IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 15, Issue 4 Ver. V (Apr. 2016), PP 115-122
www.iosrjournals.org

Attention Deficit Hyperactivity Disorder – A Review, Dental Implications and Treatment Recommendations for Dental Professionals

Dr.Amith Adyanthaya[1], Dr.Sajeela Ismail[2]
[1](Reader, Department of Pediatric and Preventive Dentistry,KMCT Dental College, Kerala, India)
[2](Postgraduate resident, Department of Pediatric and Preventive Dentistry,KMCT Dental College, Kerala, India)

Abstract : *Attention deficit hyperactivity disorder (ADHD) is a highly prevalent neurodevelopmental disorder in childhood. It is characterized by definite behavioral patterns of inattention, impulsivity and hyperactivity, which might lead to performance problems in the social, educational, or work environments. This article reviews the current evidence about the diagnosis and treatment of children and adolescents with ADHD based on the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition. Along with the discussion of etiology, classification, diagnosis and treatment approaches based on a survey of web tools primarily the MEDLINE database, the article also aims at reviewing the dental implications and recommendations for dental practitioners regarding management of children with ADHD*
Keywords: *attention deficit, hyperactivity, impulsivity, dental*

I. Introduction

Attention deficit hyperactivity disorder is one of the commonest childhood behavioral disorders encountered in outpatient settings. [1]Prevalence of ADHD varies depending on age and region but the worldwide prevalence is estimated to be 5.29%. [2]Based on several studies prevalence rates are estimated to be in the wide range of 2% and 17%. [3]An obvious gender predilection is found in prevalence of ADHD with male to female ratio being 3:1. [4]ADHD is assumed to develop in childhood, but less than 40% of these children meet the diagnostic criteria in their teenage years. [2]Although onset is usually around age of three, the diagnosis is not made until the child is in elementary school. [5]There is a dearth of data on the prevalence of this disorder in preschool children. [6, 7, 8]A recent Indian study found presence of comorbid disorders in 40.0% of children with ADHD. [9] Oppositional Defiant Disorder (ODD), Conduct Disorder (CD), and affective, anxiety and learning disorders are some of the common co-morbid disorders. [10]Attempts were made to identify relevant published studies in English language journals. The bibliographic databases included MEDLINE database and Google Scholar. These databases were searched for the period from 1900 to end of October 2014 to identify studies to be considered for this review.

II. Definition

As stated in the fourth edition of American Psychiatric Association's (APA) DSM-IV, ADHD is a behavioral and neurocognitive condition characterized by developmentally inappropriate and impairing levels of gross motor over activity, inattention, and impulsivity.[11] There are five main diagnostic criteria:

1. Onset before age 7 years
2. symptoms last more than 6 months
3. An 18-item symptom list of which 6 of 9 inattention or 6 of 9 hyperactivity/ impulsive symptoms have persisted for at least 6 months to a degree that is maladaptive and inconsistent with developmental level
4. Impairment in two or more settings; and
5. Symptoms that do not occur exclusively during the course of a pervasive developmental disorder or other psychotic disorders and those that cannot be attributed to any other mental illness, such as depression.[2]

III. History

The first mention of a disorder resembling ADHD was given by Sir Alexander Crichton in 1798.
The Goulstonian Lectures by Sir George Frederic Still in 1902 is considered to be the scientific starting point in the history of ADHD. He presented series of lectures on 43 child patients of his who were restless, impulsive and inattentive, with intense affective responses and conduct problems. [12, 13]
Similar clinical conditions were termed ''postencephalitic behavior disorder" [14], "minimal brain damage syndrome", ''minimal brain dysfunction'' [15], ''hyperkinetic impulse disorder'' [16] and subsequently, the concept of a hyperactivity syndrome was generated. [17]

American Psychiatric Association renamed the disorder ''Attention Deficit Disorder (ADD) (with or without hyperactivity)'' in Diagnostic and Statistical Manual of Mental Disorders, DSM-III published in 1980[18]. DSM-III devised three separate symptom lists for inattention, impulsivity and hyperactivity, which were more specific than previous ones [19]. The concept of two subtypes were eliminated in revision of the third edition of the DSM (DSM-III-R) in 1987 the condition was subsequently renamed ''Attention deficit-Hyperactivity Disorder (ADHD)''. [20] The symptoms of inattention, impulsivity, and hyperactivity were combined into a single list of symptoms with a single cutoff score. The subtype ''ADD without hyperactivity'' was deleted and added to the category named ''undifferentiated ADD''Based on a large field trial, the fourth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) was outlined in 1994. [21] ADHD was categorized into three subtypes. ADHD which was a heterogeneous category in DSM-III-R was subdivided into three subtypes. [22]

The DSM-5 was developed in 2013 and the definition of ADHD was changed in a way to differentiate the condition in children and adults. Subtypes were renamed as "presentations." Symptoms can change and an individual can change 'presentations' at different points in his lifetime.

