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The Etiology of Conduct Disorder
and its Relation to Antisocial Personality Disorder:
A Literature Review

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Antisocial behavior enacts a heavy price on both the individual engaged in the behavior and the society in which he or she resides. Research has shown that among a subset of individuals antisocial behavior is fairly stable from childhood through early adulthood. This review article traces the hierarchical development of antisocial behavior from childhood Oppositional Defiant Disorder (ODD) through the adult diagnosis of Antisocial Personality Disorder (APD), with particular emphasis placed upon adolescent Conduct Disorder (CD). Possible environmental etiological factors of CD are discussed, the most notable being familial discord and low socioeconomic status. Potential biological etiological determinants of antisocial behavior include brain injury and structural abnormalities, neurotransmitter regulation, neural processing, and genetic factors. The reciprocal interplay between both environment and biology as they contribute to the development of CD is explored in the context of the diathesis-stress model. Finally, limitations of the current research are examined, with suggestions made for future research directions.

Introduction
Antisocial behavior is perhaps one of the most crippling aspects of mental illness with which society must contend. It confers heavy burdens on both the individual and the public alike. Adults with Antisocial Personality Disorder (APD) frequently experience a variety of problematic behaviors and outcomes such as violent criminal behavior, substance use, and early death. Likewise, social programs, especially mental health services and the criminal justice system are heavily taxed by antisocial individuals requiring their services (Washburn et al., 2007). Indeed, recent epidemiological studies reveal that more than 33% of currently incarcerated juvenile detainees meet the criteria for Conduct Disorder (CD), an adolescent antisocial disorder (Washburn et al., 2007). Research has consistently indicated that problematic antisocial behavior has a strong continuity from childhood through adolescence and into adulthood (Farrington, 2004; Holmes, Slaughter, & Kashani, 2001). Some individuals may engage in antisocial behavior at one point in their development and later outgrow such behavior; however, of particular concern to society is the subset of the population for whom antisocial behavior is a chronic trait (Loeber, Burke, & Lahey,
2002; Lahey, Loeber, Burke, & Applegate, 2005; Washburn et al., 2007). For many individuals, adolescence in particular is a developmental period characterized by increasing antisocial behavior, particularly violence (Gretton, Catchpole, & Hare, 2004).

As such, it is vital to understand the origins of antisocial behavior, as better understanding of both the nature and etiology of this behavior may give rise to early and effective interventions to circumvent its course. From the literature, it is clear that both biological and environmental factors greatly contribute to the development and manifestation of CD in adolescents, the presence of which is a significant predictor of APD in early adulthood. Researchers believe that 30-40% of adolescents with CD progress to develop APD, significant percentages to be sure (Loeber et al., 2002). This review will trace the developmental trajectory of antisocial behavior from childhood risk factors, including Oppositional Defiant Disorder (ODD), through adolescent CD, ending with APD after the age of 18. Particular emphasis will be placed upon the environmental and biological factors related to adolescent CD, as well as the relation between CD and APD. Implications and fallacies of the current literature will be discussed. Finally, we will provide suggestions for future research directions.

Overview of the Evolution of Antisocial Behavior

Typically, the conceptualization of antisocial behavior shifts as one matures; behavioral actions categorize an individual as antisocial early in childhood and adolescence, while personality factors typically classify adults as antisocial (Blonigen, Hicks, Krueger, Patrick, & Iacono, 2006). Loeber et al. (2002) have set forth a hierarchical model of antisocial behavior, positing that childhood ODD is a necessary precursor to adolescent CD, which in turn acts as an antecedent of APD. It is important to note that the pool of antisocial individuals shrink as they mature; that is to say, only a portion of ODD children progress to CD, and only a subset of CD adolescents escalate to full-blown APD. However, retrospective studies reveal that between the ages of 13-17, 82-90% of APD cases met the criteria for CD (Loeber et al., 2002).

Childhood

As stated previously, ODD is a strong predictor of CD and later APD for many youth. The DSM-IV-TR describes ODD as “a pattern of negativistic, hostile, and defiant behavior” (American Psychiatric Association [DSM-IV-TR], 2000, p. 100). A child with ODD is often argumentative, deliberately antagonistic and vindictive, and easily annoyed by others. The ODD child typically violates rules set forth by adults and has an external locus of blame. Additionally, unwarranted anger in benign situations is a hallmark of ODD (DSM-IV-TR, 2000).

