Sensory Processing in Adult ADHD – A Systematic Review

Marcel Schulze (marcel.schulze@ukbonn.de)
Universitatsklinikum Bonn  https://orcid.org/0000-0001-8522-7156

Silke Lux
Universitatsklinikum Bonn

Alexandra Philipsen
Universitatsklinikum Bonn

Research article

Keywords: Attention-Deficit-Hyperactivity Disorder (ADHD), sensory processing, bottom-up vs, top-down, adult, electrophysiology, brain imaging

DOI: https://doi.org/10.21203/rs.3.rs-71514/v1

License: © This work is licensed under a Creative Commons Attribution 4.0 International License.
Read Full License
Abstract

Background

The way we perceive our environment is driven by our sensory nervous system and our attentional resources. Attention-deficit/hyperactivity disorder (ADHD) is a neurodevelopmental disorder characterized by inattention, impulsivity, and hyperactivity. While cognitive and behavior dysfunctions have broadly been investigated, sensory processing has received less scientific attention. It has been shown, that children with ADHD show processing and modulatory deficits in multiple sensory domains, but very few studies examine to what extent these deficits persist in adult life. We conducted a systematic review of studies investigating sensory processing in adult ADHD.

Main Body

Using the keywords ‘ADHD’ and ‘sensory processing’, Web of Science and MEDLINE database were systematically searched for all articles published up to March 2020. 53 studies were included. Mostly, visual and auditory processing are studied, few investigated multisensory audiovisual and somatosensory processing. In summary, adult ADHD is marked by increased sensory gaining and deficient sensory inhibition. These disturbed gaining and inhibitory mechanisms were most prominent in the auditory modality but also visual modality impairment in terms of stimuli modulation were evident. Electrophysiological studies show alterations across all event-related potential (ERP) components associated with distractibility at early components (bottom-up) and inhibition and stimulus discrimination at later components (top-down). Brain imaging studies on sensory processing in ADHD are scarce, few pointing to higher resting state functional connectivity in visual areas and visual crossmodal activation for auditory stimuli.

Conclusion

Sensory processing deficits extent from childhood to adult ADHD. These deficits are mainly driven by higher distractibility by irrelevant stimuli and modulatory impairment for relevant stimuli. In future studies, the relation of impaired bottom-up and top-down attentional mechanisms should be investigated and how they contribute to sensory processing deficits and clinical symptomatology in adult ADHD. This could help to gather more information about the underling processing deficits, so that specific adjusted training can be provided, that helps to overcome deficits in daily life functioning in e.g., not producing appropriate adaptive responses in social settings.

Trial registration

N/A

Background
Attention-deficit/hyperactivity disorder (ADHD) is a neurodevelopmental disorder characterized by inattention, impulsivity, and hyperactivity [1]. While ADHD has long been considered as a childhood disorder, evidence points to an ongoing course into adult life. Symptoms of impulsivity and hyperactivity have been shown to decrease whereas inattention tends to persist [2]. Associated with the main symptoms of ADHD, patients suffer from deficits in executive functioning. Working memory impairments is of the most robust finding causing impairment in patients’ daily life [3, 4]. While the main symptoms, executive functioning as well as their neuronal underpinnings has been subject of many investigations recently, one area that has been rarely investigated in adult ADHD patients is sensory processing. In order to perceive environmental stimuli properly, our nervous system constantly has to receive, integrate and organize sensory input [5, 6]. Further, modulation in terms of an adaptive inhibition or an increasing degree of processing is necessary [7]. The frontal cortex assumes to be related to the role of behaviour-guiding function and is therefore dependent on input of sensory association areas. Deficient connectivity within frontal brain areas and between frontal areas and sensory association areas is frequently reported in ADHD [8]. As a consequence, sensory input may not be properly regulated hence have consequences for higher order cognitive functioning e.g., working memory and planning [9].

