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A DEVELOPMENTAL PERSPECTIVE TO ATTENTION-DEFICIT HYPERACTIVITY DISORDER (ADHD) IN CHILDREN

Olusegun Emmanuel Afolabi
afolabi@unibotswana.ac.bw
University of Botswana, Botswana

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Abstract. The debate about diagnoses and treatment of attention deficit hyperactive disorder (ADHD) in children continue to range on between the developmental and biological perspectives. While there is increasing evidence that support the biological susceptibility of the disorder, a number of researches also emphasized the significant effect of environment on the syndrome. This study used developmental perspectives to evaluate and bring together various bio-psychosocial factors that impact on children diagnosed with ADHD. The study explored and integrated the existing and advancing study on ADHD to a more refined pattern that embraced developmental perspectives. The study also discussed how the linkage in childhood ADHD fits within the developmental psychopathology perspective. The study revealed that ADHD as a developmental disorder is influenced by prenatal, biological and psychosocial environmental risk factors, and suggested that better understanding of genomic susceptibilities, family environment and parental characteristics would transform the pathway for development of ADHD in children.

Keywords: attention deficit hyperactive disorder, developmental perspectives, childhood disorder, genetic factors, environmental factors.

1. Introduction

Attention-Deficit/Hyperactivity Disorder (ADHD) is a severe childhood disorder that affects many facets of human being, particularly young children.

populace (American Psychiatric Association, 2000) and has been a subject of intensive research for decades (Barkley, 2006). While studies over the years demonstrated the advancement made on ADHD, the intense interest on the disorder continue to produce a number of empirical data on etiological factors, complex genetic and the neurobiological variables that underlie it, particularly, the developmental causes and treatments that are relevant for diagnosing the disorder in children. For example, studies like molecular and behaviour have long offered considerable suggestions to support the significant effect of genetic factor on ADHD. (e.g., Kuntsi, & Stevenson, 2000; Sunohara et al., 2000). Additionally, a quite number of models were proposed to address the syndrome, particularly on children’s cognitive deficiency (e.g., Berger & Posner, 2000; Sergeant, 2000). However, contrary to the progress reports on bio-cognitive development, the theory on social and relational features of the syndrome remains stagnant, as general consensus showed multiple casual pathways, with environmental factors primarily labelled as ameliorating the symptom in children (Sonuga-Barke, Auerback, Campbell, Daley & Thompson, 2005).

Although research on family of children with ADHD continue to be acknowledged, (e.g., Sonuga-Barke, Auerback, Campbell, Daley & Thompson, 2005), the debates about children diagnosed of ADHD, particularly, its occurrence and origin continues to range on. More disturbing is the fact that developmental conceptualizations of the syndrome in children and adolescence have been neglected. Specifically, the clinical and social effects of the symptom have been waned, to say the least downplayed in most literature. This made it hard for practitioners and families of children diagnosed with ADHD to cope with its challenges, and projected the problems and discriminations experienced by families and children with ADHD an important issue for consideration. Therefore, much is desire on influence of the gene-environment interactions on socio-cognitive development of children diagnosed of ADHD.

2. Methods and objectives

This study provides a brief overview on the social and clinical factors associated with children diagnosed with ADHD, and uses developmental perspectives to evaluate and bring together various bio-psychosocial factors that impact on their development (e.g., Rutter & Sroufe, 2000). To achieve these objectives, this study examines and integrates the existing and advancing study on the ADHD to a more refined pattern that embraced developmental perspectives. Also, the study organized into sections, the clinical and social factors related to childhood ADHD and explained how these factors influenced children’s development. Finally, the study discussed how linkage in childhood ADHD fits within a developmental psychopathology perspective and makes recommendations for future research.