Holmes, Slaughter, and Kashani (2001) have found that ODD symptoms tend to worsen as some children age, progressing to vandalism, stealing, substance use, and aggression against others or society. Oppositional traits such as defiance and irritability are strong indicators of negative emotionality, which is believed to contribute to the development of antisocial behavior (Trentacosta, Hyde, Shaw, & Cheong, 2009). It is these children who typically escalate into CD.

In addition to a formal diagnosis of ODD in childhood, many researchers have found that specific childhood temperaments, impulsivity, fearlessness, sensation-seeking, aggression, and low scholastic achievement were present in the past histories of conduct disordered antisocial youth and young adults (Farrington, 2004; Glenn, Raine, Venables, & Mednick, 2007; Holmes et al., 2001; Trentacosta et al., 2009). One's temperament is evident within the first few months of life; Farrington (2004) acknowledges that when a 3-4 year-old child displays “difficult temperament,” predominantly in the form of high irritability in conjunction with low amenability and adaptability, it is likely that the child will have poor psychiatric adjustment at ages 17-24. According to Farrington (2004), an inability to control one's temperament is a crucial predictor of later aggression, delinquency, and convictions in early adulthood (ages 18-21). This manifests as restlessness, impulsivity,
and poor attention in children (Farrington, 2004). Moreover, 3 year olds who were “difficult to manage” have frequently been shown to be delinquent in early adolescence, at age 11 (Holmes et al., 2001).

Impulsivity is perhaps the most influential personality dimension in the development of antisocial behavior (Farrington, 2004). Indeed, impulsivity as a character trait has been most strongly correlated with antisocial behavior between the ages of 9 and 15 (Holmes et al., 2001). According to Trentacosta et al. (2009), fearlessness at age 2 significantly predicted elevated conduct problems across early and middle childhood (Trentacosta et al., 2009). Furthermore this study showed that young children who exhibit fearlessness often exhibit high levels of daring, which leads to sensation seeking and also has been linked to conduct problems.

In addition to fearlessness, other personality dimensions exhibited by children (i.e., aggression and low academic achievement) have been related to antisocial behavior and CD (Holmes et al., 2001). Moreover, these authors have shown that both children with ODD and later CD and APD individuals tend to be very aggressive early in life, which has been related negatively to the formation of beneficial peer relationships. This deficit of positive peer relationships has been associated with the formation of both CD and later APD.

Finally, decreased childhood IQ and poor scholastic achievement in preschool have been correlated with increased levels of violence, CD, and future antisocial behavior (Farrington, 2004; Holmes et al., 2001). Low IQ measurement at age 3 significantly predicted criminal offenses up to the age of 30, with frequent offenders having an average IQ of 88 at the age of 3, compared to the average nonoffender IQ of 101 (Holmes et al., 2001). In sum, the earlier aggression or academic problems emerge in childhood, the more predictive they are of future problem behavior (i.e., CD and APD).

Adolescence

According to the DSM-IV-TR (2000), antisocial behavior in adolescence is frequently conceptualized as Conduct Disorder, “a repetitive and persistent pattern of behavior in which the basic rights of others or major age-appropriate societal norms or rules are violated” (p. 93). Moreover, CD is categorized by four classes of symptoms: aggression towards people and animals, destruction of property, deceitfulness or theft, and serious rule violations. Examples of conduct-disordered behavior include physical fighting, forceful sexual activity, deliberate fire setting, cruelty to animals, running away, stealing while confronting a victim, and use of a weapon (DSM-IV-TR, 2000). CD adolescents tend to display poor verbal ability, impulsivity, neuroticism, and low constraint (LaBrode, 2007). The emergence of symptoms before age 15 classifies an individual having as childhood-onset CD, compared to adolescent-onset CD after age 15. Earlier symptomology is correlated with both a poorer prognosis and increased development of APD, compared to late-onset CD (Farrington, 2004). Consistent with trends for antisocial behavior, prevalence rates for CD are greater among adolescent boys (6-16%) than girls (2-9%; Farrington, 2004). Interestingly, the age of onset is typically later for girls than boys, and since earlier onset increases the likelihood of a worse outcome, the gender differences in CD prevalence can partially account for the gender differences in APD (Farrington, 2004).