The notable advantage of DSM-5 over DSM-IV TR is that a person can now be diagnosed with ADHD and Autism Spectrum Disorder. [23]

IV. Classification

The three subtypes of ADHD according to DSM-IV based on symptoms displayed are combined, primarily inattentive and primarily hyperactive/impulsive types. [24]In the combined subtype, symptoms of both inattention and hyperactivity/impulsivity are expressed. This is the most common subtype and also the one that has been most described. The primarily inattentive subtype, earlier termed Attention Deficit Disorder (ADD), is the second most common subdivision. This subtype generally shows no gender predilection. Some children may be initially diagnosed as primarily inattentive but gradually when symptoms of hyperactivity/impulsivity become apparent, the diagnosis gets modified as the combined subtype. The primarily hyperactive/ impulsive subtype most commonly comprises of very young children who may actually have the combined subtype but maybe of an age where inattention is barely perceivable. DSM-5 maintains these categories, but have renamed them as combined presentation, predominantly inattentive presentation, and predominantly hyperactive-impulsive presentation.

DSM-5 has an additional fourth subtype, Inattentive (Restrictive) where the criterion for inattention are met but no more than two symptoms from hyperactive/impulsive have been persisting for the last 6 months.[25]

V. Etiology

Although etiology of ADHD is not clearly determined, many studies suggest that the condition involves functional and anatomical dysfunction in the brain's frontal cortex and basal ganglia segments of cortico-basal ganglia-thalamo-cortical circuitry. [26] Research on neurobiological factors indicates that genetics and neurochemistry play critical roles. [2]

5.1 Genetics

Multiple studies indicate a genetic transmission in about 80% of ADHD cases. [24]Twin studies proves greater prevalence of ADHD in monozygotic twins. [27]As per sibling and half sibling studies, siblings of children with ADHD may be at risk of developing not only ADHD and even subclinical symptoms of the disorder. [28]Adoption studies: Though biological parents cannot be assessed, both genetic and environmental influences on the disorder are hypothesized. [29, 30]Family studies show that ADHD runs in certain families. [31, 32] First degree relatives of children with ADHD have a 20-25% risk for ADHD, compared with 4-5% for relatives of controls. [33] If a parent has ADHD, as high as 50% of his or her offspring are likely to develop the disorder. [34]Segregation Studies points at a single major gene with low penetrance. [35]

5.2 Mode of inheritance

Sex-linked transmission, polygenetic multiple threshold model and more recently, effect of several interacting genes of modest effect have been postulated to cause ADHD. [24, 36]

5.3 Molecular Genetic Studies

Various genes including the thyroid receptor B gene, dopamine type D2 receptor gene(DRD2), dopamine transporter gene(DAT1), dopamine 4 receptor seven-allele gene(DRD4), dopamine β-hydroxylase(DBH), dopamine 5 receptor(DRD5), catechol-o-methyltransferase (COMT), androgen receptors have been reported to be closely related with ADHD symptoms. [37, 38, 39, 40]

5.4 Environmental factors

The studies reviewed in this article show that many environmental risk factors and potential gene-environment interactions also increase the risk for the disorder. Several biological and environmental factors have also been proposed as risk factors for ADHD, including food additives/diet, lead contamination, cigarette and alcohol exposure, maternal smoking during pregnancy, and low birth weight.[41]Prenatal exposure to tobacco, alcohol, smoking, illicit drugs like marijuana, cocaine, amphetamines, and heroin, caffeine, antihypertensives and antidepressants have been implicated in development of ADHD. Studies and literature demonstrate an increased risk of ADHD with lead [42], manganese [43], organophosphate pesticides [44], phthalate [45], polyfluoroalkyl chemicals. [46]Lower maternal red blood cell folate and total folate intake in early pregnancy were significantly associated with higher child hyperactivity and peer problem scores at a mean age of 8.75 years. [47]Maternal pre-pregnancy obesity was found to predict high child inattentive symptoms.

