Advancing the Science of Biosocial Transactions Related to Aggression in Children and Young People: A Brief Review and Steps Forward

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Abstract

Aggression towards others produces great physical, emotional, and economic burden. An extensive body of research identifies risk factors related to aggression that span biological and environmental factors. However, much of that work identifies risk factors in isolation (or among only a few other risk factors) even though the development of aggression is a complex phenomenon involving interactions among risk and protective factors across time and across levels of analysis. The goal of this piece is to identify themes in the literature to articulate five practical steps needed to advance the science of biosocial transactions related to the development and maintenance of aggression in children and young people. Specifically, we highlight key biological (brain, genes) and psychosocial (parenting, peers) domains in aggression research as we comment on ways to improve the measurement of and quantitative methods in the study of neurocognitive process and environments related to aggression. We also discuss the science of intervention within a transactional model, highlighting the need for the science of behavior change to develop from a mechanistic framework. Our understanding of aggression is poised for transformation. We are in a position to integrate biosocial insights in ways that allow us to specify mechanisms and better understand transactional relationships that inform how and why some children grow up to display aggression.

Key words: children and young people, aggression, neurobiology, environment, genes

Aggression towards others is a heterogeneous construct that includes a wide range of behaviors, including yelling, throwing things, and physical violence. Aggressive behaviors are ubiquitous; exhibited across different developmental stages, genders, and cultures, and these behaviors are part of the diagnosis of multiple forms of mental illness. Aggression, particularly at extreme, chronic, and non-normative levels, produces great physical, emotional, and economic burden for the perpetrators themselves via altered developmental trajectories (e.g., school failure, incarceration), but also has major impacts on victims, family members, and society at-large. For example, approximately 5% of all 12–18-year-old adolescents in the US are afraid of being attacked or physically harmed at school (McFarland et al., 2019). In 2020, there were about 100,000 serious violent crimes committed by young people between the ages of 12 and 17 in the United States (Puzzanchera, 2022). Aggression and early disruptive behavior are the most common reason for referral for treatment in children and young people (Cushing et al., 2023). Moreover, compared to other disruptive behaviors (e.g., rule breaking), aggression tends to start earlier in life, be more highly heritable, and have a more chronic trajectory (Burt, 2009). The pervasiveness of these behaviors highlights the importance of identifying the specific factors that are etiologically related to the onset and maintenance of aggression.

An extensive body of research identifies risk factors related to aggression that span biological and environmental factors (see Allen et al., 2018 for reviews; Blair et al., 2006; Lansford, 2018; Wagels et al., 2022). However, much of that work identifies risk factors in isolation (or among only a few other risk factors) even though the development of aggression is a complex phenomenon involving interactions among a multitude of risk and protective factors across time and across different levels of analysis. The goal of this piece is to articulate practical steps needed to advance the science of biosocial transactions related to the development and maintenance of aggression in children and young people. We provide a selective review of neurobiological (genetic and brain; *NB:* other pieces in this special issue goes into detail about the science of hormones and aggression) and social factors (parenting, peers, community violence, poverty) that have been established in the aggression literature, with the purpose of advancing our discussion of transactional models. In doing so, we hope to better highlight the gaps in the current literature and directions for future inquiry. Given the immense burden associated with more extreme forms of aggressive behavior (i.e., non-normative aggression), our review is focused on the neurobiological and social factors that have been studied in the context of violence and antisocial behavior (aggression being one expression of antisocial behavior). Research in this area has not consistently separated out subtypes of aggressive behavior (e.g., premeditated-proactive aggression vs. frustrative-reactive aggression), and there is some debate about the utility of these subtypes of aggression when focused on more extreme behaviors (Evans et al., 2020; Polman et al., 2007; Smeets et al., 2016; Yang et al., 2016), therefore, most of this review is focused on the broad construct of aggression and on work that can be fit together at multiple levels in a transactional model.

Brief Background Review of Biosocial Factors Related to Aggression in Children and Young People

Thoughtful theories and existing empirical research provide a foundation for understanding the factors related to aggression. Much of this work has been separated into work specifying the neurobiology of aggression and antisocial behavior (Blair, 2001) or the environmental pathways to aggression (Dishion & Patterson, 2006).

Neurobiological theories of aggression in children and young people have emphasized the role of the amygdala and corticolimbic circuit, as well as the role of prefrontal regulatory (executive) control

regions (Blair, 2016; Blair et al., 2006; Wagels et al., 2022). Moreover, emerging evidence suggests that reward-related regions also may be implicated more broadly in antisocial behavior and specifically in aggression through learning mechanisms (Murray et al., 2018). The corticolimbic circuit, with the amygdala as its anchor, has received the most attention with extensive animal models linking reactive aggression to a circuit from the periaqueductal gray to hypothalamus to amygdala and medial and orbital frontal cortex (Blair et al., 2006; Bertsch et al., 2020). Ultimately, the most promising current circuits of interest for aggression include to corticolimbic, corticostriatal, and prefrontal control. Though these circuits have roles in reactivity to threat and emotion, processing rewards and learning, and response inhibition, respectively, their roles and functioning overlap substantially. Functional and structural neuroimaging studies have linked these circuits to human aggression and broader antisocial behaviors (Westerman et al., 2023). At the same time, there are thorny issues in these circuits including some lack of replication about the direction of findings, potential for a biased focused on these areas (to the exclusion of others), and the need to move to research that focuses more on large-scale brain networks (e.g., see work on the triple network theory; Menon, 2011).