It is widely agreed upon that CD is a critical predictor of APD in early adulthood (Farrington, 2004; Gretton et al., 2004; Trentacosta et al., 2009). Specifically, Lahey, Applegate, Loeber, and Burke (2005) found the number of CD symptoms during early adolescence (7-12 years), to predict early adult APD in men receiving outpatient treatment from a mental health clinic as a child. In fact, they noted that the odds of subsequent APD increased by 37% at each higher level of CD symptoms - one, two, three, and four or more, respectively. Likewise, Washburn et al. (2007) report that the chances of developing APD by early adulthood (18-19 years) significantly increased among the CD youth with 5 or more symptoms.

Beyond the connection between CD and APD, empathy – defined as the ability to understand and appreciate the emotional states and needs of
others—is thought to be a core deficit in CD adolescents (Decety, Michalska, Akitsuki, & Lahey, 2009). Because aggression and violence are chief markers of CD, researchers have proposed that this intrinsic lack of empathy, combined with a callous disregard for the welfare of others is related to a propensity for aggressive, violent behavior (Decety et al., 2009).

Early Adulthood

After the age of 18, antisocial individuals meet the diagnostic criteria for Antisocial Personality Disorder (APD). Deceit and manipulation are essential features of APD, as well as a lack of remorse, failure to conform to societal norms, impulsivity, aggression, reckless disregard for the safety of others, and consistent irresponsibility. Also, the individual must present with evidence of CD with onset before age 15 (DSM-IV-TR, 2000).

APD individuals often repeatedly engage in criminal behavior, con others for personal profit or pleasure, engage in assault, rationalize their behavior, and consistently fail to sustain work or fiscal responsibilities (DSM-IV-TR, 2000). Young adults with APD have a sense of entitlement and are affectively cold (LaBrode, 2007). As stated previously, adults with APD are at a substantially greater risk for criminal and violent behavior, early death, substance use, unemployment, and homelessness than the non-APD population (Washburn et al., 2007).

Etiology of CD

Environmental Factors

As is the case with the overwhelming majority of mental illness, environmental factors appear to play a significant role in the development of CD. An individual’s familial experiences are perhaps the most influential environmental factors in the development of both CD and later APD (Holmes et al., 2001). Martens (2000) has extensively documented the adverse effect of family variables on the development of antisocial behavior. Adolescents who come from families plagued by severe marital discord, large family size, paternal criminality, maternal mental illness, and foster care placement are at risk of developing antisocial behavior in the form of CD and later APD, particularly if they also have been diagnosed with ADHD (Martens, 2000). Parental separation and single-parent households also predict CD children. In fact, Farrington (2004) has shown that separation from one or both parents before the age of 5 predicted CD at age 15.

Researchers also have investigated the role of poor parent-child relationships in relation to CD; many adolescents with CD report a lack of parental warmth, attention, and supervision (Martens, 2000). Cycles of coercive parent-child interactions have been related to substantial antisocial conduct as well (Kotler & McMahon, 2005). In addition to relationship quality, ineffective parenting styles also have been implicated in the development of serious conduct problems in children and adolescents. Extreme parenting styles, that is to say those styles which are excessively harsh or overly permissive, and inconsistent disciplinary messages tend to produce adolescents with CD (Kotler & McMahon, 2005).

Furthermore, many antisocial individuals who presented with CD in adolescence have parents who themselves tend to come from families characterized by sexual abuse, divorce, financial struggles, increased mobility, and working women. These parents frequently lack high-ordered job skills, thus resulting in chronic unemployment (Martens, 2000). Finally, it is clear that a majority of CD youth come from families of low socioeconomic status (SES; Farrington, 2004). Several studies have shown that low SES has predicted CD in adolescents, particularly in combination with familial dependence on welfare (Farrington, 2004). Among adolescents who met the diagnostic criteria for CD, only 20% from higher SES families escalated into APD as young adults, compared to the 65% of lower SES adolescents who later met the criteria for APD (Lahey et al., 2005). It is important to note that adolescents who score higher
on daring and risk-taking scales and also live in high-risk, relatively dangerous neighborhoods (often associated with low SES) are particularly vulnerable to developing significant antisocial behavioral problems (Trentacosta et al., 2009).