With respect to Dunn's model of sensory processing, each individual's behaviour to sensory stimuli is determined by the neurological sensitivity threshold and the corresponding responding strategy. A low registration of environmental stimuli is marked by a high detecting threshold together with passive responding strategies. Those individuals with high thresholds and active responding strategies can be considered as sensory seeking. In contrast, sensory sensitivity goes along with low threshold and passive responding strategies. Having a low threshold with active responding strategies can be considered as sensory avoiding [10]. Of note, a person's ability to process sensory events is not categorical per se rather can be highly differentiated by e.g., having a sensitivity to certain sensory events while being avoidant to other. With the help of this model, a sensory profile of a person's sensory processing abilities can be compiled. Numerous studies show that children having ADHD can be distinguished in their sensory profile from children without any disability [11]. ADHD children show sensory processing deficits and modulatory difficulties by scoring lower in the visual, auditory (with increasing issues over time), touch, taste/smell, multisensory, emotional and social responses [11–13]. It can be assumed that this impairment is responsible for not producing appropriate adaptive responses at school, at home and in social settings [14]. Moreover, current studies assume that a low threshold for sensory stimuli is associated with distractibility (especially for the auditory domain) whereas a high threshold could be attributed to inattentive behaviour, since certain stimuli will be neglected [14].

On a neuronal level, smaller cerebral volume in frontal and prefrontal cortices were associated with altered brain activation in sensory cortices (auditory, visual and somatosensory areas) [9, 15, 16].

At a neuronal level, the perception of a stimulus is a complex interplay of an early analysis of stimulus features as well as integration of this information in higher order cortical areas for further processing. To generate a percept, attention is necessary which can be divided in bottom-up (sensory driven) and top-down (sensory modulation from higher cortical areas) [17].
threshold, primary sensory cortices trigger attentional resources by recruiting ‘higher order’ brain areas (from unisensory to heteromodal association areas to parietal and frontal regions) [17, 18]. Attentional top-down mechanisms enable a selective process by binding several stimuli categories (attention specific perceptual binding, see [19]), reweight sensory information further distinguishing noise from act-relevant stimuli [20, 21] and faster/ more accurate responses [22].

The following review gives an overview of current studies of sensory processing in adult ADHD. Furthermore, it will be discussed whether sensory perception in ADHD might be a deficient process in a bottom-up manner (by failing at the early stages of stimuli processing as well as to capture attentional deployment of higher areas) or whether sensory perception in ADHD is marked by impaired top-down processing (by failing e.g., stimuli enhancement or reweighting of stimuli). To elucidate a potential deficit of bottom-up and top-down attention, the current review also considers electrophysiological and neuroimaging findings.

**Methods**

Web of Science and MEDLINE database were systematically searched for all articles published up to March 2020. The keywords used were ‘ADHD’, ‘attention deficit hyperactivity disorder’, ‘adult ADHD’, ‘sensory’, ‘sensory processing’ as well as all possible combinations. Articles have to be published peer-reviewed and written in English. Four hundred twenty abstracts were retrieved and scanned.

Reference lists of obtained articles were also considered. Finally, 53 studies were included. This review was performed according to the PRISMA guidelines.

