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Toward a holistic understanding of conduct disorder across the lifespan

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Abstract

Conduct Disorder is a developmental disorder characterized by clinically significant and culturally unexpected aggressive and antisocial behaviors. While much work has focused on the numerous predisposing genetic, neurobiological, psychological, and environmental risk factors for Conduct Disorder, its causal mechanisms, several developmental trajectories, and interacting risks have still yet to be properly elucidated within the context of these factors. With the aim of integrating the literature's current understanding of Conduct Disorder, we searched APA PsycNet and Google Scholar using a scoping review to select peer-reviewed articles relating to age of onset, presentation, trajectory, persistence, and outcomes of Conduct Disorder. Of the 29 papers found, abstract screening and full text review identified 21 relevant peer-reviewed articles. When taken together and critically examined from a developmentally informed perspective, this holistic review of the literature highlights age of onset and persistence as important influences of disorder trajectory and outcome. The vast heterogeneity of Conduct Disorder should be given greater weight in future research, diagnosis, and early intervention efforts.

Keywords: conduct disorder, risk factors, development, lifespan, clinical psychology

Toward a Holistic Understanding of Conduct Disorder Across the Lifespan

The *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.; DSM-5) defines Conduct Disorder (CD) as a disorder in childhood and adolescence that is characterized by clinically significant and culturally unexpected aggressive and antisocial behaviors, and involves difficulties with self-regulation (American Psychiatric Association [APA], 2013). Specifically, CD requires symptoms of heightened aggression, destruction of property, deceitfulness, and rule-breaking behaviors that lead to impairment in areas of the youth's life (APA, 2013; Frick, 2012, 2016). For diagnosis, CD requires that at least three of its fifteen diagnostic criteria be clinically significant (APA, 2013). A diagnosis of CD can be either 'Mild,' 'Moderate,' or 'Severe' and the DSM-5 further provides three specifiers to categorize the disorder's age of onset—childhood-onset type, adolescent-onset type, and unspecified onset—and an additional 'with limited prosocial emotions' specifier to capture presentations that are callous, of blunted affect, or lacking in empathy (APA, 2013).

As identified in the literature (and as alluded to by its diagnostic criteria), CD is quite heterogeneous, with many genetic, neurobiological, psychological, environmental, and interactional risk factors predicting different severities, developmental pathways, and outcomes. CD also has broad multifinality, suggesting that understanding its development, maintenance, and life-course are all extremely important for informing future diagnostic criteria and developing proper treatments. As this review will elucidate, current research typically focuses on singular risk and protective factors (e.g., genetic, neurobiological, developmental, psychological, or environmental) that influence CD. By failing to consider these factors together, the extant literature limits its understanding of the CD etiology. From the work reviewed, it is clear that the

developmental pathways of CD are complex and, with the literature still narrowly focused and developing, not well understood.

Given CD's multiple presentations, pathways, and outcomes, the goal of this paper is therefore to adopt, through a scoping review, a holistic psychological lens to examine the literature's current understanding of the risk and protective factors influencing the disorder's development. Age of onset and persistence will be introduced as main aspects of the disorder, and notable differences in their unique risk factors will be discussed. The paper concludes with discussion on what age of onset means for the youth's behavior and level of attainment over their lifespan, including discussion on the relationship between Conduct Disorder in adolescence and Antisocial Personality Disorder in adulthood. By bringing several risk and protective factors (e.g., genetic, neurobiological, psychological, or environmental) together, as existing work has yet to do, we will approach the holistic understanding of the disorder's etiology required to improve the identification and treatment of youth with more severe, earlier-onset, and persistent Conduct Disorder presentations.

Methods

Search Strategy

Given that the goal of this review is to construct a more comprehensive understanding of Conduct Disorder by examining its identified risk and protective factors together, a scoping review of the current literature is employed. A scoping review (see Kastner et al., 2012; Tricco et al., 2016) is an exploratory method of literature review that seeks to understand the current state of a research area, in this case, Conduct Disorder. This scoping review will progress through four stages: (1) identifying a review goal, (2) defining search criteria, (3) screening studies for relevance, and (4) organizing and summarizing the identified literature.