**Biological Factors**

While one’s surroundings certainly have been shown to be relevant to the development of both CD and APD, Martens suggests that biological effects are both longer lasting and more influential than environmental factors (2000). The following biological areas of importance have been identified in relation to CD and antisocial behavior in general, and will be discussed below: brain injury and structural abnormalities, neurotransmitter regulation, neural processing, and genetic factors.

First, antisocial personality changes have been observed in formerly prosocial patients who suffered a traumatic brain injury, suggesting that abnormalities in brain structure and function may play a role in antisocial disorders (Martens, 2000). The frontal lobe of the brain is primarily responsible for executive, high level functioning; it is this region of the brain that allows individuals to exercise judgment, mediate impulse control, and exert planning. As such, early damage or abnormalities present at birth within the frontal lobe may contribute to some of the core symptomology of antisocial behavior. Indeed, frontal lobe lesions are consistently linked to disinhibition and impulsivity, both of which are central features of APD (Martens, 2000). Likewise, the amygdala often has been associated with emotional processing and response; atrophy and lesions of the amygdala have been linked to impulsively aggressive behavior, poor emotional regulation, and limited awareness of the moral implications of one’s actions (Decety et al., 2009).

Researchers also are interested in the role that neurotransmitters play in the formation of antisocial behavior and CD. Studies recently have begun to evaluate the role of serotonin in aggressive behavior. Findings suggest that decreased serotonin function in the brain, specifically concerning the permutations 5-hydroxytryptamine and 5-HT, contributes to the manifestation of aggression (Martens, 2000). Emotionally driven, impulsive acts of destruction also have been associated with low serotonergic neurotransmission (Hofvander, Ossowski, Lundstrom, & Ankarsater, 2009). In individuals with ODD and CD, a low cerebrospinal fluid concentration of serotonin metabolites was associated with aggressive antisocial behavior (Hofvander et al., 2009).

However, the results of a case study of a 15 year-old homicidal boy with CD conducted by Virkkunen et al. (2003) stand in direct contrast to many findings which indicate that low levels of serotonin precipitate aggressive antisocial behavior. These researchers measured serotonergic metabolism in the form of amino acid levels present in the cerebrospinal fluid of the offender. Compared to 10 normal controls, researchers found a stark contrast in levels of amino acid. The offender had an 84% higher level of plasma total L-tryptophan and a 143% higher level of free L-tryptophan than the average mean among controls. These findings map onto the results of an earlier study (Tiihonen et al., 2001), which concluded that young adults with APD had higher levels of plasma total L-tryptophan (137%) and free L-tryptophan (160%). As these amino acids levels reflect metabolized serotonin levels, the Virkkunen et al. (2003) findings indicate that higher than average levels of serotonin may in fact give rise to violent and aggressive behavior. Though more research is certainly needed to ascertain the complicated impact of serotonin on violent and aggressive behavior, it is safe to say that starkly abnormal levels of this neurotransmitter likely play a role in the manifestation of violent and aggressive behavior.

As mentioned previously, prevalence rates of antisocial behavior are significantly higher for boys than girls (Farrington, 2004), leading some researchers to speculate that testosterone may play a role in CD
and APD. Early aggression in boys and increased aggressive behavior in premenstrual girls — both times of development during which testosterone levels are disproportionately high — gives credence to the theory that testosterone may fuel aggressive behavior (Hofvander et al., 2009).

CD youth also display neural processing abnormalities. In response to the fact that the overwhelming majority of CD youth frequently lash out against others, Decety et al. (2009) used fMRI studies to examine differences in empathetic response when viewing others in pain among CD and control adolescents. The neural responses of eight CD youth when viewing others in pain were examined against eight control youth. Though the findings were complex, it is important to note that when observing pain intentionally inflicted by another, CD youth displayed no activation in neural regions that contribute to self-regulation, moral reasoning and metacognition such as the anterior cingulate cortex and orbital frontal cortex. The control group did display activation in the aforementioned areas, suggesting that empathic responses to others in pain are severely diminished in CD adolescents. While provocative, these findings require further substantiation before definitive conclusions can be drawn.