5.5 Parenting and parental mental health

Probability of children with ADHD having a parent with a history of ADHD is very high. [48] Parenting styles, psychosocial support, socioeconomic status, and parental psychopathology have been pointed out as important factors which influence the development of symptoms. [49]Children belonging to families where there is marital disharmony, family dysfunction, and where there is overt hostility between child and parent have been found to develop hyperactive features. [50]

5.6 Other psychosocial factors

Adverse social factors [51] and comorbid symptoms of depression and anxiety [52] also exacerbate the symptoms of ADHD.

VI. Diagnosis And Assessment

Diagnosis is complicated, and so assessment should be meticulous and methodical. The diagnostic interviews should be based on the following: An interview with parents to establish the child's developmental history, family history, presenting problems, and other relevant information, including systematic information about the presence or absence of diagnostic criteria. [53]

Feedbacks from teachers are vulnerable in deducing the presence or absence of diagnostic criteria and special educational needs.

- Various Rating scales and checklists for diagnosing ADHD include the 'strengths and difficulties' questionnaire [54], Achenbach's child behavior checklist [55],Conners' parent and teacher's rating scales [56],Rutter's parent [57] and teacher scales [58], and the Barkley and Du Paul ADHD rating scale. [59]
- Scrupulous observation of the child can be a key in assessment.
- Medical evaluation to exclude physical causes of attention or activity problems.
- Cognitive testing might be employed for evaluation of specific difficulties.
- In confirming the diagnosis, children still should have six or more symptoms of the disorder. In people 17 years and older the DSM-5 states that they should have at least five symptoms. [23]
- Combined inattentive & hyperactive-impulsive presentation: Has symptoms from both of the other presentations.

VII. Management Of ADHD

ADHD can be managed by three treatment modalities:

(1) Medication management, (2) behavioral therapy, and (3) a combination of the two approaches. [60]

Concomitant interventions include social skills training, family work, and cognitive therapy. [53]

7.1 Medication

Stimulants of the phenylethylamine variety, such as methylphenidate and dexamphetamine, have proven short-term benefits for children with ADHD. [61, 62, 63]Although Xanthine stimulants, such as caffeine can be possible enhancers of concentration, it has also been found to increase restlessness and hyperactivity. [64] Atomoxetine, a non-stimulant, highly selective noradrenaline reuptake inhibitor is a recent advance in the pharmacological management of ADHD. [65, 66] Additional pharmacological options include the antidepressants: bupropion and desipramine; and the antihypertensives: clonidine and guanfacine. [65]

7.2 Behavioral therapy

Parents are advised to have positive interactions with their affected child. Reinforcement of positive behaviors by praise, using daily contingency charts (star or 'happy face' charts), active ignoring of negative behaviors, and even effective punishment for intolerable behaviors are recommended. [67, 68] Reprimands and redirection, response costs, self-instruction/cognitive behavior training, and task or environmental

stimulation/biofeedback are practical approaches in managing children with the disorder.Interventions at school include seating the child near the teacher to minimize classroom distractions, or assigning a specific teacher to review daily assignments with the child in order to improve the behavior and academic performance of affected children. [69]Works when given in small quantities with breaks in between, [70] when novel tasks with multimodal presentation and decreased noise levels are found to enhance their performances. [71]

7.3 Social skills training

Social skills training have been found to cause improvement in behavior of ADHD children.

7.4 Medication and behavioral intervention combined

Treatment approaches of either medication or behavioral modification on its own have several shortcomings. These can be reduced when the two approaches are combined, with suggestions that the primary symptoms are most successfully treated by medication, and the secondary symptoms by behavioral approaches. [72]

VIII. Oral Manifestations In Children With ADHD

Children with ADHD have been found to be at a greater risk of developing carious lesions than control groups in terms of more decayed, missing and filled teeth as assessed by the DMFT index. [73]