As previously noted, this scoping review aims to synthesize the current state of Conduct Disorder research so as to come to a better understanding of its risk and protective factors, its developmental pathways, and its outcomes. Given this goal, we constructed five relevant search strings that were used in both APA PsycNet and Google Scholar. These search strings are as follows: (1) “Conduct disorder onset”, (2) “Conduct disorder” AND “antisocial personality”, (3) “Conduct disorder review”, (4) “Conduct disorder” AND “risk factor*”, (5) “Conduct disorder” AND “environment”. From these five search strings across two scholarly databases, 29 unique peer-reviewed research articles were identified.

Screening

The abstracts of all 29 peer-reviewed research articles were read in full and relevant findings were summarized. As per step (3) of our scoping review process, articles were deemed “relevant” if their findings related directly to Conduct Disorder and to the disorder’s etiology, multifinality, or to one or more of its risk and protective factors (i.e., genetic, neurobiological, developmental, psychological, or environmental). Of the 29 abstracts read and summarized, 21 papers were deemed “relevant”, based on these criteria, and were thus read in full.

To complete the scoping review process, the findings of all 21 peer-reviewed articles were organized and summarized. Specifically, the findings were discussed in terms of genetic, neurobiological, developmental, psychological, and environmental risk and protective factors, as well as developmental pathways, disorder age of onset, and disorder persistence.

Risk and Protective Factors

Genetic Influences

Conduct Disorder has a strong genetic basis. Twin studies find that about 50% of CD’s phenotypic variance is attributable to genetic heritability (Salvatore & Dick, 2018; Wesseldijk et

al., 2018). Further, genetics appear to account for 91% of CD persistence between childhood and adolescence (Wesseldijk et al., 2018). Several genes have been associated with the disorder's presentation. The GABRA2 gene, implicated in impulse control and reward behaviors, as well as the MAOA, SLC6A4, and AVPR1A genes, all related to aggression, are genes associated with CD symptoms and maintenance (Holz et al., 2018; Salvatore & Dick, 2018). While promising, this work is in its infancy and replication should be emphasized (Salvatore & Dick, 2018).

Neurobiological Influences

Youth with Conduct Disorder tend to have several neurostructural and neurofunctional differences, including a smaller prefrontal cortex (PFC), amygdala, and insula; regions associated with executive function, theory of mind, reward, and affect (Fairchild et al., 2011; Holz et al., 2018; Junewicz & Billick, 2020; Matthys et al., 2013). There is also evidence of altered structure and function of the basal ganglia, a region related to emotional processing and executive function (Holz et al., 2018). While most functional results are consistent across studies, some find reduced amygdala functioning, suggesting a dull threat response, while others find increased amygdala functioning, suggesting an oversensitivity to threat (Holz et al., 2018; Junewicz & Billick, 2020). These differences may be reconcilable by controlling for psychopathy traits (Holz et al., 2018; Junewicz & Billick, 2020). One additional study using machine learning techniques identified the middle frontal gyrus, parietal lobe, superior temporal gyrus, and occipital lobe as areas whose activation differentiated children with diagnosed Conduct Disorder from healthy controls (Zhang et al., 2020). While these areas are broadly implicated in attention, somatosensory processing, and social attention, more research will be needed to properly parse these effects and their meanings (Bigler et al., 2007; Japee et al., 2015).

Neurochemically, reduced serotonin and oxytocin may be associated with the decreased punishment response seen in CD. Diminished sensitivity to dopamine may lead to thrill seeking and reduced reward significance, while increased testosterone may contribute to heightened aggression (Junewicz & Billick, 2020; Matthys et al., 2013). Brain connectivity in the default mode network, a system thought to be related to morality, theory of mind, and emotional regulation, appears to be decreased in youth with CD (Junewicz & Billick, 2020). Reduced heart rate, skin conductance, and startle response are also common, suggesting lower anticipatory fear and under-arousal (Junewicz & Billick, 2020; Matthys et al., 2013).