Genetics is another area of great interest to researchers searching for causal factors in antisocial behavior. Though studies that examine the relation between APD in parents and subsequent diagnoses of antisocial disorders in their children are rare, family and twin studies suggest that antisocial disorders are heritable (Forsman, Lichtenstien, Larsson, & Andershed, 2008). It has been found that children with antisocial fathers, regardless of whether the father was present in the home during the child’s formative years, have an increased risk for APD, suggesting that genetic influences in part determine antisocial developmental trajectories (Martens, 2000). Aggressive antisocial behavior in particular seems highly heritable, with 65% of variance between APD and control individuals attributed to genetic factors (Hofvander et al., 2009).

Likewise, CD symptoms tend to be concentrated in families; Lahey et al. (2005) found that maternal APD predicted the development of APD in their sons, though paternal APD did not. This stands in contrast to earlier findings discussed above, in which having an antisocial father predisposed children to becoming antisocial themselves. However, it is possible that paternal APD was not a significant predictor in this study because so many biological fathers were no longer in contact with their families at the time of investigation, thereby forcing researchers to rely on retrospective reports from the mothers concerning their partners’ antisocial behavior. 

Biological-Environmental Interaction

To be sure, the etiology of antisocial personality disorder is a complicated issue. The diathesis stress model of mental illness contends that individuals inherit a genetic vulnerability or predisposition to developing a particular disorder; however, the disorder is only manifested if triggered to develop by an environmental stressor. Many researchers have echoed this sentiment as it applies to CD and APD, and it is important to remember that the cause of APD is likely neither wholly biological nor wholly environmental. In fact, Martens (2000) suggests that biologically vulnerable individuals can be protected against developing APD if not exposed to social triggers, while individuals born into risky social contexts but without biological vulnerabilities may escape antisocial disorders. Martens also has posited that negative influences such as environmental stress and adverse family situations, combined with a lack of positive stimulation, may cause biological reactions which result in antisocial disorders. He believes that these environmental triggers lead to neurophysiological insensitiveness and mental indifference as a means of self-protection. Likewise, Holmes et al. (2001) posit that the interplay of genetic vulnerabilities coupled with environmental triggers induces a cyclical worsening of
behavioral and psychological symptoms, eventually resulting in a CD/APD phenotype. Both theories are predicated on the interaction of biology and environment, though they differ slightly. Martens believes that environmental stressors may trigger biologic compensatory changes in an individual, whereas Holmes et al. discuss the possibility of physiological factors inducing the early childhood behavioral symptoms of CD. Certainly, further research is necessary to ferret out the root causes of antisocial behavior. However, the authors find this line of theory entirely plausible, likely even. Many behaviors have been shown to have underlying biological causes, as is the case in Turret’s Syndrome. As science becomes increasingly more precise, perhaps the conceptual ambiguity between behavior and biological influence will be reduced.

Limitations

While contemporary research has spurned a slew of thought-provoking findings in recent years, there are still several areas which require tighter control and improvement. For instance, when examining causal factors of CD and APD, scholars routinely have failed to control for the interaction between biological and environmental factors (LaBrode, 2007). As such, the preponderance of research findings to date cannot yield conclusive data on the unique role of environmental or biological factors.

Furthermore, twin studies and other biological evaluations, which are typically used to shed light on the biological correlates of aggression and antisocial behavior, historically utilize very small sample sizes. Indeed, Decety et al. (2009) had a mere sample of 16, while the revolutionary findings concerning elevated levels of serotonin and violent aggression of Virkkunen et al. were based on a case report of a single CD adolescent male. Such minute sample sizes cause one to wonder whether study findings can be accurately replicated with a larger subject pool. With such limited sample sizes, it is possible that the results are simply due to chance.

Additionally, many studies draw from the pool of incarcerated youth (i.e., Gretton et al., 2004; Lahey et al., 2005; Washburn et al., 2007). While it is certainly true that many CD youth and APD adults engage in criminality and run a greater risk for incarceration than the general public, one must question the generalizability of studies which only include an incarcerated sample. Results may not apply to those antisocial individuals who have escaped jail; since manipulation, guile, and deceit are hallmarks of APD, it stands to reason that the subset of the antisocial population that avoids incarceration may be fundamentally different, perhaps even more pathologically sophisticated, than the portion that does not.