**Results**

1.1 Behavioral Studies

There has been a long tradition considering ADHD as a discrete entity. However, attentional resources and the respective variation in activation level or perception fall on a continuum with different inter-individual distributions. Therefore, recent developments consider ADHD as one extreme on a continuum negating a categorical view [23, 24]. By measuring ADHD traits in a large cohort where most of them have not received a diagnosis in the past supports a dimensional point of view. Panagiotidi et al. 2017 assessed sensory responsivity across sensory domains and relates these capacities to ADHD traits in a sample of students (n=234) [25]. Sensory qualities were assessed with the Glasgow Sensory Questionnaire (GSQ; [26]) allowing the investigation for hyper-and hyposensitivity across sensory modalities. As a result, GSQ scores on all sensory modalities were positively correlated with ADHD traits. Also, ADHD-traits and age were a robust predictor for GSQ suggesting that ADHD-traits are associated with altered sensory responsiveness. In diagnosed ADHD patients these sensory processing deficits were demonstrated in terms of perceptual modulation (e.g., being flooded by sensory events), distractibility (e.g., difficulties to focus when background noise is present) and over-inclusion (e.g., noticing slightest sound changes in the
This finding is further supported by a survey study from Bijlenga et al. 2017 who screened 116 diagnosed adult ADHD patients [28]. Low registration and less sensation seeking behavior was reported. The latter contradicts the clinical practice of ADHD where patients often report high sensation seeking. Less sensation seeking behavior as well as avoidance of certain sensory events may be a consequence of a lower detection threshold [28].

Auditory processing

Overall highest sensitivity was found for the auditory modality (marked by the inability to suppress irrelevant noises e.g., footsteps in the background while doing another task) [29]. Auditory hypersensitivity is in line with a finding that processing deficits in this modality seem to increase with age in children with ADHD whereas processing in other modalities seem to improve slightly [12]. In a study comparing auditory temporal thresholds (assessed by judging the order of incoming dichotic tones) between unmedicated ADHD patients, medicated ADHD and healthy controls, Fostick et al could show higher temporal order judgement thresholds in unmedicated adult ADHD patients [30]. Of note, under methylphenidate (MPH), medicated patients’ thresholds decreased similar to healthy controls. However, it is challenging to disentangle, whether MPH directly influences early sensory processing or whether it has an indirect effect on sensory processing by modulating later stages. One study investigating the effect of MPH on various measures of attention shows that MPH had a beneficial effect on alertness, selective attention, divided attention but not on integration of sensory information (i.e. the process of combining different sensory modalities) [31]. This might be a hint that early sensory processing is not influenced by MPH. In a combined MEG/EEG experiment Korostenskaja et al (2008) demonstrated that mismatch-negativity (MMN, reflective for pre-attentive detection of stimulus) is unaffected under MPH in healthy participants but MPH may be beneficial for modulatory (e.g., top-down selection attention on stimulus features), processes of the stimuli (but see below for electrophysiological sensory processing in ADHD) [32].

Visual processing

In the visual domain, ADHD-traits were tested in adult healthy participants to study reaction time costs when presented peripheral checkerboards distractors in a sustained attention to response task (SART) [33]. As a result, higher ADHD traits were related to less distraction. Since the distractor always appeared at a fixed time before stimulus presentation, the authors argued that the distractor served as a cue which allowed participants having high ADHD-traits to allocate attentional resources. This supports the idea that children with ADHD show optimal performance when noise or other types of stimuli are available [34]. The authors of the study argued that by reducing the distractor-stimulus interval, the beneficial effects for participants having high ADHD traits would be diminished. Indeed, as soon as the cueing effect of the distractor was removed, performance changed similar to those with low ADHD-scores. This suggests that cued stimuli that occur at a specific time interval triggers attentional allocation more beneficial for those scoring high on ADHD traits [21]. The higher sensitivity in the peripheral visual system
was reasoned with an enhanced activity of the superior colliculus, which is associated with higher
distractibility in ADHD [33, 35].

In a visual crowding perceptual interference task Stevenson et al. showed that ADHD patients are more
sensitive to perceptual interference (i.e. fail to suppress distractors) when visual crowding is increased
[36]. Of note, the authors controlled for attention allocation hence this finding is supporting bottom-up
difficulties in adult ADHD.

In ADHD children, several ophthalmologic deficits have been found e.g., color perception [37, 38].
Especially the short-wavelength cones (perception of the color ‘blue’) are affected in ADHD which is
reasoned with the retinal dopaminergic hypothesis by Tannock et al. 2006 [39]. Here, abnormal
dopamine-levels are assumed to induce a hypo-dopaminergic tone in the retina leading to deficits in
perception of the short-wavelength (since these cones show sensitivity to dopamine) [37, 39]. A deficit in
perception of the blue spectrum was also present in adults with ADHD [40]. Further, visual deficits were
reported for in-depth perception, peripheral vision, visual search and visual processing speed [40].