3. The study

3.1. Scientific status of attention deficiency hyperactive

ADHD is multidimensional disorder that exacts a significant effect on individual and society. This disorder has negative impact on families, as well as
academic and vocational outcomes of vulnerable children (Biederman et al 2004). As the most generally diagnosed neurobehavioral illness in children, the disorder is mostly treated with stimulant and non-stimulant drugs (United States, 2003, & 2007; Pastor, & Reuben, 2008). Even though the exact causes of the ADHD are still unknown, past and present research confirmed the significant effect of genetic and environmental factors on the disorder (Nigg, Nikolas, & Burt, 2010, Thapar, Langley, & Asherson P, 2007). Besides, research on ADHD emphasised more on the period of birth by establishing a strong correlation between period of birth and children psychological and behavioural disorders (Tochigi, Okazaki & Kato, 2004), This is in contrast with several other disorders where a reliable seasonal form is yet to be established. (Atladóttir, Parner , & Schendel, 2007; Hauschild, Mouridsen, & Nielsen, 2005).

As an unsatisfactory umbrella term, ADHD is applied to children with broadly differing temperaments and functional problems in school, home, and social settings. This group of children shared certain core features, such as limited sustained attention span, poor impulse control, and motor over activity. They also developed abnormal syndromes, such as severe development, distraction and thoughtlessness that cause severe impairment in their learning (Hauschild, 2005). Research on ADHD also showed a strong genetic orientation on the disorder. For example, the inattentiveness aspect of the disorder is documented as fantasizing, distractibility, and associated with problems, such as lack of concentration on specific task for a lengthy period, while the hyperactivity element of the syndrome is pronounced as fidgeting, unnecessary talking, and restiveness (Farone, Perlis, Doyle, Smoller, Gornick & Holmgren, 2005). The signs of ADHD are also predisposed to accidents, strain interpersonal relationships, disruptions and improper conduct. However, apart from its association with clinically oriented disorder in children, ADHD also linked to characteristics in adulthood, such as drugs and alcohol misuse; socio-cognitive disorders; disruptive conduct and delinquency (Thapar, Langley, O’Donovan, 2006).

Despite the above illustrations and evidences, the developments of ADHD remain debatable, as the causes of the symptom pointed toward multidimensional perspectives and linked to children and adults mental health, (Thapar et al, 2006). This further shows the effect of genetic factors on its development. It also shows that its relations with ecological risk variables are complex. Based on this foregoing, there is a need to ponder on the evolving nature of the symptom and the differences in the phenotypic indicator, particularly, the influence of ecological factors on childhood ADHD (Thapar et al, 2006).

3.2. Diagnosis consideration of ADHD

Research in the last 60 years has witnessed the use of several terminologies for attention deficit– hype reactivity disorder (ADHD). Some of this terminology includes: hyperkinetic impulse disorder, minimal brain dysfunction, hyperactivity, attention deficit disorder. However, the core characteristics of the disorder are inattention, impulsivity, and hyperactivity and affects about 4% of all children.
Besides, the signs of the syndrome are more noticeable in young people and vary between 3 to 11% or more (Berger, 2011, Childress & Berry, 2012). However, despite its occurrence in young children, the origin of the disorder is yet to be identified. This difference in expression revealed the diverse conceptions of the primary symptoms and its assumed fundamental pathophysiology.

Literature on ADHD revealed that the prospect of finding a diagnostic indicator for the disorder is not achievable. This is due in part, to the nature and complexity of the syndrome (Baumeister, & Hawkins, 2001, Zimmer, 2009). However, research identified three subtypes of ADHD and each of this subtypes were differs on symptomatology. For example, for a child to be diagnosed of ADHD, and labelled with particular subtypes, he/she must display 6 symptoms for a period of 6 months. Although achieving such diagnostic criteria is difficult, this method is used as a bench mark for diagnosing the disorder in children. Children diagnosed with ADHD also showed some degree of functional impairment in multiple settings (Berger, 2011, APA, 2013). However, due to the parallel characteristics of the disorder, the comorbidities, such as anxiety disorders and ODD, influenced its sub-type in children.