Lastly, it is worth noting that criteria which rely primarily on behavioral problems in order to diagnose CD may lead researchers to overestimate the portion of youth that is at risk for chronic antisocial problems (Gretton et al., 2004). As mentioned previously, only a subset of children with ODD mature into adolescents with CD, and in turn it is believed that less than half of CD youth become adults with APD. Both researchers and society alike are at a loss to understand this phenomenon. Perhaps clinicians rely too heavily on behavior problems as indicators of antisocial psychopathy in adolescence; maturity and age may curb such behavior in a significant portion of the CD population, leaving only those marred by serious personality, as compared to behavioral, defects vulnerable to APD as young adults.

Implications

Research pertaining to both causal factors and the interconnectedness of antisocial disorders is not only fascinating, but also necessary. Studies consistently have shown that the tendency towards violent aggression is a relatively stable character trait over the course of one’s lifetime (Gretton et al., 2004).
Similarly, offenders with antisocial pathologies tend to commit more numerous and varied crimes than pathologically normal offenders, and utilize violence in a premeditated, instrumental manner (Kotler & McMahon, 2005). Taken together, these findings paint a picture of chronically volatile individuals who have an enduring involvement with the penal system. Not only is this a drain on societal resources, but antisocial individuals pose a danger to the public at large as well. It stands to reason that successful interventions for antisocial behavior would reduce a considerable percentage of violent crime, thus easing the burden on social institutions and improving the quality of life for offenders and potential victims alike.

Furthermore, it is a universal truth that increased knowledge can lead to better understanding, which in turn enables researchers to target specific problematic areas. Simply put, when clinicians and academics alike have a clearer picture of what contributes to and exacerbates CD and APD, they can begin to focus their efforts on circumventing its course. Additionally, clearer understandings of the etiology of CD and APD will dictate any subsequent interventions. After all, for a solution to be successful, it must match the cause. Once research findings are definitive and substantial, clinicians can begin to practically tailor and implement treatments.

Of particular concern are the childhood and adolescent permutations of antisocial disorders, ODD and CD respectively. As discussed above, the earlier aggressive antisocial behavior emerges, the more likely it is to be chronic and escalatory. With this in mind, early interventions should not only successfully mitigate such behavior, but may actually be easier to employ, since the young have likely not yet become as entrenched and sophisticated in their ways as APD adults.

**Future Research Directions**

Future studies on APD should aim to replicate and substantiate recent controversial findings. Decety et al. (2009) have heralded in a new facet of research, using fMRI equipment in order to evaluate neural responses of CD adolescents. While certainly thought-provoking, it remains to be seen whether their findings will stand the test of time. However, this study has likely opened the door for a sophisticated new line of neural research; in addition to the strict neuronal abnormalities of CD, scholars in the future should focus on the neural activation pathways of aggressive and hyperactive youth at varying levels of SES. It also would be interesting to conduct longitudinal evaluations of neural cognition in ODD children, screening for differences among those who progress to CD and those who do not.

In addition to continuing the work of Decety et al. (2009), the line of inquiry started by Lahey et al. (2005) also should be advanced. In their sample, 50% of CD adolescents developed APD in early adulthood, the other 50% did not. In the future scholars should strive to identify group differences in those who persisted in serious antisocial behavior and those who desisted with age. Maternal APD was a predictive factor in filial development of CD and APD within the sample, yet those findings were fairly new and require further substantiation. Similarly, more studies are needed to determine whether paternal APD is truly not a significant predictor of son’s CD and APD. A lack of relation between paternal APD and filial APD would point to genetic abnormalities of the donated mother X chromosome in the development of antisocial behavior.

Finally, the preponderance of the literature examines environmental and biological influences upon the development of CD and APD, yet one has yet to identify possible prenatal factors. As maternal substance use has been associated with several mental illnesses in offspring (Holmes et al., 2001), it is worth
investigating the role of alcohol use, drug abuse, and stress levels during pregnancy in conjunction with antisocial behavior. In sum, CD and APD are complex disorders which hold great ramifications later in life, for both the individual and society as a whole. The evolution of antisocial behavior should not be looked upon lightly, as a portion of antisocial children progress into violent and aggressive antisocial adults. While there is clear evidence of both biological and environmental bases in the etiology of CD and APD, further research is needed to pinpoint causal factors, and in turn create evidenced-based interventions and treatments.

References