Multisensory audiovisual processing

Two studies investigated the effects of multisensory (i.e. audiovisual) processing in adult ADHD yield to
mixed results. In Michalek et al 2014 understanding speech in noise led to comparable results in patients
and a healthy control group. However, by adding the speaker’s faces as visual cues, speech-to-noise
understanding in patients was not enhanced, while the controls benefited from the additional visual
information [41]. This suggests a deficient process in the neural integration of multisensory cues for adult
ADHD patients. In contrast, such a deficit was not found when comparing responses to unisensory
auditory, unisensory visual with multisensory audiovisual cues in ADHD. In theory, one would expect
longer reaction times in multisensory scenarios compared to unisensory events, if multisensory
integration does not take place properly. In the study by McCracken et al. 2019, patients and controls were
similarly able, to integrate multisensory cues, reflected in faster reaction times for multisensory cues
compared to unisensory cues [42]. More studies in the field of multisensory integration are necessary to
elucidate the ability to bind different modalities to form a unified percept in adult ADHD.

Sensorimotor processing

In ADHD-children lower sensory-motor abilities and motor coordination was reported compared to healthy
controls [13]. Two studies investigated postural sway (assessed with balanced boards) in adult ADHD.
Compared to controls, higher postural sway was found indicating that sensorimotor deficits extent from
childhood to adulthood. These postural abnormalities were associated with hyperactivity/impulsivity
rather than with inattention [43, 44].

1.2 Electrophysiological findings on sensory processing

Electroencephalography (EEG) allows to study cell activity within milliseconds range with event-related
potentials (ERP; the averaged pyramidal cell activity time-locked to the stimuli) [45]. Commonly studied
ERP's in the context of stimulus perception and modulation are components such as P50 (a positive deflection 50ms after stimulus onset), N1 (negative deflection 100ms after stimulus onset), P1, P2 and N2. Later ERP's e.g., P3 already are considered to be involved in top-down cognition e.g. attention allocation [46]. The perceptual process is a fine-tuned process which involves filtering of sensory information (sensory gating capacities marked by P50 ERP;[47]), extraction of relevant sensory information (N1 ERP), further processing or automatic stimulus discrimination (P2 ERP), and endogenous mismatch-detection process related to stimulus discrimination (N2 ERP; commonly known as mismatch-negativity-MMN) [48, 49]).

Adult ADHD patients often report being flooded by sensory input [50]. Higher sensory gating was associated with an abnormal P50 suppression across multiple studies [29, 51, 52], while one study did not find a difference between ADHD, Schizophrenia and normal controls [53]. Most recently, higher distractibility as measured with P50 was found to be inversely correlated with the P3 ERP which indicates that attention allocation cannot take place properly, since higher distractibility hinders a proper attentional selection hence difficulties to focus [51, 54]. A disruptive process on stimuli filtering also support the pathophysiology of prefrontal-cortex maturation according to Halperin & Schulz et al. 2006 [55]. Here, higher order executive and attentional deficits are considered to be the consequence of a disrupted lower sensory processing mechanism.

In an experiment conducted with visual checkerboards and auditory tone stimuli, Gonen et al. investigated P1 and N1ERP's [56]. While the averaged components did not differ between adult ADHD patients and controls respectively, larger trial-to-trial variability in both P1/N1 were found. This enhanced trial-to-trial variability was reasoned with a higher fluctuation in neural activity generally found in ADHD. A higher fluctuation in neural activity underlies an impaired mechanism of the default mode system, which is an interplay of brain areas usually suppressed in the presence of a task [57].