There also is research identifying environmental exposures that increase risk for the development and maintenance of aggression in children and young people (Lansford, 2018; Valois et al., 2002). One of the most heavily studied environmental risks for aggression is parenting. Parenting that is harsh and inconsistent has been linked robustly to the development of aggression (Kang & Guo, 2022; Masud et al., 2019; Reid & Patterson, 1989; Reid et al., 2002; Romano et al., 2005). Harsh parenting is thought to model aggression for children and young people, to undermine their ability to develop emotion regulation skills necessary for related constructs such as empathy, and inconsistency in parenting is thought to lead to reward contingencies that make aggression useful in some contexts. Beyond parental influences, interactions with deviant and aggressive peers have been studied extensively as a risk factor for aggression (as well as broader related behaviors like antisocial behavior and substance use) (Burnside et al., 2018; Monahan et al., 2009; Quarmley et al., 2022). The influence of peers may be particularly important during adolescence when children gain more independence. Deviant peers have been shown to reward children and young people for using aggression and to teach them that aggression is valued. Another risk factor is exposure to community violence (Anderson, 1994; Beyers et al., 2001; Durant et al., 1994; Haynie et al., 2006; Javdani et al., 2014; Miller & Tolan, 2019; Pabayo et al., 2014). These exposures provide observational learning that aggression can be useful, but are also stressful and scary, which can lead to increase in vigilance and reactions to threat (Estrada et al., 2020). It is important to note that these and other risk factors are typically clustered together, meaning that children and young people are often exposed to multiple risk factors (Bronfenbrenner & Ceci, 1994; Hyde et al., 2020; Hyde et al., 2022). These multi-exposures often come about because of broader structural factors (e.g., poverty) which are established risk factors for aggression and also increase the risk of exposure to other risk factors (e.g., poverty stresses parenting leading to greater exposure to harsh parenting, lower-income neighborhoods are more likely to have community violence).

Some of the environmental risk factors (e.g., parenting, community violence, poverty) have been related to the structure and function of aggression-related neural circuits. For example, harsh parenting has been associated with altered structure and function of the amygdala (Farber et al., 2022; Hyde et al., 2022). Similarly, exposure to community violence has been recently linked to greater responses to threat in the amygdala (Hyde et al., 2022; Suarez et al., 2022). Moreover, poverty, has been linked to altered structure and function in corticolimbic circuits, corticostriatal circuits, and prefrontal cortex related to inhibitory control (Johnson et al., 2016; Palacios-Barrios & Hanson, 2019). Thus, emerging studies

suggest that one way these environments may increase risk for aggression is by altering the development of these key neural circuits. For example, a recent paper showed that harsh parenting in early childhood predicted altered threat-related amygdala reactivity in early adulthood, which in turn predicted symptoms of antisocial personality disorder, an adult disorder marked by aggression (Gard et al., 2017). However, research on environment-brain relationships in the study of aggression is fairly limited. To build on this work, it is important for researchers to re-imagine ways to capture the complex transactions between the person, neurobiology, and their environment. Here we outline five methodological domains where researchers can take steps to advance the study of aggression.

Practical Steps to Advance the Science of and Intervention on Aggression in Children and Young People

Step 1: Improving cross-species study of neurocognitive processes underlying aggression

Aggression has been widely studied using animal models (see Haller, 2022 for review) and these models can inform our understanding of genetic, environmental, and neural contributions to the development and maintenance of aggression, as well as specifying, with greater causal inference, how these factors fit together across development. Many animal studies have focused on behavior associated with defending territory or pups, but aggression in such circumstances can be considered as functional and adaptive and these paradigms may not offer the optimal set-up for trying to understand pathological forms of aggression. The psychopathology-oriented models of animal aggression have focused on systematically studying genetic and environmental adversity conditions that give rise to increased risk of aggressive behavior and have highlighted the causal role of both. However, what we are still lacking is systematic development of homologous task assays for aggression for animals and humans, which would enable us to better understand the neurocognitive mechanisms associated with risk for aggression and how these shape and are shaped by the social environment. Animal models naturally enable a deeper mapping of circuits supporting task behavior and afford causal inference. If task assays are comparable across animals and humans, neural signatures identified in animal studies also can be tested in humans using non-invasive neuroimaging. Experimental manipulations on animals can constrain the search space for future behavioral, pharmacological, or neurofeedback/neurostimulation interventions in humans. Human studies, in contrast, can be used to study the identified neurocognitive mechanisms in richer contexts than what is possible to generate in animal models. The promise of this approach is beginning to be realized in relation to other psychiatric phenotypes. For example, recent work has demonstrated abnormal belief updating and hallucination-like perception in a rodent model (Schmack et al., 2021), and parallel human and rodent work has shown that and that inference, when measured using species-appropriate tasks, is awry in schizophrenia (Barron et al., 2020; Nour et al., 2021).

Step 2: Improving measurement of aggression-related cognitions and environments in humans

Measuring Neurocognition. There is currently no agreed set of paradigms for assessing the neurocognitive processes underlying risk for aggression. The extant tasks vary in task demands and the precise processes that they tap into. This means that it is difficult to compare findings between studies or conduct meaningful meta-analyses, as we cannot be confident that different studies are capturing the same constructs. It would benefit the field to generate a set of commonly agreed measures. These could include measures of emotion processing and empathy, as well as a set of reinforcement learning and executive function paradigms. Further it is important to find paradigms that could be used across different developmental stages to assess comparable neurocognitive processes (Pezzoli et al., 2023; Sanislow et al.,

2022), which are of course themselves changing over time. We need more work validating age-adapted measures of the same constructs and examine their associations with other measures.