Children with ADHD have a higher prevalence of mineralization disturbances compared to control groups in various studies. [74, 75]Higher rates of bruxism [74], were also found in children with ADHD especially among those medicated with amphetamine or methylphenidate. [76]Risk of sustaining traumatic dental injuries has also been reported to be higher in ADHD cases. [77]Amphetamine medication may predispose to gingival enlargement. [78]Obnoxious oral habits including nail biting, lip biting, and/or biting or chewing on other objects (pens and pencils) have been found. [74] Disruptive behavior, dyskinesia (tics) or hyperactivity following medications prescribed for ADHD can pose difficulties during a dental appointment. [76]Studies indicate that children with ADHD have inappropriate health behavior, such as higher risk for overeating and consumption of alcohol and tobacco use. [79, 80, 81, 82]Children with ADHD are more likely to exhibit significant anxiety associated with dental treatment. [83]Caffeinated beverages, including coffee and soda, which have been used for improving cognitive performance, renders an individual more susceptible to caries because of the sugar and acid contents in the same. [84]Non-effective tooth brushing has been found in these children due to difficulties with attention and starting and completing activities that are not simulating to them. [85, 86, 87]

In addition, there are often parent-child conflicts, which might influence their oral health behavior. [88]

IX. Treatment Considerations And Approaches In Dental Office

Considering the prevalence and chronic nature of ADHD, it is very likely that these patients will be seen in all dental practices.

- Treating the ADHD patient in the dental office requires knowledge of health history/medication implications, recognition of possible oral presentations, and flexibility in adjusting the treatment plan.
- Dentist should recommend the parent/caregiver to have a talk with the child regarding the dental appointment, taking caution as to avoid obnoxious terms like ‘shot’ ‘pain’ ‘drill’ etc.
- Timing the appointment is important. Many of the children will be under medications. ADHD medications should ideally be taken 30-60 minutes prior to the dental visit, under the influence of the drug, procedures can be carried out under enhanced cognition and behavior. [84] Between doses of short-acting stimulants, children are not covered by medication and thus may be highly symptomatic of ADHD. Further, they may experience “rebound” between doses. It is advisable to avoid dental appointments during this phase.
- ADHD children are inattentive, easily get distracted, have a short attention span and tend to forget instructions that come in a sequence. Therefore, instructions should be short, direct and issued one at a time. [89]
- It should be explained to the child at the outset of the visit as to what behavior is expected of him or her. Positive reinforcements including praising appreciable behavior and small, tangible rewards are likely to induce continued good behavior. [24]
- Behavioral contracting can also be tried by the clinician – either in verbal or written form. For example, the clinician might offer that when he or she is done treating the child’s teeth, then the child can play with his or her favorite toy for a brief period of time.
- Breaks are an important component in working with ADHD children. For guidelines on whether breaks will be necessary, how often to provide breaks, and duration of breaks, the clinician should consider consulting with the child’s parents. The child can be informed prior to starting procedure that breaks will be given during which the child will be able to play.
- Though the child would appreciate a break from the dental procedure, it will most probably be difficult getting the child back to the dental chair to resume the procedure after a break. It is always preferable to

give the child an advance notice to facilitate this transition. For example, the clinician can inform the child that the break is almost over, he can play for another couple of minutes and then he has to return to the dental chair. It should be kept in mind to praise the child if he sticks to the time and returns to the chair without fussing.

- Parents/caregivers can be consulted regarding the behavior, likes and dislikes of the child can be taken into consideration in an attempt to make the visit successful. [89]
- Consultation with primary care physician is extremely important especially in cases of multiple comorbid conditions.
- Comprehensive dental history should be recorded which includes history of oral habits, [74] bruxism, [76] dyskinesia/tics, smoking, self-medications, [84] caffeinated beverages, dietary habits including frequency of eating, types of snacks, drinks between meals etc. [88]
- Topical fluoride treatments should be considered to help impede the decay process.
- Local anesthesia should be used with caution. Attempts should be made at achieving profound anesthesia, so that endogenous epinephrine is not released which could react with medications (especially methylphenidate).
- Use of Tell-Show-Do technique of behavior management has been found to be useful in managing children with ADHD during a dental procedure. [90]
- Multiple short visits will be more successful than one prolonged visit.

X. Tables

Inattentive presentation:
• Fails to give close attention to details or makes careless mistakes.
• Has difficulty sustaining attention.
• Does not appear to listen.
• Struggles to follow through on instructions.
• Has difficulty with organization.
• Avoids or dislikes tasks requiring a lot of thinking.
• Loses things.
• Is easily distracted.
• Is forgetful in daily activities

Table 1. Symptoms of attention deficit as stated in DSM-5

Hyperactive-impulsive presentation:
• Fidgets with hands or feet or squirms in chair.
• Has difficulty remaining seated.
• Runs about or climbs excessively in children; extreme restlessness in adults.
• Difficulty engaging in activities quietly.
• Acts as if driven by a motor; adults will often feel inside like they were driven by a motor.
• Talks excessively.
• Blurts out answers before questions have been completed.
• Difficulty waiting or taking turns.
• Interrupts or intrudes upon others.