In an intermodal oddball task, Barry et al 2009 revealed increased auditory N1, P2, and smaller N2 activity in ADHD patients compared to healthy controls [48]. For the visual domain mixed results are reported, some indicating smaller P1, increased P2, increased N1, while others some show responses similar to healthy controls [48, 58–61]. Overall, smaller P3 activity was reported [48, 60, 62]. Increased P2 responses accompanied with delayed peak latencies between 130 to 350 ms post-stimulus were found in a pop-out search task, further supporting the hypothesis that attention selection of relevant stimuli features is deficient in ADHD patients [58]. In summery, the findings outlined above seem to represent deficient inhibitory processing in conjunction with an overall heightened activity in the mismatch-detection process for target stimuli and an inappropriate allocation of attentional resources.

While most studies investigate the visual and auditory modality, two studies from Dockstader et al investigate somatosensory processing in adult ADHD. Somatosensory processing is reflected in the sensorimotor oscillations of 8-12Hz, also known as mu rhythm, which is suppressed during movement preparation (mu rhythm-event-related desynchronization; mu-ERD). The ERD-reactivity pattern is shown to
be lowered in adult ADHD patients which might have consequences for attentional alerting when an unexpected stimulus occurs [63, 64].

To sum up: ERP components associated with auditory and visual processing respectively, show alterations, some leading to mixed results. These alterations are associated with higher distractibility at early components as well as inhibition and stimulus discrimination process at later components. Future studies should investigate not only single components but focus on the whole time-course allowing for estimating the relationship between early stimulus detection and later stimuli processing. Further, somatosensory processing as abnormal regulated as reflected in the mu rhythm. The behavioral consequences of a diminished mu rhythm remain elusive.

1.3 Brain-imaging findings of sensory processing

Resting-state functional connectivity (rsFC) is a correlational measure of activity between brain areas without an external task given [8, 65]. In children with ADHD, one study investigated rsFC of primary sensory areas to higher order attentional networks [66]. Results demonstrated higher rsFC of primary sensory areas to its neighboured areas and reduced rsFC to attention-regulatory networks compared to controls. This might reflect enhanced sensitivity to sensory events at rest and a disrupted between-network communication to attention-related areas. Our systematic literature search yields no study addressing bottom-up sensory rsFC and its relation to attentional networks in adult ADHD. In healthy participants, sensory hypersensitivity was linked to reduced dopamine levels in several brain regions. Especially the precuneus seem to play a role in suppression of genes responsible for sensory processing sensitivity [67]. The precuneus as part of the default mode network is consistently associated with weaker within-network connectivity and stronger connectivity to other networks compared to healthy controls [8, 68, 69]. Therefore, we only can speculate that in adult ADHD sensory information may be abnormally integrated and regulated via the precuneus to higher order processing areas. Future studies are needed to explore the role of the precuneus in bottom up sensory processing in ADHD.

One study found enhanced rsFC in visual sensory processing areas and regions involved in somatosensory processing. As the authors state in their paper, a possible explanation of this finding is that ADHD patients are more delay avers than healthy controls during the scanning, therefore allocate their attention to the environment [70].

In a functional magnetic resonance imaging (fMRI) study, Salmi et al. disentangled sensory bottom-up processing (assessed by visual and auditory discrimination tasks) from top-down processing (divided attention, focused attention) [69]. During the auditory task, enhanced visual cross-modal activation was found, whereas this was not evident during visual stimulation in auditory cortices. This finding is contrary to studies done in healthy participants, where a decreased activation in the unattended modality is reported [71]. In the focused attention task and divided attention task, higher activation in cuneus, precuneus and posterior cingulate cortex (PCC) was found. In conclusion this study demonstrates deficits in bottom-up sensory attention and top-down attentional selection. In future studies, clarification is
needed regarding crossmodal activation whether this reflects a deficient suppressing mechanism of visual bottom-up sensation [69].