Such measures would also need to undergo rigorous psychometric validation – something which is not currently the case for the majority of experimental and functional neuroimaging measures (e.g. Pezzoli et al., 2023; Sanislow et al., 2022). Without psychometric validation, neurocognitive paradigms cannot be deployed in a longitudinal framework to study individual differences in transactional risk trajectories. The majority of currently used experimental and fMRI tasks stem from a research tradition where tasks are typically designed to minimize between individual variation and to capture effects across all humans or within a specific group – rather than to sensitively and reliably capture individual differences (Elliott et al., 2020; Pezzoli et al., 2023). Developing paradigms that can isolate particular cognitive-affective mechanisms and their neural correlates, and sensitively quantify variation in them, will increase the precision with which we can map neurobiological risk for aggression. Further, combining neuroimaging with other biological measures (e.g., EEG, fNIRS, peripheral physiology) may provide a richer way to uncover individual differences in neurobiological mechanisms.

Measuring Environment. It is of course not sufficient to just pay attention to the experimental and neuroimaging paradigms that we use. We also will need sensitive measurement of the social environment. Children and young people exposed to one risk factor for aggression are likely to be exposed to many (Estrada & Baskin-Sommers, 2023; Estrada et al., 2021; Leventhal et al., 2009). For instance, the family stress model posits that poverty and economic instability stress parents, undermining their emotional resources, and leading to greater family conflict, and eventually harsher parenting (and child aggression). This model has been supported by a host of empirical work (Conger & Donnellan, 2007; Conger et al., 2002; Gard et al., 2020; McLoyd, 1990), highlighting that broader structures play a role in shaping the proximal environment for the developing child. Further, deviant peer influence may amplify the effects of earlier harsh parenting and children and young people engaged with deviant peers may learn new ways to undermine the parent-child relationship to provide less oversight of the child's activities (Patterson et al., 1989).

In order to capture the complex interactions among environmental experiences leveraging observational, experience sampling, social network, and virtual reality approaches may be useful (see e.g., Low et al., 2013; Moore et al., 2019; Odgers & Russell, 2017; Russell et al., 2016; Verhoef et al., 2021). Though there exist some good measures in each of these domains, they have not been combined together, particularly not with neuroimaging.

Measurement Timescales**.** Moreover, our current measures largely provide static snapshots, as opposed to more dynamic indices of environment and neurocognition. For example, dense sampling of environmental contingencies and child cognitions at shorter time intervals could provide rich information on how particular interactions unfold and reinforce and are reinforced by cognitions. These measurements could be combined with brain imaging at very short time intervals to test true notions of the biosocial transactions at these shorter timescales (shorter relative to the year-to-year frequency of most longitudinal studies). Additionally, hyperscanning, i.e., measuring the activity of multiple brains simultaneously, using fMRI, M/EEG, and fNIRS can provide estimates of real-time dynamics between two or more interacting brains (Babiloni & Astolfi, 2014; Czeszumski et al., 2020). Combining hyperscanning with broader measures of the child's social environment can highlight how the environment may shape neurobiological factors and contribute to social behavior as it is measured in 'real time'. Alternatively, virtual reality could be used to construct very specific environmental conditions (with individual or dyadic interactions) and study their impact on behavior in real time, which could be connected to neurobiological assessment.

Step 3: Utilizing genetically informed designs

Linking Measured Gene to the Brain and to Aggression. Much of molecular genetic research has focused more broadly antisocial behavior, rather than on aggression specifically. However, this research has shown that aggression, as an expression of antisocial behavior, is moderately to strongly heritable (Burt, 2009; Rhee & Waldman, 2002; Tuvblad & Baker, 2011) - thus genes likely play a substantial role in conferring vulnerability to individual differences in aggression. Molecular genetics studies have focused especially on genes related to serotonin transmission, given the evidence from animal models linking serotonin to aggression (Baker et al., 2006; Nelson, 2005). Studies also have explored genetic variation related to other neurotransmitters including dopamine and related neuropeptides like oxytocin, given the role that dopamine plays in reward and learning and oxytocins role in bonding and potentially prosocial behavior. These neuromodulators also are key for signaling in the neural circuits related to aggression (e.g., serotonin receptors are dense in the amygdala). There is limited data linking candidate genes to brain phenotypes, but some prior work has shown that genes related to serotonin have been related to structure and function of the corticolimbic circuit and genes related to dopamine transmission have been related to corticostriatal circuit structure and functioning (Holmes, 2008).

Although this research has been promising, most of it has focused on individual candidate genes and little has replicated consistently across time (findings for broader antisocial phenotypes also are limited). Existing genomewide association studies have not returned 'hits' that correspond with candidate gene studies, for example a recent large-scale genome-wide association meta-analysis on childhood aggression (with sample size of nearly 90 000 individuals) returned very few 'hits', and these were in genes previously associated with educational attainment (ST3GAL3, PCDH7, IPO13) accounting for less than 1% of variance in aggression (Gard et al., 2019; Ip et al., 2021; Tielbeek et al., 2017; Viding & McCrory, 2012). Rather than specific genes or even small concerts of genes, aggression is likely due to both additive and interactive effects of a large number of genes, each of which has a very small effect individually. The upshot is that previous quantitative genetic research tells us that genes are important, but we need larger scale, molecular genetic investigations to identify the specific genes that increase risk of developing aggressive behaviors. It is likely that these genes have an effect via shaping the neural circuits related to aggression, though in concert with risky environments.

