative affect, conflict, and harsh control—and lower levels of sensitivity, warmth, and positive control. As reported in the literature reviews of all of the papers in the section, this overall pattern of parent-child relationship dynamics in families of youth with ADHD is well documented (represented as path "C" in Fig. 2). The current studies provide further evidence for that pattern, but also offer a crucial expansion of the literature by addressing parent ADHD symptoms and their potential role in these family processes (represented as path "D" in Fig. 2). The correlations shown in the figure again are the most global averaged estimates—as before, averaged across both parents and multiple dimensions of parent or child ADHD (when applicable), and dimensions of parenting (scaled such that a higher score corresponds with more negative and less positive parenting). As with other paths, a higher positive correlation would indicate that more ADHD symptoms are associated with more negative and less positive parent-child relationship processes; a larger negative correlation would indicate that more ADHD symptoms are linked with less negative and more positive parenting. In the current set of studies, the overall averaged correlation was 0.18 (for child ADHD and parent ADHD symptoms alike) with parent-child relationship processes, indicating a consistent and usually modest (depending on the study in question) association between more symptoms, and greater negativity or less positivity in the parent-child relationship.

It is worthwhile to pause and consider a potentially major problem for interpretation of the effects in paths C and D in Fig. 2. Specifically, because the effects are based almost entirely on correlational studies of genetically related parents and youth, it is plausible that the effects simply reflect the "background" genetic similarity of the parents and children (referred to as "passive gene-environment correlation), rather than reflecting social relationship and learning processes. Considering passive gene-environment correlation is important. Ignoring it leaves open the possibility that intervening on parenting and parent-child relationship processes only addresses another aspect of symptoms of family risk for ADHD, rather than addressing an actual social-relational cause of growth or decreases in symptoms over time and between generations.

Few of the current section's studies tested for passive gene-environment correlation explicitly, but there are several pieces of evidence suggesting that passive gene-environment correlation is not an issue. First, in the multiple current studies that found significant associations between parenting behavior and the parent's or child's ADHD symptoms, those associations usually were not substantially affected by statistically controlling for the child's or parent's (respectively) ADHD symptoms. If passive genetic correlation effects were explaining the results, statistically controlling for the other family members' symptoms would fully account for the observed associations between ADHD symptoms and parent-child relationship variables. Second, one of the longitudinal studies

(Moroney et al. 2017) found that parental negativity toward the child statistically mediated or accounted for the association between parent and child ADHD symptoms over time, while controlling for both individuals' prior symptoms. Again, if passive genetic correlation was explaining the results, this longitudinal predictive pattern would not be seen. Third, in a direct test for passive gene-environment correlation, Nikolas and Momany (2017) found no associations of variations in a specific candidate gene for ADHD symptoms (i.e., dopamine receptor 4), and variation in parenting behaviors. Fourth, Wymbs et al. (2017) used an experiment involving "confederate" child actors who played the role of a child with or without ADHD symptoms during observed interaction with couples (Wymbs et al. 2017). Their findings showed the expected link between child challenging behaviors and negative adult behavior toward the child (path C in Fig. 2), and then some. The average correlation was 0.40 between ADHD status of the confederate child actor, and greater negative or less positive parenting behavior across mothers and fathers. Because the adults and children in the observed interactions were genetically unrelated, the association between observed child symptoms and observed parenting behavior reflected social-behavioral influences between child and adults, rather than underlying passive gene-environment correlation effects. Similar results also have been reported in analyses of genetically unrelated children and teachers (Greene et al. 2002; Mejia and Hoglund 2016).

The special section also caused me to reconsider my prior collaborative work examining genetically related and unrelated (i.e., adoptive, foster) parent-child dyads (summarized in Deater-Deckard 2009). Those studies showed similar effects overall, for genetically related and unrelated (i.e., adoptive) dyad types. Furthermore, we found that the same mother's differential positive parenting toward her genetically identical twins was correlated with identical twin differences in levels of attentional control (with effect sizes in the 0.20 range, similar to effect sizes for path C in Fig. 2). Taken together—the modest parent-child similarity in ADHD symptoms (path B in Fig. 2), the similar effect sizes for associations between child symptoms and parenting in genetically related and unrelated dyads (or even larger effects for unrelated dyads), and the similar effect size for identical twin within-family differentiation in attention problems —the evidence suggests that passive gene-environment correlation effects are negligible.

Two of the special section's papers show examples of potential additive and interactive effects of measured genetic variations in dopamine transporter and receptor-4 genes (Auerbach et al. 2017; Nikolas and Momany 2017). As these and several other papers in the section emphasize, the future of family research on parent and child ADHD and comorbid conditions will need to include adequately powered studies of gene-by-gene and gene-by-environment interaction effects. Similarly, there is a growing need for studies of the very earliest intergenerational transmission processes that begin prior to conception, and that influence fetal and post-natal child development. In an adult romantic relationship in which one or both partners have ADHD, some aspects of their individual and dyadic functioning that affect parenting after a child is born or is adopted, may have existed prior to the child's arrival. For example, consider the family context during pregnancy. ADHD and comorbid symptoms in family members can contribute to stress in the pregnant mother, and that stress can have major consequences for the neurological development of the fetus. This is because exposure to high levels of stress hormones in utero can lead to "epigenetic" modifications of DNA (i.e., molecular alterations to the structures surrounding the DNA molecule that influence its functioning). These epigenetic changes can have lasting effects on gene expression in ways that modify the developing structures and functional effectiveness of the young child's nervous system (Nigg 2016). Epigenetic alterations can increase risk for hypo- or hyper-reactivity, deficits in self-regulation, and psychopathology (Mulder et al. in press; Neuenschwander and Oberlander in press). Over time after the arrival of the child, the stress from problems in the couple's and parent-child relationships in the family can further exacerbate prenatal and post-natal epigenetic alterations of the child's developing neurobiology. In future research, it will be increasingly important to also include pre-conception and prenatal time periods and family relationship contexts, as our field strives to better understand families and ADHD as interweaving systems.

Conclusion

Although translating empirical findings from family-wide studies of ADHD to clinical practice will be a major challenge, testing competing hypotheses about familial transmission matters. In practice, when working with families while holding incorrect but testable and refutable assumptions unnecessarily increases the odds of failure in prevention and intervention. Elucidating all of the many complex bioecological family system processes will be arduous, but it is necessary. For instance, the monoamine neurotransmitter system includes dopamine, serotonin, and epinephrine and norepinephrine (among other molecules). There are numerous candidate genes in relevant regions of DNA, with many common and rare variations that are still being discovered. Many of these variations are implicated in the etiology of ADHD and relevant underlying risk factors (e.g., executive function deficit, hyperactivity, impulsivity; for examples of meta-analyses see Barnes et al. 2011; Neale et al. 2010). In future, the family system of environments and genetic risks increasingly will be operationalized with greater precision, and with an eye toward supporting meta-analytic statistical approaches that integrate findings across

many studies, as well as community and clinical contexts (Le Novère 2015; Lee 2015). In the face of this complexity, the theory and empirically tested models presented in the current special section move us another step forward as we strive to continually improve the rigor and replicability of the evidence in our developmental clinical science.

Compliance with Ethical Standards

Conflict of Interest The author declares that he has no financial conflict of interest.

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