On a structural level, voxel-based morphometry (VBM) revealed smaller grey matter volumes in Brodmann areas 17 and 18 involved in primary visual processing (V1, V2) [72]. These gray matter volumes were inversely correlated with symptoms of ADHD during childhood. From this study, it cannot be concluded whether this volume reduction can be associated with early sensory stimuli analysis (the authors of the study did not obtain functional visual data to obtain visual impairments) or top-down attentional modulation because V1,V2 are already considered as part of the visual attentional network [73–75].

To sum up: Few neuroimaging studies are available investigating sensory processing in adult ADHD. Existing evidence point to bottom-up (marked by a possible dysfunctional inhibition of the irrelevant sensory modality) difficulties, as well as to a top-down attentional selection dysfunction. Future studies are needed to clarify e.g., whether bottom-up deficiencies arise as a consequence of abnormal within- and between network rsFC which is not properly down-regulated at task.

**Discussion**

The aim of our systematic review was to summarize the findings on sensory processing in adult ADHD.

Most of the current studies focus on the auditory/visual modality leaving a research gap for other sensory modalities. Available studies point to impaired sensory processing regarding enhanced distractibility and modulating impairments such as stimulus feature discrimination or inhibition of sensory information. The majority of studies indicate an auditory hypersensitivity, while the evidence of such a hypersensitivity is lower for the visual modality. It remains unclear whether this is due to the inherent feature of the auditory sense that processing of input is involuntary (automatic). In contrast, exogenous input to the visual modality may cause attention shifting, which implies voluntary action e.g., turning the head towards a stimulus [76, 77]. This can explain why the auditory modality is generally more prone to certain distractors but it cannot account for the difference between ADHD-patients and neurotypical controls. Therefore, one important issue for future studies is to study the mechanism behind the auditory hypersensitivity in adult ADHD.

Perceptual processing in adult ADHD is accompanied with numerous irregularities at the electrophysiological and haemodynamic level. As described in the paragraphs above, these irregularities suggest impaired bottom-up and top-down attentional mechanisms. The role of these disturbed bottom-up/top-down attentional mechanism for sensory processing is still understudied and not well understood. It is possible that an early sensory processing deficit (early ERP-components), i.e., no proper filtering of incoming stimuli, leads to poor allocation of attention (late ERP-components) and, hence, to an overall higher sensitivity to other incoming stimuli. A failure in suppressing irrelevant sensory modalities may have consequences for higher order areas processing (fronto-parietal regions) that cannot weight the input properly leading to impaired stimulus integration.
Dependent on the perceptual load (i.e., the amount of information involved in processing task-relevant stimuli), attentional selection occurs at early stages when the sensory demand is high and at later stages when sensory demand is low [78–80]. To achieve efficient behavioral responses to environmental cues, the process of early vs. late attentional selection is determined by a network of prefrontal and parietal brain regions constantly adapting to high or low loads [81]. In adult ADHD it is unclear, whether these patients have a higher baseline perceptual load due to higher sensory gaining. In theory this would lead to enhanced top-down processing in order to overcome sensory overload. However, since top-down attention in ADHD is abnormally regulated, it might be that the switching mechanism between early vs late selection is impaired as well. Future studies are needed to identify a possible enhanced baseline-perceptual load and whether switching between early vs late selection is impaired.

Sensory processing difficulties are also reported in other psychiatric disorders such as autism, anxiety, bipolar disorder depression, and schizophrenia [82–85]. Therefore, it is questionable whether sensory processing deficits are ADHD-specific. Since ADHD shares common pathophysiologic neurocircuitry with sensory processing disorder, as well as with other neuropsychiatric diagnoses [86, 87], one is tempted to consider sensory processing deficits as something nonspecific to ADHD. However, such a consideration would again support a categorical thinking of sensory processing rather than regarding it as an added dimension on a continuum [88, 89].