While the criteria listed in DSM-V for ADHD is more or less broader over DSM-IV-TR, the issue of sex differences in children hyperactive disorder continue to range on (Berger, 2011, APA, 2000). For instance, male child are 3 times potential of having ADHD and display hyperactive behaviour or combination of it, than female child, (Childress, & Berry, 2012). Also, females are more expected to display predominantly absent-minded subtype and suffer from mental impairment and eating disorders (Trent, & Davies, 2012). Further, there is higher sense of aggressiveness and abuse of law among male than female diagnosed of ADHD (Trent, & Davies, 2012). On the basis of this assumption, it is imperative for professionals working with children diagnosed of hyperactive disorder to be consciously aware of its sexual and developmental variances. This would prevent over-or under diagnosed of the disorder in children. Furthermore, there should be proper analysis and assessment of parents and teachers reports, so as not to mislabel the underline disorders (Dopheide, 2005, Rader, McCauley, & Callen, 2009).

3.3. Etiological model of ADHD

While it was established that the main aetiology of ADHD is unknown (Sonuga-Barke & Halperin, 2010), it is important to understand its aetiology and other associated disorders that relates with the syndrome. This would help clinicians to identify the interactions between the genetic and environmental factors and how they increase vulnerability in young children. The process would also offers a way out for the heterogeneity of the disorder in a meaningful manner, as research showed lack of systematic incorporation of the findings across multiple levels of analysis (e.g., Coghill, Nigg, Rothenberger, Sonuga-Barke, & Tannock, 2005; Sonuga-Barke & Halperin, 2010). Therefore, etiological models on ADHD emphasised the impacts of genetic and environment factors; their correlations and interactions; influence on brain composition and function, and the mediating role on
the symptom expression, As a result of these challenges, more investigation is needed to create a clear relationships between supposed fundamental genetic and neural processes, and the behavioural manifestations of the disorder. This would increase and encouraged new and effective treatments (biological and non-biological), and offered necessary information on the framework that supports the management of ADHD particularly, in hypothesising, diagnostic of boundaries and current arrangement of the illness.

In addition, the hypotheses for reducing brain function in children diagnosed of ADHD were grounded on several observations that reduced the volume of gray and white matter in the brain. This causes shortfalls in cognitive processing, responsiveness, motor planning, speed of processing responses, and other related behaviour in the disorder (Cortese, 2012). Though, prefrontal cortex (PFC), caudate, and cerebellum were the primary source of shortfalls in children diagnosed of hyperactive disorder, this was formed by different neurons that together, regulate attention, thoughts, emotions, behaviour, and negative actions in children (Arnsten, & Pliszka, 2011, Kesner, & Churchwell, 2011). Poor development of PFC11 reduced the activity of the PFC, caudate, or cerebellum (Arnsten, & Pliszka, 2011). The system activity between the regions is “subtle to the neurochemical environs,” (Arnsten, & Pliszka, 2011) and sustained by the combination of neurotransmitters (NTs), dopamine (DA), norepinephrine (NE) and multiple receptors (Arnsten, 2007, Robbins, 2003).

Etiological model also identified aggressiveness, impairment and other related problems, (i.e., antisocial conduct) as the key goals of the symptom in children. Though, medication was identified as a way of reducing hyperactive disorder in children, its long-term supports for the broader outcomes of ADHD are yet to be established. These underscore the importance of identifying the genetic-environment factors that caused the negativity and impairment in children with ADHD, and provide answer to the growth of active risk decline tactics in the long-term management of the disorder. Based on this aforementioned, it is imperative for research to focus on understanding the genetic and environmental risk factors that associated with ADHD, as well as the clinical characteristic that projected the outcomes of the disorder in children. This would target resources and monitor children at risk of adverse concerns.