Table 2 Symptoms of hyperactivity as stated in DSM-5

XI. Conclusion

ADHD is a highly prevalent condition among children. It presents a clinical challenge to the oral health professionals. Oral health care and maintenance is compromised in children with ADHD. Professionals should understand that ADHD is a complex disorder that could require an adjustment in their treatment plan and daily schedule in order to deliver successful treatment and promote good oral health for this special group of children.

References

[1]. Swanson JM, Sergeant JA, Taylor E et al. Attention-deficit hyperactivity disorder and hyperkinetic disorder. Lancet 1998;351: 429-33

[2]. Kaplan, H. I.; Sadock, B. J. (2009). Kaplan & Sadock's comprehensive textbook of psychiatry. Philadelphia, Wolters Kluwer Health/Lippincott Williams & Wilkins

[3]. Scahill L, Schwab‐Stone M, Merikangas KR, Leckmaz JF, Zhang H, Kasl S. Psychosocial and clinical correlates of ADHD in a community sample of school‐age children. J Am Acad Child Adolesc Psychiatry 1999;38:976‐8

[4]. Venkata JA, Panicker AS. Prevalence of attention deficit hyperactivity disorder in primary school children. Indian J Psychiatry 2013;55:338-42

[5]. Pliszka SR. Patterns of psychiatric comorbidity with attention deficit/hyperactivity disorder. Child Adolesc Psychiatr Clin North Amer 2000; 9: 525-40

[6]. American Psychiatric Association. Diagnostic and statistical manual of mental disorders, 4th ed. Washington DC: American Psychiatric Association 1994

[7]. Biederman J, Faraone SV, Knee D, Munir K. Retrospective assessment of DSM-III attention deficit/hyperactivity disorder in nonreferred individuals. J Clin psychiatry 1990; 51: 102-6

[8]. (Lahey BB, Pelham WE, Stein MA et al. Validity of DSM-IV attention-deficit/hperactivity disorder for younger children. J Amer Acad Child Adolesc Psychiatry 1998; 37: 695-702

[9]. Bhatia MS, Nigam Vr, Bohra N, Mailk SC. Attention deficit disorder with hyperactivity among paediatric outpatient. J Child Psychol Psychiatry 1991; 32: 297-306

[10]. Malhi P, Singhi P. Spectrum of attention deficit hyperactivity disorder in children among referrals to psychology services. Indian Pediatr 2000; 37: 1258-60.

[11]. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders, 4thed. rev. Washington, DC: American Psychiatric Association; 2000

[12]. Still GF. The Goulstonian lectures on some abnormal psychical conditions in children. Lancet 1902;1:1008-12, 1077-82, 1163- 8.

[13]. Barkley RA (2006b) The relevance of the Still lectures to attention deficit/hyperactivity disorder: a commentary. J Atten Disord 10:137–140

[14]. Rothenberger A, Neuma¨rker KJ (2005) Wissenschaftsgeschichte der ADHS. Kramer-Pollnow im Spiegel der Zeit, Steinkopff, Darmstadt

[15]. Ross DM, Ross SA (1976) Hyperactivity: research, theory and action. Wiley, New York

[16]. Laufer MW, Denhoff E, Solomons G (1957) Hyperkinetic impulse disorder in children's behavior problems. Psychosom Med 19:38–49

[17]. Barkley RA (2006a) Attention-deficit hyperactivity disorder. A Handbook for Diagnosis and Treatment, Guilford, New York

[18]. Douglas VI (1984) Citation classic. Stop, look and listen: the problem of sustained attention and impulse control in hyperactive and normal children. Soc Behav Sci 44:16

[19]. American Psychiatric Association (1980) Diagnostic and statistical manual of mental disorders (DSM-III), 3rd edn. American Psychiatric Association, Washington DC

[20]. American Psychiatric Association (1987) Diagnostic and statistical manual of mental disorders (DSM-III-R), 3rd edn rev. American Psychiatric Association, Washington DC

[21]. Lahey BB, Applegate B, McBurnett K, Biederman J, Greenhill L,Hynd GW et al (1994) DSM-IV field trials for attention deficit hyperactivity disorder in children and adolescents. Am J Psychiatry 151:1673–1685

[22]. American Psychiatric Association (1994) Diagnostic and statistical manual of mental disorders (DSM-IV), 4th edn. American Psychiatric Association, Washington DC