These structural, functional, connective, and physiological differences may be summarized by construing them as deficits in punishment processing, reward processing, and cognitive control; clusters mapping well onto CD's associated behaviors (Matthys et al., 2013).

Environmental Influences

Perinatal Influences

Conduct Disorder is closely related to a broad set of environmental risk and protective factors that hold variable levels of influence across the entire lifespan. Starting prenatally, CD is associated with maternal education, alcohol consumption, anxiety, depression, social stress, family income, unpredictable or unsafe home environments, and neighborhood safety factors (Frick, 2012; Holz et al., 2018; Jennings et al., 2018; Latimer et al., 2012). Having a teenage mother or a mother who smoked during pregnancy is also a risk factor for the development of CD (Gutman et al., 2019).

Childhood and Adolescent Influences

In childhood, many of these perinatal risk factors retain their salience and several additional risk factors such as family separation and poor parenting styles arise (Frick, 2012;

Goulter et al., 2020; Holz et al., 2018, Latimer et al., 2012; Yockey et al., 2021). Specifically, harsh punishment between kindergarten and grade two predicts CD symptoms in adolescence (Goulter et al., 2020). Interestingly, low parental warmth during this time does not predict CD symptoms, but rather predicts a blunted affect and lacking empathy; traits associated with CD (APA, 2013; Goulter et al., 2020; Gutman et al., 2019). Additional family processes such as eating dinner apart, having divorced, unemployed, or incarcerated parents, witnessing violence in the home, and having a parent with mental health or substance use issues are also associated with CD in adolescence (Gutman et al., 2019; Yockey et al., 2021).

Adverse Childhood Experiences (ACEs; Felitti et al., 1998), negative early life experiences such as abuse, parental mental illness, and violence, are related to the development of Conduct Disorder (Holz et al., 2018; Moore et al., 2017). Childhood is a time when youth begin socializing outside of their household. This is seen in the increasing role of peer deviance and peer victimization in CD risk (Holz et al., 2018). With their increasing autonomy, children foster resilience through positive teacher-child relationships and develop positive coping strategies, prosocial skills, impulse control, emotional regulation, and self-esteem; all of which serve as protective factors (Holz et al., 2018). Outside of the shared home environment, effects of unique environment increase from explaining roughly 13% of CD individual differences to explaining over half of the variance by adolescence (Wesseldijk et al., 2018). Considered as a whole, these risk factors appear to exert varying but cumulative effects across different developmental stages such that they interact with each other to exacerbate the risk of Conduct Disorder symptomology (Holz et al., 2018).

Neighborhood Influences

At a more macro-level, Conduct Disorder appears to be associated with specific adverse neighborhood characteristics (Jennings et al., 2018). One recent literature review found evidence that exposure to neighborhood violence was predictive of externalizing behavior and that this relationship is mediated by stressful life events and peer choices (Jennings et al., 2018). Evidence shows that factors such as neighborhood social cohesion can mitigate these links (Jennings et al., 2018).

Interactions Among Risk and Protective Factors

Just as environmental risk factors interact with previous environmental factors in a cascade, genetic factors interact with the child's environment to increase risk. The phenotypic expression of genes associated with CD appears to be moderated by the experience of early factors such as parenting style and parent-child attachment (Salvatore & Dick, 2018). Interestingly, only 12.1% of youth with genetic risk and 6.7% of youth with environmental risk exhibit antisocial behavior while 40% of youth with both genetic and environmental risk exhibit these behaviors (Cloninger et al., 1982). One pioneering gene-environment interaction study examines the relationships between monoamine oxidase-A (MAOA), a gene that regulates serotonin receptors, and ACEs on conduct behaviors later in life (Caspi et al., 2002). MAOA allele type was found to moderate the effects of maltreatment on antisocial behaviors in adolescence (Caspi et al., 2002).