3.4. The need for a new model on ADHD

A decade of scientific study on ADHD has highlighted the need for a new theory that explains the syndrome; as ADHD is confirmed as a disorder particularly, in respects to its basic nature. Most research on the ADHD is more or less investigative and descriptive, with exception of two. First, Quay's (1988a, 1988b, 1996) used the neuropsychological model of anxiety developed by Gray's (1982) to describe the source of the poor inhibition manifested in ADHD. This model relates thoughtlessness to under-functioning of the brain's behavioural inhibition system. Also, it explained that children with hyperactive disorder are highly subtle to the signs of conditioned punishment, and less sensitive to passive avoidance models
(Quay, 1988b). The second model failed in its attempt to set up a concept similar to the one established in Quay-Gray theory. The model makes a comprehensive theory construction that offers coalescing explanation on various mental shortfalls that are related to children diagnosed with attention/hyperactive disorder.

3.5. The developmental approach

The desire for a theory that embraced the clinical and social aspects of attention/hyperactive disorder has prompted the need for developmental approach to the ADHD. Although a comprehensive neuropsychological model of ADHD has yet to be proposed, other models of psychopathologies was previously recommended (Gray, Feldon, Rawlins, Hemsley, & Smith, 1991). Developmental approach entails the correlation between the etiological heterogeneity, high level of comorbidity, and biological and psychosocial/family of ADHD (Cummings, Davies, & Campbell, 2000; Sonuga-Barke & Halperin, 2010). These interactions underscore the need to posit a multiple developmental pathways to treatment of children diagnosed of ADHD (Sonuga-Barke et al., 2005; Sonuga-Barke & Halperin, 2010), and were mediated by a variety of within child and family contextual factors that associates with either the diminution or exacerbation of the symptoms over time.

For example, dynamic developmental psychopathology approach offers an explanation on how attention/hyperactive disorder evolved, and how the interactions between multiple risk and protective factors impact on children development (Rutter & Sroufe, 2000). The model proposed that children in the course of their development were influenced by biological risk factors, with a relatively lesser impact from the ecological factors. The model also highlighted that, across children and across time, there are variables that influence the development of attention/hyperactive disorder. The theory predicts that though, precise symptom of ADHD at a particular time in life varies, they are influenced by factors that have positive or negative effects on the symptom development. Further, the theory explained that, individual differences in dopamine functioning have significant impact on motor functions and children learning. This produced behaviours, such as attention problems, hyperactivity, and impulsiveness that associated with ADHD, and predicted an increase in children’s behavioral variability. Overall, dynamic developmental theory proffer better explanation on how person predispositions interacted with the above mentioned conditions and relatively created behavioral, emotional, and cognitive effects that balanced the behavioral patterns of children with ADHD. Thus, a child’s characteristics coupled with the family situation exerted collaborating influence on ADHD and offered unique opportunity for analysing the disorder symptomatology.

3.6. Psychosocial adversity and its developmental course

Though, many studies have proposed significant evidence for the existence of psychosocial problems in children with hyperactive disorder, such evidences predicts the socio-cognitive and emotional development, rather than precise predictors of the disorder. Therefore, it remains uncertain whether experience of
violence in infancy is a risk factor for ADHD, as there was no theoretical basis for observing this possible relationship. For example, exposure to violence in a household may act through psychosocial adversity and lead to permanent brain change that occurs as a result of prolonged exposure of the developing brain to steroid hormones (Yehuda, 2000).

However, Rutter et al (1975) reported that the combination of environmental factors (i.e., severe marital discord, low social class, paternal criminality, maternal mental disorder), rather than existence of a single factor, promote psychopathology in children. This argument was supported by a lot of scholars, such as Campbell (2000); Faraone and Biederman (1998); Rutter and Sroufe (2000); and Taylor (1999) where they established that genes-environment multiple interactions are linked to attention/ hyperactive disorder in children. Similar findings by Biederman et al (1995b,) corroborated earlier work by Rutter and his colleagues to establish that negative family–environment significantly influenced children with ADHD. In addition, the finding established that, exposure to parental psychopathology (particularly maternal) is more pertinent to families of children with ADHD than the control families (Biederman et al 1995b).