**Conclusion**

This review demonstrates that the relationship of sensory processing deficits to the core symptoms of ADHD is not properly understood yet. It is therefore necessary to investigate sensory processing in adult ADHD more detailed. Especially, the influence of impaired bottom-up sensory processing to top-down attentional selection should be targeted in future studies. This could help to gather more information about the underling processing deficits, so that specific adjusted training can be provided, that helps to overcome deficits in daily life functioning in e.g., not producing appropriate adaptive responses in social settings.

**Abbreviations**

ADHD – Attention-deficit/hyperactivity disorder

DMN – default mode network

EEG – Electroencephalography

ERP – event-related potential

fMRI – functional magnetic resonance imaging

GSQ – Glasgow Sensory Questionnaire
MEG – Magnetoencephalography

MMN – Mismatch-negativity

MPH – Methylphenidate

rsFC – resting-state functional connectivity

SART – sustained attention to response task

Declarations

Ethics approval and consent to participate

Not applicable

Consent for publication

Not applicable

Availability of data and materials

N/A (review article).

Competing Interests

AP declares that she served on advisory boards, gave lectures, performed phase 3 studies and received travel grants in the last 3 years from Eli Lilly and Co., Lundbeck, MEDICE Arzneimittel, Pütter GmbH and Co. KG, Novartis, Servier and Shire; she has authored books and articles on ADHD published by Elsevier, Hogrefe, Schattauer, Kohlhammer, Karger and Springer.

Funding

No funding was obtained for this study.

Authors’ Contribution

MS: Literature search, writing.

SL, AP: writing, supervision.

All authors have read and approved the manuscript.

Acknowledgements

Not applicable
References

1. Hoogman M, Bralten J, Hibar DP, Mennes M, Zwiers MP, Schweren LSJ, et al. Subcortical brain volume differences in participants with attention deficit hyperactivity disorder in children and adults: a cross-sectional mega-analysis. The Lancet Psychiatry. 2017.

2. Uchida M, Spencer TJ, Faraone S V, Biederman J. Adult outcome of ADHD: an overview of results from the MGH longitudinal family studies of pediatrically and psychiatrically referred youth with and without ADHD of both sexes. J Atten Disord. 2018;22:523–34.

3. Willcutt EG, Nigg JT, Pennington BF, Solanto M V, Rohde LA, Tannock R, et al. Validity of DSM-IV Attention Deficit/Hyperactivity Disorder Symptom Dimensions and Subtypes. J Abnorm Psychol. 2012;121:991–1010.

4. Matt Alderson R, Kasper LJ, Hudec KL, Patros CHG. Attention-deficit/hyperactivity disorder (ADHD) and working memory in adults: A meta-analytic review. Neuropsychology. 2013.

5. Hebert K. The association between impulsivity and sensory processing patterns in healthy adults. Br J Occup Ther. 2015;78:232–40.

6. Miller LJ, Anzalone ME, Lane SJ, Cermak SA, Osten ET. Concept evolution in sensory integration: A proposed nosology for diagnosis. American Journal of Occupational Therapy. 2007.

7. Miller LJ, Lane SJ. Toward a consensus in terminology in sensory integration theory and practice: part 1: taxonomy of neurophysiological processes. Sens Integr Spec Interes Sect Quaterly. 2000.

8. Gao Y, Shuai D, Bu X, Hu X, Tang S, Zhang L, et al. Impairments of large-scale functional networks in attention-deficit/hyperactivity disorder: A meta-analysis of resting-state functional connectivity. Psychol Med. 2019.

9. Dionne-Dostie E, Paquette N, Lassonde M, Gallagher A. Multisensory integration and child neurodevelopment. Brain Sci. 2015;5:32–57. doi:10.3390/brainsci5010032.

10. Dunn W. The Sensations of Everyday Life: Empirical, Theoretical, and Pragmatic Considerations Background Knowledge Related to Sensory Processing. Am J Occup Ther. 2001.