Conduct Disorder is best predicted by the confluence of genetic-neurobiological and environmental factors (Trentacosta et al., 2013, as cited in Chan et al., 2022). Recent research using machine learning is beginning to show quantitatively the extent to which these genetic, neurobiological, and environmental factors interact to predict the development of Conduct

Disorder (Chan et al., 2022; Zhang et al., 2020). In one recent study, advanced machine learning methods took a biopsychosocial interactive approach by using measures of fMRI connectivity, neurocognitive ability, existing diagnoses of Attention-Deficit/Hyperactivity Disorder (ADHD) and Oppositional Defiant Disorder (ODD), neighborhood crime, peer interactions, and parenting practices at ages 9 and 10 to predict diagnoses of CD two years later (Chan et al., 2022). The interaction of these biopsychosocial variables predicted CD with over 91% accuracy (Chan et al., 2022). While the machine learning methods employed in this study were able to test the interactions among all predictors, a significant disadvantage to such work is that the specific nature of these interactions cannot be easily teased apart (Chan et al., 2022). This is a job for future research.

Developmental Pathways

On average, youth with Conduct Disorder exhibit decreasing physical violence and increasing non-violent conduct with age (Burt, 2015). While informative to aggregate across trajectories like this, it is imperative to note that CD can present in many ways. This heterogeneity is important from a treatment and prevention perspective. CD trajectories may help predict developmental outcomes and inform treatment (Frick, 2012, 2016; Walters & Knight, 2010).

Age of Onset

A robust finding is that Conduct Disorder age of onset predicts trajectory (Frick, 2012, 2016; Gutman et al., 2019; Walters & Knight, 2010). This important distinction is recognized by the DSM-5 with Childhood-onset (symptoms identifiable before the age of 10 years) and Adolescent-onset (symptoms identified after 10) specifiers (APA, 2013). Youth with childhood-onset CD tend to show greater levels of aggression across the lifespan, greater symptom

maintenance, impairment in verbal learning and memory, higher incidence of psychosis, lower educational attainment, lower levels of cognitive function, and are more often from backgrounds where childhood maltreatment is prevalent (Fairchild et al., 2011; Frick, 2012, 2016; Johnson et al., 2015; Walters & Knight, 2010). These deficits specific to childhood-onset CD were originally thought to be the result of marked structural differences in the insula and prefrontal cortex (Fairchild et al., 2011; Huebner et al., 2008; Moffitt, 1993), but newer work suggests that these differences are no more pronounced than those in youth with adolescent-onset CD (Fairchild et al., 2011). Thus, while youth with childhood-onset CD tend to exhibit more behavioral indicators of CD and greater cognitive impairment, these differences do not appear to their have roots in biology and may instead be due to differing gene-environment interactions or greater childhood maltreatment (Burt, 2015; Fairchild et al., 2011).

Even though adolescent-onset CD carries the same brain differences relative to healthy controls as childhood-onset CD, the later developing trajectory is much less associated with violence, poor cognitive functioning, ADHD, ACEs, emotional regulation difficulties, and impulsivity (Fairchild et al., 2011; Frick, 2012; Gutman et al., 2019; Johnson et al., 2015; Moore et al., 2017; Walters & Knight, 2010; Yockey et al., 2021), and more associated with environmental factors such as being from families with socioeconomic risk (Gutman et al., 2019; Yockey et al., 2021). This path has been described as “an exaggeration of the normative process of adolescent rebellion” (Frick, 2012, p. 379) partly because it is characterized by severe rule-breaking more than violence, and is more associated with peer deviance, low parental supervision, and rejection of social status hierarchies; all possible results of environmental self-selection (Burt, 2015; Frick, 2012).