Interestingly, while some studies in the field of developmental approach established that children are born with a genetic predisposition that relates to hyperactive disorder (e.g., Faraone, Perlis, Doyle, Smoller, Goralnick, Holmgren, 2005), others maintained that heredities are rarely the sole reason for the development of attention/ hyperactive disorder, as MZ concordance rates is not near 100% (Faraone & Biederman, 2000; Kuntsi & Stevenson, 2000). Besides, some scholars maintained that 50% of children with hyperactive disorder do not display the biological anomaly associated with congenital factors (Swanson, et al., 1998). Therefore, in situation where biological predisposition is strongly established, family characteristics was viewed as reflection of the indicator and consequence of the disorder in children.

Furthermore, the categorization of relative contributions of shared versus non-shared hereditary and ecological menaces within the families of children diagnosed of ADHD is important for proper analysis of the disorder. For example, in a situation where there is a problem in a family, which is due to the disorder, or shared genetic susceptibilities, the family environments must be related to the child characteristics. On the other hand, when family breakdown is linked to the child empathy, the constancy of the disorder became aggravated. In this case, the family environment is associated with attention/hyperactive disorder not as a main cause, but as a factor that increased and influenced its development. However, children with ADHD develops relatively little tendency to the disorder, as confusing and uncaring family setting increase their behaviours (e.g., Carlson, Jacobvitz, & Sroufe, 1995). This means that, the degree of intellectual and physical stimulation that children received in their immediate environment impacted on their brain development and behavior (Halperin & Healey, 2011). Therefore, responsive and sensitive parenting promotes child self-regulation skills and parental difficulties that harmonize parents’ activities with child’s desires for development of disinhibited
behaviour (e.g., Carlson, Jacobvitz, & Sroufe, 1995). So, when the family and child characteristics work in tandem, child’s temperament antecedents of inattentiveness and impulsivity that create or exacerbate parents’ problems are moderated.

3.7. Genetic contributions to ADHD and developmental course

ADHD is not a genetic disorder in a clear sense, but can be categorised as a genetic factor that was shaped by developmental pathways. (Thapar, O’Donovan, & Owen, 2005). While past and present research continues to highlights the importance of genetic factors on ADHD (Faraone & Doyle 2001; Faraone & Tsuang 1995), attempts to recognize its source of using a candidate gene method to detect common hereditary variant have been less successful (Neale et al., 2010). Thus, genetic explanations of ADHD are determined by data, such as family and twin studies that shows ADHD as a familial and highly hereditary. This heritability was estimated to be in average of 76% (Faraone et al., 2005). While it was established that attention/hyperactive disorder is a family oriented symptom, the first-degree families of affected persons displayed higher rates of the disorder (relative risk 4–5). In addition, it was confirmed that the threats of the disorder are higher in families of those with history of hyperactive disorder (Faraone et al., 2005). This finding highlighted the significant agreement between early studies of children diagnosed with hyperactivity syndrome (Morrison and Stewart 1971) and successive studies that uses DSM-III and DSM-III-R definitions of ADHD (Biederman, Faraone, Keenan, Knee, & Tsuang, 1990).

A meta-analysis study conducted by Faraone et al., (2005) revealed a small but significant impacts for a number of assumed functional variants in genes controlling brain neurochemistry particularly, in the dopamine system (e.g., D4 and the dopamine transporter (DAT1). The common variants in genes of other neuromodulator systems (i.e., serotonin and norepinephrine) was also related with genes that control the general brain function and growth (e.g., Brophy, Hawi, Kirley, Fitzgerald, & Gill, 2002, Oades et al., 2008).