[23]. American Psychiatric Association (2013). Diagnostic and statistical manual of mental disorders (DSM-5), Washington, D.C.: American Psychiatric Association

[24]. Efron LA, Sherman JA. Attention deficit disorder: implications for dental practice. Dent Today. 2005 Feb;24(2):134-139

[25]. Patricia Frese, R. D. H., and R. D. H. Elizabeth McClure. "Dental Implications of the ADHD Patient." Provider 501: 211886

[26]. Friedlander AH. Attention-deficit hyperactivity disorder: setting the record straight. Spec Care Dentist. 2004 Sep-Oct;24(5):249

[27]. Sherman DK, McGue MK, Iacono WG: Twin concordance for attention-deficit hyperactivity disorder: a comparison of teachers' and mothers' reports. Am J Psychiatry 1997;154:532–535

[28]. W. Chen, K. Zhou, P. Sham et al., "DSM-IV combined type ADHD shows familial association with sibling trait scores: a sampling strategy for QTL linkage," The American Journal of Medical Genetics Part B: Neuropsychiatric Genetics, vol. 147, no. 8, pp. 1450–1460, 2008

[29]. Sprich S, Biederman J, Crawford MH, et al: Adoptive and biological families of children and adolescents with ADHD. J Am Acad Child Adolesc Psychiatry 2000;39:1432–1437

[30]. Alberts-Corush J, Firestone P, Goodman JT: Attention and impulsivity characteristics of the biological and adoptive parents of hyperactive and normal control children. Am J Orthopsychiatry 1986;56: 413–423

[31]. Morrison JR, Stewart MA: A family study of the hyperactive child syndrome. Biol Psychiatry 1971;3: 189–195

[32]. Stewart MA, DeBlois CS, Cummings C: Psychiatric disorder in the parents of hyperactive boys and those with conduct disorder. J Child Psychol Psychiatry 1980;21:283–292

[33]. Biederman J, Faraone SV, Keinan K, Benjamin J, Krificher B, Moore C, and others. Further evidence for family-genetic risk factors in attention deficit hyperactivity disorder: patterns of comorbidity in probands and relatives in psychiatrically and pediatrically referred samples. Arch Gen Psychiatry 1992;49:728–38

[34]. Morrison JL, Stewart MA. A family study of the hyperactive child syndrome. Biol Psychiatry 1971;3:189–95

[35]. Maher BS, Marazita ML, Moss HB, et al: Segregation analysis of attention-deficit hyperactivity disorder. Am J Med Genet 1999;88:71–78