Persistence

While youth with Conduct Disorder are sometimes seen as a homogenous group, Moffitt (1993) makes a distinction between two pervasive subgroups in her seminal *developmental taxonomy theory of antisocial behavior*. Now widely accepted and studied, Moffitt (1993) notes that CD's significant continuity across the lifespan contradicts the fact that CD prevalence increases dramatically for only the adolescent years. Moffitt (1993) therefore proposes a theory of two distinct Conduct Disorder trajectories. Adolescent-onset CD typically resolves by early adulthood in a pathway known as adolescent-limited (AL; Moffitt, 1993). On the other hand, those showing persistent patterns of antisocial behavior across life typically first exhibit CD early, in a pathway known as life-course persistent (LCP; Moffitt, 1993; Moore et al., 2017). Indeed, the pathways of most youth with CD will fit these labels; recent work shows that 9.9% of youth with CD follow an LCP trajectory and 42.1% fit an AL trajectory (Moore et al., 2017).

Much like age of onset, the earlier-manifesting and more perennial life-course persistent (LCP) CD appears to be associated with environmental risk factors such as childhood ACEs, low family socioeconomic status, quality of parent-child attachment, parental discipline style, prenatal maternal depression, having a teenage mother or mother who smoked during pregnancy, and difficult childhood temperament (Gutman et al., 2019; Moffitt, 1993; Moore et al., 2017). Unlike age of onset, however, recent research shows that youth with LCP CD exhibit brain deficits related to executive functioning, emotion regulation, and motivation that are much more profound than those in youth with adolescent-limited (AL) CD (Carlisi et al., 2020). Thus, much like youth with childhood-onset and adolescent-onset CD, LCP CD is more associated with early life environmental risk than AL CD, but unlike childhood-onset and adolescent-onset CD, LCP CD is related to much more profound neurostructural deficits than AL CD (Carlisi et al., 2020;

Moffitt, 1993; Moore et al., 2017). Taken together, one interpretation of these findings is that, while childhood-onset CD is more often associated with poor environment in the early years, it is the interaction of this environment with profound brain differences that contribute to the life-course persistent trajectory of Conduct Disorder.

Through trajectory modelling, Gutman and colleagues (2019) supported and extended this work by confirming the existence of an AL CD symptom group, with symptoms increasing in middle childhood, and an LCP CD group, with high persistent symptoms from early childhood. A third childhood-limited symptom trajectory was also identified in the data (Gutman et al., 2019). Interestingly, the LCP symptom group evidenced a markedly smaller group membership than the childhood-limited (CL) group (representing 7.7% and 23.2% of the sample, respectively; Gutman et al., 2019), suggesting that LCP CD is actually a non-normative pathway. While early socioeconomic, family, and child risk were predictive of all three CD pathways, prolonged socioeconomic, family, and child adversity were predictive of the AL and LCP groups and not the CL group (Gutman et al., 2019). Future work should try to tease these two childhood-onset CD paths apart.

Though there is utility in grouping by onset and persistence, it is important to consider that there exists variability within the heterogeneous trajectory groups themselves. For example, Brislin and colleagues (2021) found evidence for three distinct profile groups within a group of children diagnosed with childhood-onset CD. These profiles differed on impulsivity, punishment sensitivity, as well as experiences of guilt and empathy (Brislin et al., 2021).

Despite the robust research support for these developmentally informed categories and their unique predisposing risk factors (Gutman et al., 2019; Moffitt, 1993; Moore et al., 2017), the AL and LCP labels are not discussed in the DSM-5 (APA, 2013). Their addition in future

iterations of the DSM as subtypes, specifiers, or even simply as discussion points in the disorder's 'Diagnostic Features' section could greatly help clinicians determine probable persistence and support the selection of efficacious interventions for their client based on these different developmental pathways (Moore et al., 2017).