Gene-Environment Correlation. A challenge in this area is the potential presence of gene-environment correlations. Research on parenting and aggression helps to illustrate this challenge. There are a number of observational studies that have replicated the finding that harsh parenting is associated with elevated levels of child aggression (Patterson et al., 1989; Pinquart, 2017; Reid et al., 2002; Shaw et al., 2003). It could be that harsh parenting causes aggression by modeling aggression, by undermining children's capacity to develop emotion regulation and empathy skills, and also may be inadvertently rewarding children for their use of aggression (Patterson et al., 1989). At the same time, it could be simply that parents who are more aggressive have genetic vulnerabilities to difficult temperament and behavior, which they pass onto their children. These children then are more aggressive via genetic processes and have parents who are harsher and more aggressive than the population average. If this is the case, the association between the harsh parenting and child aggression could be, in part, due to genetic confounding, rather than a product of environmental causation. This sort of confound is described as a passive gene-environment correlation-- the child's genes are related to the environment being provided. An alternative example could be that children who are more difficult in temperament from birth onwards (because of the genes they inherited) evoke harsher parenting because they are more challenging to

parent, a process termed evocative gene-environment correlation. In this case the child's genes evoke a different environment. Outside the family context, as children grow up, they also may actively seek out environments where they can behave aggressively. These could include joining social groupings or gangs that provide opportunities for fighting or other forms of releasing aggression. Again, before we can conclude that the peer context causally increases the risk of aggression (e.g., via peer deviancy training; Dishion & Patternson, 2015), we would have to rule out the possibility of genetic confounding, termed active gene-environment correlation (in this case, separating out selection of peers versus peer influence).

There only are a very limited number of studies that have examined aggression specifically, in relation to gene-environment correlation (but see DiLalla & DiLalla, 2018 for a demonstration of evocative gene-environment correlation in the context of peer play). There is a more substantive body of evidence regarding broader antisocial behavior phenotypes (which include aggressive behaviors). These studies (utilizing twin and adoption data) have demonstrated the presence of passive-, evocative-, and active gene-environment correlations in relation to parenting and peer variables and antisocial outcomes (Jaffee & Price, 2008; Moffitt, 2005; TenEyck & Barnes, 2015). As an example, twin modeling has indicated that a common genetic factor accounts for most of the observed relationship between children's antisocial behavior and their experiences of corporal punishment (Jaffee et al., 2004). Thus, researchers must be aware that observational studies are not necessarily providing causal evidence. At the same time, genetically informative studies also provide the most convincing evidence for causality of environmental influences and in most cases genetic confounding is not sufficient to explain all the association between environmental variables and child outcomes. For example, there is evidence from twin and adoption studies that some of the association between harsh parenting and child aggression or antisocial behavior is due to non-heritable/environmental factors (Burt et al., 2021; Moffitt, 2005; Sellers et al., 2020).
There are also data from genetically informed studies that suggest that the etiology of antisocial and aggressive behavior can vary as a function of the environment (Burt, 2022). When there is more environmental risk, aggression may be less heritable. This phenomenon has been explained by the "social push" hypothesis, which posits that when there are more and stronger environmental pushes towards aggression (e.g., harsh parenting in the context of deviant peers and widespread exposure to violence), genetic variation may matter less. In the presence of the risk environments, more children and young people are being pushed toward aggression, so their individual differences in genetic risk for aggression matters less (Burt & Klump, 2014; Burt, 2015; Raine, 2002). However, gene-environment correlation is not always modeled in these studies and future genetically informed work that has the power to triangulate on different mechanisms of gene-environment interplay simultaneously will be important as the field goes forward.

In summary, the role of genetic and environmental influences are challenging to separate and are intimately intertwined. As we endeavor to increase our understanding of how aggression develops and the neurobiological processes involved, we need to study environmental processes within genetically informed study designs. This way we can develop a better understanding of whether environmental processes are genuinely causal, or instead, in part, an epiphenomenon of familial risk. The implications of finding genetic confounding are not that environments do not matter or that the interventions should move away from addressing the 'environment'. Instead, they can tell us something about how individuals co-create particular environments and should encourage us to work around particular profiles of strengths and difficulties in both the parents and the children.

Step 4: Improving quantitative methods that capture complex systems

Based on the existing research, we can stitch together various studies to support a compelling conceptual model of the development of aggression from genes and environments to neurobiology. These types of models have been offered for how environments shape child behavior (Conger & Donnellan, 2007; Patterson et al., 1989), and recent neurobiological research suggests that brain structure and function may fit well within these frameworks (Hyde et al., 2020). However, this relatively simplistic model is only supported in the broadest sense with little research testing multiple components of the model in the same study (e.g., almost no studies have included environment, brain, and aggression in a single study). The complexity aggression requires that we test a multi-level transactional model (Bronfenbrenner & Ceci, 1994; Bronfenbrenner & Morris, 2007; Sameroff, 2010; Sameroff, 2000).

A seminal example of work focusing on transactions in the development of aggression is work by Patterson and Dishion (Dishion & Patterson, 2006; Shaw et al., 2003). First, in early childhood, children with a more difficult temperament may be more challenging to parent and elicit more harsh and inconsistent parenting. This harsh parenting undermines the development of prosocial behaviors and skills and children tend to reward the parent for harsh parenting (by stopping their behavior temporarily) only to resume later (leading to greater escalation of behavior by both child and parent). These micro learning contexts shape parents to be harsher, but also less consistent (ultimately withdrawing from the interactions that are increasingly aversive), while showing the child that escalating their behavior can work for them to achieve their goals. The child brings this learning history to school where they begin these types of cycles with peers and teachers, leading to trouble in school, rejection from prosocial peers, and commitment to a deviant peer group. In turn, as these children and young people select deviant peers, their peers also model and reward deviant behavior (Burt, McGue & Iacono, 2009), leading to greater escalations in aggression and other antisocial behaviors and further reinforcement learning about the rewards of such behavior in context.