Furthermore, the analysis of comorbid psychiatric disorders supported the inherent heterogeneity of the ADHD in children. This established a significant degree of ADHD among families of adults with ADHD (Biederman et al. 1995a). For example, the independent samples of children with DSM-III attention-deficit disorder and DSM-III-R ADHD are related to familial susceptibilities (Biederman et al., 1990; 1991b; 1992), while attention/hyperactive disorder and bipolar conditions was established as a separate familial subtype of ADHD in children (Faraone, Biederman, & Monuteaux, 2001a). Attention/ hyperactive disorder were also found to be familiarly free from anxiety disorders and learning disabilities (Faraone et al. 1993). Based on this foregoing, we can conclude that stratification by behaviour and bipolar disorders divides the life of children diagnosed of ADHD into more familial related subgroups, and that major depressive disorder is a generic expression of different subtypes of ADHD in children. Therefore, persistent attention/hyperactive disorder are a useful phenotype for molecular genetic studies (Faraone et al. 2001). However, despite the inaccessible findings in literature, individual gene
relationships account for modest variation in ADHD expression in children (Faraone et al., 2005; Neale et al., 2010).

3.8. Twin and adoption studies
Due to the genetic nature of ADHD, twin’s studies are consistently used to establish the heritability, or the level at which genetic characteristics influence attention/hyperactive disorder (Hudziak, Rudiger, Neale, Heath, & Todd, 2000; Kuntsi and Stevenson 2001; Martin, Scourfield, & McGuffin, 2002). The studies also offered a reliable evidence to support that, hereditary factors add to the aetiology of ADHD i.e., (60–91%) (Thapar et al, 2005b). Twin studies also confirmed that inherited factors are the main source of continuousness of attention/hyperactive disorder particularly, the relationship between the disorder and disruptive behaviour (Thapar et al, 2006). The studies revealed that inherited factors impacted on ADHD and its developmental progression. Research on twins and adoption studies also established extra risk factors that do not have any significant influence on the origin of ADHD, but contributed to its clinical developmental outcomes. However, this notion was condemned because children genetic factor are chosen with a priori notion of genetic involvement in the syndrome, while in neuropsychiatric illnesses, the pathophysiology is typically unidentified.

3.9. Biological adversity
Research suggested that some biologic factors, such as lead contamination, food additives/diet, cigarette and alcohol exposure, to mention a few, contributed to the development of attention/hyperactive disorder in children. Though, Feingold Diet for ADHD was promoted by the media and acknowledged by most parents as a contributing factor, scientific enquiry showed that the idea is ineffectual, as addictiveness to food cannot cause attention/hyperactive disorder (Conners 1980). Research also argued that exposure to lead pollution causes restlessness, hyperactivity, distractibility, and lower intellectual ability in children diagnosed with hyperactive disorder. This idea was opposed by other studies, as it was established that lead account for only few of the majority of ADHD issues in children. This means that exposure to high lead environment does not necessary lead to hyperactive disorder in children. Further, research identified complications during pregnancy and delivery (i.e., maternal age, poor maternal health, and duration of labour) to mention a few as influenced development of ADHD in children (Sprich-Buckminster, Biederman, Milberger, Faraone, & Krifcher, 1993). They also confirmed that maternal smoking is related to the pathophysiology of ADHD that caused disruption to nicotinic receptors and changed dopaminergic activity.

3.10. Gene-environment interaction and ADHD
Though, studies on children with ADHD revealed a significant relationship between heredity and attention/hyperactive disorder, there are quite a number of environmental factors that connected with ADHD symptoms. Two of these factors have been systematically analysed and reported as a contributing factors for
development of ADHD. These are: exposure to maternal smoking in pregnancy (Langley, Rice, & van den Bree, 2005) and low birth weight/prematurity (Bhutta, Cleves, & Casey, 2002). However, not all the vulnerable children that are exposed to environmental severity developed attention hyperactive disorder. The effects of gene–environment interaction on ADHD occurs when genes responds to environmental adversity. This is documented as important features of attention/hyperactive disorder in children.