[36]. Schonwald A. Update: attention deficit/hyperactivity disorder in the primary care office. Curr Opin Pediatr. 2005 Apr;17(2):265-274.
[37]. Faraone SV, et al. Molecular genetics of attention-deficit/hyperactivity disorder. Biol Psychiatry 2005;57(11):1313–23
[38]. Faraone SV, Khan SA J Candidate gene studies of attention-deficit/hyperactivity disorder Clin Psychiatry. 2006;67 Suppl 8:13-20
[39]. Bobb, A.J., Castellanos, F.X., Addington, A.M., Rapoport, J.L. Molecular genetic studies of ADHD: 1991 to 2004.American Journal of Medical Genetics Part B: Neuropsychiatric Genetics. 2005;132B:109–125
[40]. Li Z,Chang S. H., Zhang L. Y., Gao L., Wang J. (2014b). Molecular genetic studies of ADHD and its candidate genes: a review. Psychiatry Res. 219, 10–24. 10.1016/j.psychres.2014.05.005
[41]. Banerjee TD, Middleton F, Faraone SV: Environmental risk factors for attention-deficit hyperactivity disorder.Acta Paediatr 2007, 96(9):1269-1274.
[42]. Nigg JT. What causes ADHD? Understanding what goes wrong and why. New York: The Guilford Press; 2006
[43]. Farias AC, Cunha A, Benko CR, et al. Manganese in children with attention-deficit/hyperactivity disorder: relationship with methylphenidate exposure. J Child Adolesc Psychopharmacol. 2010;20:113–118
[44]. Marks AR, Harley K, Bradman A, et al. Organophosphate pesticide exposure and attention in young Mexican-American children: the CHAMACOS study. Environ Health Perspect. 2010;118:1768–1774
[45]. Kim BN, Cho SC, Kim Y, et al. Phthalates exposure and attention-deficit/hyperactivity disorder in school-age children. Biol Psychiatry. 2009;66:958–963
[46]. Hoffman K, Webster TF, Weisskopf MG, Weinberg J, Vieira VM. Exposure to polyfluoroalkyl chemicals and attention deficit/hyperactivity disorder in U.S. children 12–15 years of age. Environ Health Perspect. 2010;118:1762–1767.
[47]. Schlotz W, Jones A, Phillips DI, et al. Lower maternal folate status in early pregnancy is associated with childhood hyperactivity and peer problems in offspring.J Child Psychol Psychiatr Allied Disc.2010;51:594–602
[48]. Nigg JT, Hinshaw SP. Parent personality traits and psychopathology associated with antisocial behaviors in childhood attention-deficit hyperactivity disorder. J Child Psychol Psychiatry 1998; 39(2): 451-466)
[49]. Edwards MC, Schulz EG, Long N. The role of the family in the assessment of attention deficit hyperactivity disorder. Clin Psychol Rev 1995; 15: 375-394
[50]. Barkley RA. Attention deficit/hyperactivity disorder, self regulation and time: Toward a more comprehensive theory. Dev Behav Paediatr 1997; 18: 271-279
[51]. Teeter PA, Semrud-Clikeman M. Integrating neurobiological, psychosocial,and behavioral paradigms: A transactional model for the study of ADHD. Arch Clin Neuropsychol 1995; 10: 433-461
[52]. Biederman J, Newcorn J, Sprich S. Comorbidity of attention deficit hyperactivity disorder with conduct, depressive anxiety, and other disorders. Am J Psychiatry 1991b; 148: 564-577.
[53]. Williams C, Wright B, Partridge I. Attention deficit hyperactivity disorder--a review. The British Journal of General Practice. 1999;49(444):563-571
[54]. Goodman R. The strengths and difficulties questionnaire: a research note. J Child Psychol Psychiatry 1997; 38: 581-586
[55]. Achenbach TM. Manual for the Child Behavior Checklist 4-18 and 1991 Profile. Burlington, VT: University of Vermont, Dept of Psychiatry, 1991
[56]. Conners CK. Rating Scales for use in Drug Studies with Children. Pharmacol Bull 1973; 9: 24-29
[57]. Berg I, Lucas C, McGuire R. Measurement of behavior difficulties in children using scales administered to mothers by computer. Reliability and validity. Eur J Child Adolesc Psychiatry 1992; 1: 14-23
[58]. Rutter M. A children's behavior questionnaire for completion by teachers. Preliminary findings. J Child Psychol Psychiatry 1967; 8 1-11.
[59]. Barkley R. Attention Deficit Hyperactivity Disorder: A handbook for diagnosis and treatment. New York: Guilford Press, 1990
[60]. The MTA Cooperative Group. A 14-month randomised clinical trial of treatment strategies for attention-deficit/hyperactivity disorder. Arch Gen Psychiatry 1999;56:1073–86.
[61]. Barkley RA. A review of stimulant drug research with hyperactive children. Arch Gen Psychiatry 1977; 18: 137-165)
[62]. Ottenbacher KJ, Cooper HM. Drug treatment of hyperactivity in children. Dev Med Child Neurol 1983; 25: 358-366
[63]. Elia J, Borcherding BG, Rapoport JL. Methylphenidate and dextroamphetamine treatments of hyperactivity: are there true nonresponders? Psych Res 1991; 36: 141-155
[64]. Elkins RN, Rapoport JL, Zahn TP. Acute effects of caffeine in normal prepubertal boys. Am J Psychiatry 1981; 138: 178-183
[65]. Reeves G, Schweitzer J. Pharmacological management of attention-deficit hyperactivity disorder. Expert Opin Pharmacother 2004;5:1313– 20
[66]. Christman AK, Fermo JD, Markowitz JS. Atomoxetine, a novel treatment for attention-deficithyperactivity disorder. Pharmacotherapy 2004;24:1020–36.
[67]. Pelham WE Jr, Fabiano GA. Behavior modification. Child Adolesc Psychiatr Clin N Am 2000;9:671– 88
[68]. Karande S. Attention deficit hyperactivity disorder: A review for family physicians. Indian J Med Sci 2005; 59:547-56
[69]. Hoffman JB, DuPaul GJ. Psychoeducational interventions for children and adolescents with attention-deficit/hyperactivity disorder. Child Adolesc Psychiatr Clin N Am 2000;9:647–61
[70]. Zentall SS, Dwyer AM. Colour effects on the impulsivity and activity of hyperactive children. J School Psychol 1998; 23: 83-93.
[71]. Zentall SS, Shaw JH. Effects of classroom noise on performance and activity of second grade hyperactive and control children. J Educ Psychol 1980; 72: 830-840
[72]. Pelham WE. Pharmocotherapy for children with attention deficithyperactivity disorder. School Psychol Rev 1993; 22: 199-227
[73]. Broadbent JM, Ayers KM, Thomson WM. Is attention-deficit hyperactivity disorder a risk factor for dental caries? A case-control study. Caries Res 2004; 38: 29-33
[74]. Atmetlla G, Burgos V, Carrillo A, Chaskel R. Behavior and orofacial characteristics of children with attention-deficit hyperactivity disorder during a dental visit. J Clin Pediatr Dent 2006; 30: 183-90
[75]. Noren JG, Gillberg C. Mineralization disturbances in the deciduous teeth of children with so called minimal brain dysfunction. Swed Dent J 1987; 11: 37-43
[76]. Malki GA, Zawawi KH, Melis M, Hughes CV. Prevalence of bruxism in children receiving treatment for attention deficit hyperactivity disorder: a pilot study. J Clin Pediatr Dent. 2004 Fall;29(1):63-67
[77]. Sabuncuoglu O, Taser H, Berkem M. Relationship between traumatic dental injuries and attention-deficit/hyperactivity disorder in children and adolescents: proposal of an explanatory model. Dent Traumatol 2005; 21:249-53
[78]. Hasan AA, Ciancio S. Relationship between amphetamine ingestion and gingival enlargement. Pediatr Dent 2004; 26: 396-400