Though this work has been very influential in research on developmental psychopathology, it has focused relatively narrowly on learning mechanisms. Furthermore, the transactional models have not been widely deployed in more biologically focused work on aggression, nor in neuroscience. Thus, we posit here that we need more work examining the brain as embedded within these types of ongoing transactions that occur within hierarchical and clustered settings of risk for understanding the neurobiology of this behavior (Viding et al., 2023) (see Figure 1).

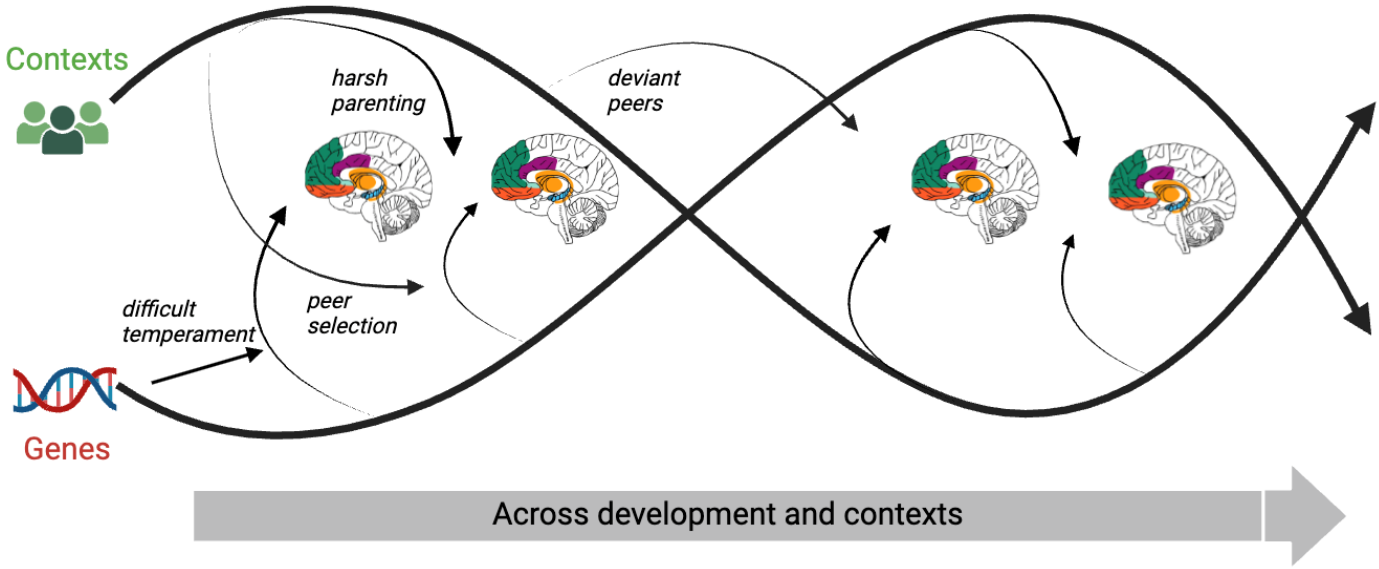

Figure 1. Transactional model for understanding the development of the neurobiology of aggression. From the outset, children are endowed with variable, partly genetically influenced traits and propensities, which mean that different children evoke different reactions from the environment and seek our different environments. These transactions build on each other to cluster more and more risks together as the child's developmental trajectory unfolds. These transactions shape the developing brain in ways that may be adaptive to the current and unfolding context of the child but may not be well suited to the broader societal context as aggression escalates. Green=corticolimbic (socioemotional processing); Purple=executive control (self-regulation); Orange=corticostriatal (reward processing)

Longitudinal Modeling. To identify and test complex transactional models of the development of aggression across levels and time, will require longitudinal biosocial designs that allow for estimation of development across stages, time, and/or periods. Most of the extant neuroimaging work has treated the neural differences as static (potentially genetic and early emerging), rather than emergent phenomena, and have either not examined their role in predicting future aggression or have done so within a unidirectional framework. With the increase in population neuroscience datasets (e.g., Adolescent Brain Cognitive Development Study; Healthy Brain Network), researchers can employ various forms of structural equation modeling (e.g., cross-lagged panel model, latent change score modeling) to examine changes in the environment, brain, and aggressive behavior across time.

It also would be useful for researchers to increasingly model the co-occurring relationships among variables (including neuroimaging data). Group-based multi-trajectory modeling is a form of latent class growth analysis that identifies subgroups of individuals who show similar patterns of change over time on multiple variables simultaneously (Nagin et al., 2018). This approach is an extension of the dual-trajectory approach, which estimates the association between two variables by measuring the probability of a trajectory for the first variable given membership in a specific trajectory for the second variable. In contrast, the group-based multi-trajectory approach examines the association by defining trajectory groups based on patterns of change for multiple variables simultaneously. For example, this modeling technique would allow for estimation of co-occurring change in neurocognitive performance, executive control network functioning, and parenting environmental factors that span risk and protective factors. Though trajectory modeling with aggression as a single variable has been used extensively (e.g.,

Broidy et al., 2003; Girard et al., 2019; Jennings & Reingle, 2012), much less work has modeled joint trajectories, and no work has examined multiple co-occurring trajectories. Examining co-occurring trajectories would provide strong evidence that factors theoretically integral to aggressive behavior empirically align.