Multifinality

Antisocial Personality Disorder (APD) is a personality disorder diagnosable in adulthood that is characterized by a persistent disregard for the rights of others and is associated with significant public health costs in the medical and correctional sectors (APA, 2013; Scott et al., 2001). There is much empirical evidence for a relationship between Conduct Disorder in adolescence and the development of Antisocial Personality Disorder (APD) in emerging adulthood (APA, 2013; DeLisi et al., 2019; Frick, 2016; Junewicz & Billick, 2020; Walters & Knight, 2010; Wesseldijk et al., 2018). Indeed, between 80 and 90 percent of those with diagnosed APD exhibited CD earlier in life (DeLisi et al., 2019). Biological similarities, such as lower than typical levels of cortisol, serotonin, and oxytocin, exist between youth with CD and adults with APD (Junewicz & Billick, 2020). These differences are consistent with the reduced stress reactivity and aggressive disinhibition seen in both disorders (Junewicz & Billick, 2020). CD in childhood and adolescence is also significantly correlated with later antisocial behaviors indicative of APD (rs = .20-.38; Wesseldijk et al., 2018). In recent work, Goulter and colleagues (2020) found that low parental warmth between kindergarten and grade two, but not harsh parenting style, is predictive of clinically significant ASPD in adulthood and that this relationship is mediated by trait callousness (i.e., blunted affect, lacking empathy); a symptom of more severe CD (APA, 2013; Frick, 2012, 2016; Holz et al., 2018; Junewicz & Billick, 2020).

While the disorders most commonly associated with CD, namely ODD and ADHD (Chan et al., 2022; DeLisi et al., 2019; Moore et al., 2017; Yockey et al., 2021), are not predictive of APD, previous CD diagnosis and symptoms are strongly associated with a later diagnosis of APD in adulthood (DeLisi et al., 2019; Walters & Knight, 2010). Though number of criminal offenses is not related to likelihood of APD diagnosis, earlier age at time of first offence significantly increased the odds of an APD diagnosis in adulthood (Walters & Knight, 2010). This finding is consistent with theory suggesting that APD is most closely related to youth with life-course persistent CD (Frick, 2016; Moffitt, 1993; Moore et al., 2017). As seen, some pathways and traits are more associated with lifelong antisocial behavior than others (Frick, 2016). Breaking outcomes down by trajectory may help identify groups that are most at risk and could aid in early prevention and treatment, but much research is still needed to better determine what risk factors are associated with which CD trajectory so that appropriate interventions can be designed (Frick, 2012).

While the complex developmental origins, pathways, and outcomes of Conduct Disorder still need to be properly elucidated, the psychosocial difficulties it presents in adolescence have the potential to set young adults back in their development, possibly implying enduring challenges (Frick, 2012, 2016).

Limitations and Future Directions

This review has served to elucidate the numerous genetic, neurobiological, psychological, environmental, and interactional risk and protective factors in the development of Conduct Disorder. The disorder, however, is not homogenous and has many possible developmental pathways. Age of onset and persistence were discussed as key factors that define disorder trajectories and outcomes. As reviewed, while age of onset is a core specifier of Conduct

Disorder in the DSM-5, life-course persistence—which is predictable by type of risk exposure, and which carries clinical implications for psychosocial and physical health outcomes later in life—is not so much as mentioned. Future research should therefore critically re-evaluate the DSM-5 (APA, 2013) diagnostic criteria through a developmentally-informed lens to ensure that clinically meaningful factors such as life-course persistence are accounted for in diagnosis. Secondly, because of the persistence and significant public health costs of Antisocial Personality Disorder, work should continue to address the link between Conduct Disorder and APD so that the downstream public health costs of antisocial behavior can be mitigated. Finally, future work should examine in greater detail how the many risk factors for Conduct Disorder interact to predispose youth to these differential trajectories (Frick, 2012, 2016). While this review notes strong evidence for their interaction, the nature of these interactions is not well understood. Identifying the interactive causal mechanisms of each trajectory will be important for ensuring that youth at risk of a more severe, earlier-onset, and persistent Conduct Disorder presentation can receive appropriately intensive prevention and intervention efforts in a timely manner.

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