[79]. Upadhyaya HP, Rose K, Wang W, O'Rourke K, Sullivan B, Deas D,Brady KT. Attention-deficit/hyperactivity disorder, medication treatment,and substance use patterns among adolescents and young adults. J Child Adolesc Psychopharmacol 2005; 15: 799-809
[80]. Whalen CK, Jamner LD, Henker B, Delfino RJ, Lozano JM. The ADHD spectrum and everyday life: experience sampling of adolescent moods, activities, smoking, and drinking. Child Dev 2002; 73: 209-27.
[81]. Molina BS, Pelham WE, JR. Childhood predictors of adolescent substance use in a longitudinal study of children with ADHD. J Abnorm Psychol 2003;112: 497-507
[82]. Cortese S, Isnard P, Frelut ML, Michel G, Quantin L, Guedeney A, Falissard B, Acquaviva E, Dalla Bernardina B, Mouren MC. Association between symptoms of attention-deficit/hyperactivity disorder and bulimic behaviors in a clinical sample of severely obese adolescents. Int J Obes (Lond) 2007; 31: 340-6
[83]. Carlsson V, Hakeberg M, Blomkvist K, Wide Boman U. Attention deficit hyperactivity disorder and dental anxiety in adults: relationship with oral health. Eur J Oral Sci. 2013 Jun;121:258-263
[84]. Friedlander AH, Yagiela JA, Paterno VI, Mahler ME. The pathophysiology, medical management, and dental implications of children and young adults having attention-deficit hyperactivity disorder. J Calif Dent Assoc. 2003 Sep;31(9):669-678.
[85]. Holbrook WP, Arnadottir IB, Takazoe I, Birkhed D, Frostell G.Longitudinal study of caries, cariogenic bacteria and diet in children just before and after starting school. Eur J Oral Sci 1995; 103: 42-5.
[86]. Holt RD. Foods and drinks at four daily time intervals in a group of young children. Br Dent J 1991; 170: 137-43
[87]. Kandelman D. Sugar, alternative sweeteners and meal frequency in relation to caries prevention: new perspectives. Br J Nutr 1997; 77 (Suppl): 121S-8S.
[88]. Blomqvist M, Holmberg K, Fernell E, Ek U, Dahllöf G. Dental caries and oral health behavior in children with attention deficit hyperactivity disorder. Eur J Oral Sci. 2007 Jun;115(3):186-191
[89]. University of Washington, School of Dentistry. Patients with special needs, dental fact sheet for children. Accessed June 3, 2013
[90]. Felicetti DM and Julliard K Behaviors of children with and without attention deficit hyperactivity disorder during a dental recall visit. J Dent Child 2000 67:246-249