As children and young people develop, is it important not only to explore how risk and protective factors change together, but also to identify critical transition points. The concept of tipping points, which originated in physics and has been used in disciplines such as environmental science and sociology (Lenton, 2013), may be useful in the study of aggression. We know that aggressive behavior can fluctuate across situations and across time for a given individual. A big question remains, though, about what small perturbation in the social environment might trigger a larger response that shifts both the internal state (brain) and external world (social environment)? For example, a move to a new neighborhood or the introduction of a new positive adult figure in the children and young people's life can alter behavior. Do those small agita lead to shifts in neurobiological and/or behavioral changes? That is, just as we know that risk can compound on itself, creating fewer new options, are there times and experiences that can shift the whole system/trajectory of child development to a new state? And what experiences might just reflect stochastic effects? At another level, in biology and sociology, tipping points associated with changes in the ecological system, such as transitions in neighborhood racial composition or violent infighting, can provide feedback that can impact individual and collective behaviors. An open question is then how and when do socio-structural shifts impact the development and change in individual behavior?

Alternatively, when are points when shifting to new trajectories is particularly difficult? We know very little about how behavior becomes canalized (for better or worse) and how, when, and what type of tipping points can shift individual development into new options and systems of behavior. Levering longitudinal data and applying segmented or 'piecewise' regression or inflection point analyses can be useful in advancing our understanding of how aggressive behavior grows and changes within children and young people. The use and application of complexity theory (Taylor, 2001) and quantitative approaches from systems theories (Smith & Thelen, 2003) will be key towards understanding these complex transitions and how intervention can help promote these shifts.

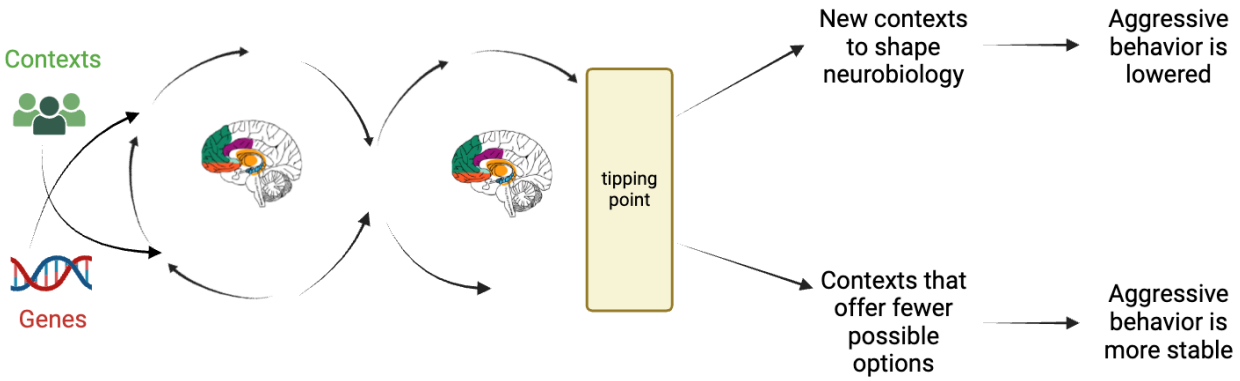

Figure 2. Illustration of tipping points. Tipping point theory seeks to identify what perturbations in the social environment might trigger shifts in the internal state (brain) and the external world (environment). Exploring at what point in time and what factors might lead to a shift (i.e., tip) in brain development and behavior will be useful in advancing our understanding of aggressive behavior. Green=corticolimbic (socioemotional processing); Purple=executive control (self-regulation); Orange=corticostriatal (reward processing)

Bayesian Modeling of Cross-Sectional Experimental Data. Longitudinal designs and associated analyses are important in capturing developmental change. However, these approaches also are quite costly. Moreover, changes are happening at multiple time scales (e.g., from moments to days to weeks to years) and no longitudinal study can capture all these timescales well. Further, it is debatable whether we have a full picture of the mechanisms that support aggressive behavior, and how those mechanisms interact with environmental systems. Therefore, there are ways in which our experimental or cross-sectional research can benefit from some re-analysis that leverages the advances in statistical modeling.

Existing research has clearly identified neurobiological and environmental factors that contribute to aggressive behavior. Now, it is important to develop a multi-level transactional model of the mechanisms that lead to the development and maintenance of such behavior. However, to estimate these transactions, new methods are needed to handle multiple types of data and estimate their interactions within individuals. One promising approach is to use forms of Bayesian latent profile analysis to estimate person-centered profiles of factors relating to aggression. Bayesian methods for latent profile analysis have fewer assumptions than conventional latent profile analysis. They also have been shown to maximize the identification of distinct, but nuanced, within-person variation in a set of variables (Paskewitz et al., *revise and resubmit*; Ruiz et al., 2023). For example, researchers can combine multiple measures of environment (e.g., family income, exposure to community violence, peer interactions) and neurocognition (e.g., amygdala reactivity on a threat task, performance on a working memory or response inhibition task) to identify subgroups of individuals who express these measures in distinct ways and relate these subgroups to aggressive behavior. These types of methods can help address the challenge of finding novel ways to use data on environment and neurocognitive functioning to better describe the individual (Brazil et al., 2018; Ruiz et al., 2023). Such advances are particularly relevant given the call for person-centered diagnostics and tailored interventions for individuals showing these burdensome behaviors.