However, only few works have probed the influence of G6E on development of children hyperactive disorder. For instance, a recent research on the issue established strong link between a DAT1 haplotype (combination of risk alleles) and attention/hyperactive disorder when mother is alcoholic during pregnancy (Brookes, Mill, & Guindalini, 2006), while others studies reported the DAT1 risk allele earlier found to be related with attention/hyperactive disorder as associated with hyperactive–impulsive symptoms found in children exposed to maternal smoking during pregnancy (Kahn, Khoury, & Nichols, 2003). Further, studies that focus on childhood behavioural disorder symptoms reported that children who carried the COMT gene risk variant are more vulnerable to the negative effects of lower birth weight (Thapar et al, 2005a). While all these findings require replication, the indication so far showed that, some genetic factors influenced children sensitivity to ecological adversity and the developmental sequence of attention/hyperactive disorder.

4. Discussion

This systematic review used developmental perspectives to address the clinical and social factors associated with children diagnosed of ADHD. Specifically, it demonstrates that gene- environment interactions are important factors in the development of attention/ hyperactive disorder in children. By focusing on developmental perspective, the paper provided considerable evidence to support the influence of bio-psychosocial factors on behaviour of children diagnosed with attention/hyperactive disorder. Therefore, the present study supported the growing body of research that emphasised the use of developmental perspective as opposed to clinical treatment of children with attention/hyperactive disorder. The study also charted a developmental framework as bases for conceptualizing the effect of gene- environment interaction on children with ADHD, and reviewed the consequences and limitations of existing studies on the symptom by exemplifying the areas where untimely deductions have been obtained and where further effort is desirable.

Also the study established that parent–child interactions and gene-environment interaction impacted on the development of children with attention/hyperactive disorder. This means that, the stressful demanding and intrusive nature of children diagnosed with attention/hyperactive disorder evoked negative reactions from other family members and disrupted family relationships (Langley et al, 2005). The review of literature in this present study also revealed that children with attention/hyperactive disorder influenced their parent’s behaviour and adjustment, and that parent’s behaviour also impacted on development of children diagnosed
with the disorder (Brookes et al, 2006). This further confirmed family characteristics and histories as the cause of attention/hyperactive disorder in children, as parent behaviour was linked to children conduct problems (Thapar et al, 2006).

Overall, there is a general concession of continuum association between genetic and environmental factors in children diagnosed with ADHD, as family factors were mentioned as the most influential variable that promote attention/hyperactive disorder in children. Though, the number of unsupportive or inconclusive studies actually limits these conclusions, this present review motivated research and hastens full informed conclusions about the clinical and social factors associated with children diagnosed of ADHD. Therefore, the dynamism of social and biological variables in children diagnosed with attention/hyperactive disorder is not only influenced by environmental factors, but also by common genetic characteristics of the parent and the child (Biederman, Faraone, et al., 1995).

5. Conclusions and recommendations

Although attention deficit hyperactive disorder is a predominant neurobehavioral illness in children, the symptom is characterised by factors such as hereditary, ecological, and biologic aetiologies that begin from conception to adulthood. Although its aetiology remains indeterminate, the developing evidence on the symptom documented its strong neurobiological and hereditary foundations and emphasised the phenotypic difficulty of disorder on children development. Therefore, there is a need to understand how genomic susceptibilities, family environment, parental characteristics, and children’s experiences interrelate and modify its developmental pathway in children; as such efforts would prospectively enlighten and proffers intervention strategy that support its diagnose. Based on these assumptions, the following recommendations are suggested:

1. Effort should be directed toward understanding the mechanisms that underlie the associations between parental maladjustment and development of ADHD in children.

2. Future research should focus on developmental progression of attention/hyperactive disorder in children and underlie observed associations of family characteristics on the disorder.

3. Future research should focus on addressing the gaps and the great inconsistencies in the area of families characteristic and childhood attention/hyperactive disorder, because such inconsistencies remain unclear.

4. Future research should regularly embrace multiple informants and impartial assessments on childhood attention/hyperactive disorder, so that more confidence can be placed on the associations that are revealed.

5. Lastly, future research should be directed toward development of better focus theoretical models that focus on family influences and childhood attention/hyperactive disorder, as most of the existing theory on the topic were focused on either the biological contributions of families or the contributions of family environment.
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