Another way Bayesian modeling can help uncover more specific mechanisms in existing data is to move beyond reaction time or accuracy estimates as key indicators of performance. Bayesian computational models can provide rich information about how individuals incorporate prior assumptions and integrate probabilities in their perception, learning, and decision-making. More specifically, in a Bayesian framework, a person's beliefs about relevant variables in their environment (e.g. whether a given person will hit them, the reward or punishment following an aggressive action) are modeled as probability distributions. Inferences about the current state are based on a combination of a prior distribution (representing beliefs already held) and the likelihood (current information). The prior distribution is continually updated based on new experiences, forming a natural representation for learning (Van de Schoot et al., 2014). Additionally, it is possible to model within the Bayesian framework how volatility/stability and predictability of the environment impacts individual behavior.

A Bayesian framework can help translate transactional theories about aggressive behavior into computational terms that provide insight into precise neurocognitive mechanisms that underlie certain behaviors (Smeijers et al., 2019). For example, using a Bayesian approach it is possible to measure individual differences in prior beliefs from trial-by-trial behavior. Perception can be modeled as a Bayesian inference process, in which prior beliefs influence the interpretation of an otherwise ambiguous stimulus (e.g., Sterzer et al., 2018). Thus, hostile attributions can be understood as developed from a prior belief that people will be hostile, which would influence their categorization of people as hostile or not hostile. Using this approach may result in a better understanding of the underlying causes of hostile interpretations and aggressive behavior. This, in turn, might be beneficial for clinical practice as it would

be possible to develop and target interventions more specifically towards the individual patient's needs (Brazil et al., 2018). As another example, basic Bayesian learning models weigh new information based on the variance of outcomes: if an outcome is highly variable then its associations are updated more slowly (Van de Schoot et al., 2014). For example, if discipline is not predictable, then a Bayesian learner will infer that it has a large variance. This will make it more difficult to associate certain actions - such as aggressive behavior - with punishment. Thus, harsh and inconsistent parenting might make a child less sensitive to punishments because of how information is weighed. Thus, re-analyzing existing data within a Bayesian framework might allow researchers to identify more precise mechanisms that subserve information processing and result in aggressive behavior. This precision can be translated into increasingly individualized interventions.

Step 5: Situating the science of intervention within a transactional model

Interventions for aggressive and disruptive behavior have remained relatively stagnant for decades, although individuals with these problems are at risk of dying early and experience poor quality of life - with impairments across mental, physical, educational, and social domains extending throughout the life course (Baskin-Sommers et al., 2022; De Brito et al., 2021; Fairchild et al., 2019; Scott et al., 2001). This illustrates that preventing and treating aggression is challenging (though incredibly important). This conclusion leads to several important considerations and next directions for treatment.

First, we already possess an array of relatively effective treatments and prevention strategies (Baskin-Sommers et al., 2022; Lochman et al., 2019; Michelson et al., 2013). They may not work for all children and young people, but they can for some children and young people (e.g., Kazdin, 1997). And, generally speaking, the interventions that work best take into account biosocial transactions (e.g., addressing parenting and behavioral control issues) that exist within and around the child. The problem is that many of the children and young people and families that need these interventions and preventative interventions do not receive them for an array of reasons. Clinicians may not be trained or have high fidelity to this evidence-based practice. The treatments may not be available at all because there are shortages of clinicians, available clinicians are not trained, or there is no structural support for implementing these interventions. For example, insurance may not reimburse for these treatments or families needing these treatments may not have insurance (e.g., in the US) or the waitlist may be long for access. Beyond these issues, families and children that need these treatments the most may not seek care. Parents may not see that their child is exhibiting problematic behavior (or may see it but not think it is problematic), they may not trust treatment providers or the system more broadly, they may be pessimistic about whether treatment will help, and/or they may be faced with so much adversity that these specific child treatment needs are not a high priority (especially when treatment may be hard to obtain logistically or financially). Thus, even to the extent that treatment exists, it may not reach those who need it most.

Second, as scholars spend time debating the relative heritability of environments, this work misses the point that highly heritable outcomes can be influenced by the environment (and similarly behaviors with neurobiological correlates are not immutable). Thus, heritability tells us little about whether treatment can work or what type of treatment works. However, as described above, understanding heritability and gene-environment correlation can help us understand how the system can become more fixed as children and young people age, suggesting that earlier intervention may be more effective if it can target a system that is less entrenched. Moreover, this work can highlight the need for more intensive, longer, and/or multi-context treatments since we know these behaviors and associated neurobiology are embedded within a transactional system with correlated exposures.

Heritability work also can help inform our compassion for those involved - understanding that some children and young people started with more risk and may have parents with higher risk who may themselves need particularly intensive support and scaffolding. This observation can inform treatment development in understanding that parent and child traits (and/or other contexts like peers) are likely to be moderately correlated and thus strong interventions will need to consider what the child and parent are bringing to the interactions. Naturally how genetics findings are communicated is very important, in order to guide against potentially misguided or deterministic interpretations of heritability statistics (Harden, 2021).

Third, to effectively prevent and treat aggression, we need a tiered and multi-pronged approach using a public health model (Dodge, 2008). This starts with universal policy and health options, screening to identify those at risk to engage in selective preventative interventions (Dodge, 2020), and finally a robust system of indicated interventions that can be personalized or matched to subtypes of aggression or related forms of antisocial behavior. For example, universal (or selective) nurse visitation programs can help set up all families for success and help to prevent risk factors like child abuse from developing (Dodge, 2018). Then, via primary care or school settings, children and young people can be screened for early risk factors and families engaged in selected preventative interventions during early childhood. Finally, interventions need to be available that target multiple contexts (e.g., incredible years program across school, home, and child domains; Pidano & Allen, 2015) and at developmentally appropriate level of intensity (e.g., multisystemic therapy is necessarily more intense as it targets adolescents who may be further entrenched in behaviors with more entrenched contexts around them; Henggeler, 1999). Though these components exist, they are seldom combined in a comprehensive way, nor available and accessible to those families who may have more risk (Baskin-Sommers et al., 2022).

One specific implementation within this tiered model is the use of a health maintenance model for either universal or selective targeting. One example of this approach is the Family Check-Up, a yearly check-in with at-risk families that uses motivational strategies and measurement of family risk via family report (Dishion et al., 2008). This check in can potentially capture, with more precision, when children and young people may be at tipping points towards more aggression or away. Moreover, through yearly check-ins (that can be expanded into parent management interventions, among others) the intervention can catch parents when they are ready to engage. This model may help address challenges in both parents and children at higher risk. Moreover, because the intervention is family-centered, it can be personalized more, potentially leading to more engagement and success.

Moreover, this type of program highlights that more regular treatment, across longer time scales (and potentially across contexts), may be more successful, particularly for those at higher risk. As described above, given the ways in which risk compounds over time, more intensive interventions may be needed at first, but then more regular intervention over longer periods, that are developmentally tailored, may yield more success. That is, instead of treatment being seen as a 12-week program that ends, it may need to be seen similar to how we treat chronic health conditions - an initial treatment followed by continued evaluation of progress and trajectory to intervene when symptoms increase or contexts shift.

Fourth, beyond the need for personalized, longer-term treatments and/or those with a health maintenance approach, complex system-level contexts may best be targeted by a sequence of treatments. Moreover, the order of these treatments may matter to their success. For example, children and young people with aggression may have attention deficit-hyperactivity disorder (ADHD) and parent management training may be more effective only after effective treatment (with medication) of ADHD symptoms (Wells et al., 2000). Additionally, some children and young people and families may respond

differently to the first intervention, in ways that may inform what next intervention may work best. This sort of tailoring based on initial characteristics of treatment response can help inform more effective second line treatments. These types of complex treatment questions are rarely addressed in typical clinical trials. However, Sequential Multiple Assignment Randomized Trials (SMARTS) can address these questions by examining multiple sequenced interventions to identify and describe dynamic treatment regimens (Kidwell & Almirall, 2023; Kidwell & Hyde, 2016).

Relatedly, beyond larger timescales and more intensive treatments that target behavior over months and years, transactional research on parenting and peers highlights the may mechanisms driving aggressive cognitions and behaviors (and likely shaping brain responses over time), and that these mechanisms occur at a shorter time scales (moment-to-moment). We propose that mechanistically motivated interventions (mechanistic Randomized Controlled Trials, mRCTs) could be used as causality levers to examine potential active ingredients of positive change. By measuring cognitive, neural, and social/environmental factors at baseline and subsequent time points, such trials could not only give us more information about a causal status of particular protective factors but could also help us refine our understanding of who benefits from what. As an example, we could systematically and longitudinally measure child and parent emotion regulation, cognitive flexibility or agency, and study whether they can be changed via cognitive training and reinforcement learning approaches and how that would, in turn, impact social functioning. Naturally the success of any endeavor like this would depend on the quality of measures that are deployed (see Step 2). Further, micro-randomized trials offer the opportunity to intervene in small discrete ways at shorter time scales (Walton et al., 2018). Importantly, like SMARTS, these trials can inform us, not only about personalization, but also about how sequences of interventions (or small nudges) can shape behavior in ways that informs our understanding of the development of aggression and its associated neurobiology.

Conclusion

Our understanding of aggression is poised for transformation. We now are in a position to integrate insights from biologically- and environmentally-focused research in ways that allow us to specify mechanisms and better understand transactional relationships that inform how and why some children grow up to display aggression. To fully capitalize on this potential, measures and models of neurocognitive factors as well as the environment are needed to capture the complexity of aggression. Aggression does not emerge simply because of genes, the brain, or the environment, but instead it is a transactional unfolding of brain and neurocognitive development within context.

Despite the pervasiveness and burden of aggression, there is a relative lack of funding to study the mechanisms driving aggression or treatments designed to address aggression. One possible explanation is the reluctance to label children, to avoid any potential risk of stigmatizing them. This is a reasonable concern; however, it does not seem to apply to other behaviors that cause problems in young people (e.g., anhedonia, social withdrawal). There is an assumption that aggressive behaviors are viewed differently because of the risk for harm, but that assumption not only considers aggression as somehow only affecting society (rather than also the suffering of the individual engaging in this behavior) but also neglects to understand the financial and emotional harm to others caused by more internalizing behaviors (e.g., healthcare costs, loss of work by caregivers to care for a child). Therefore, we must ask ourselves why aggressive behavior in children and young people has not been handled in a similar was to other issues, and often is seen as a matter for the criminal legal system rather than mental health services. Children and young people who display repeated aggression do not always elicit sympathy due to the very

nature of their behavior. The lay public and even the law tends to see repeated aggression as a moral failure. By doing so, we ignore the strong science behind how aggression develops and how it can be managed. Integrating research on the biosocial mechanisms that contribute to aggression in children and young people can potentially reduce the blame and personal responsibility ascribed to these youth. We can view their behavior through a lens of complex interactions that affect how child and young people take in information and what information they are exposed to in specific contexts; interactions that can be addressed by targeted interventions. At the same time, we must carefully discuss the role of biology in our conceptualization of aggression so as to avoid assumptions of determinism and prognosis (Lebowitz, 2014). Ultimately, we must provide a balanced narrative about aggressive behavior. We need to change the narrative that labels these young people as “bad” or “predators” and commit to an era of understanding rooted in science.

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