11. Dunn W, Bennett D. Patterns of sensory processing in children with Attention Deficit Hyperactivity Disorder. OTJR-OCUPATION Particip Heal. 2002;22:4–15.

12. Cheung PPP, Siu AMH. A comparison of patterns of sensory processing in children with and without developmental disabilities. Res Dev Disabil. 2009;30:1468–80. doi:10.1016/j.ridd.2009.07.009.

13. Ghanizadeh A. Sensory processing problems in children with ADHD, a systematic review. Psychiatry Investig. 2011;8:89–94. doi:10.4306/pi.2011.8.2.89.

14. Shimizu VT, Bueno OFA, Miranda MC. Sensory processing abilities of children with ADHD. Brazilian J Phys Ther. 2014;18:343–52. doi:10.1590/bjpt-rbf.2014.0043.

15. Xavier Castellanos F, Lee PP, Sharp W, Jeffries NO, Greenstein DK, Clasen LS, et al. Developmental trajectories of brain volume abnormalities in children and adolescents with attention-deficit/hyperactivity disorder. J Am Med Assoc. 2002.
16. Castellanos FX, Giedd JN, Marsh WL, Hamburger SD, Vaituzis AC, Dickstein DP, et al. Quantitative brain magnetic resonance imaging in attention-deficit hyperactivity disorder. Arch Gen Psychiatry. 1996.

17. Sarter M, Givens B, Bruno JP. The cognitive neuroscience of sustained attention: Where top-down meets bottom-up. Brain Research Reviews. 2001.

18. Mesulam M-M. Large-scale neurocognitive networks and distributed processing for attention, language, and memory. Ann Neurol. 1990.

19. Degerman A, Rinne T, Pekkola J, Autti T, Jääskeläinen IP, Sams M, et al. Human brain activity associated with audiovisual perception and attention. Neuroimage. 2007.

20. Pessoa L, Kastner S, Ungerleider LG. Neuroimaging studies of attention: From modulation of sensory processing to top-down control. Journal of Neuroscience. 2003.

21. Hartcher-O’Brien J, Soto-Faraco S, Adam R. Editorial: A matter of bottom-up or top-down processes: The role of attention in multisensory integration. Frontiers in Integrative Neuroscience. 2017.

22. Zuanazzi A, Noppeney U. Distinct neural mechanisms of spatial attention and expectation guide perceptual inference in a multisensory world. J Neurosci. 2019.

23. McLennan JD. Understanding attention deficit hyperactivity disorder as a continuum. Canadian Family Physician. 2016.

24. Hudziak JJ, Achenbach TM, Althoff RR, Pine DS. A dimensional approach to development psychopathology. Int J Methods Psychiatr Res. 2007.

25. Panagiotidi M, Overton PG, Stafford T. The relationship between ADHD traits and sensory sensitivity in the general population. Compr Psychiatry. 2018;80:179–85. doi:10.1016/j.comppsych.2017.10.008.

26. Robertson AE, Simmons DR. The relationship between sensory sensitivity and autistic traits in the general population. J Autism Dev Disord. 2013.

27. Sable JJ, Kyle MR, Knopf KL, Schully LT, Brooks MM, Parry KH, et al. The Sensory Gating Inventory as a potential diagnostic tool for attention-deficit hyperactivity disorder. ADHD Atten Deficit Hyperact Disord. 2012.

28. Bijlenga D, Tjon-Ka-Jie JYM, Schuijers F, Kooij JJS. Atypical sensory profiles as core features of adult ADHD, irrespective of autistic symptoms. Eur Psychiatry. 2017.

29. Micoulaud-Franchi JA, Vaillant F, Lopez R, Peri P, Baillif A, Brandejsky L, et al. Sensory gating in adult with attention-deficit/hyperactivity disorder: Event-evoked potential and perceptual experience reports comparisons with schizophrenia. Biol Psychol. 2015.

30. Fostick L. The effect of attention-deficit/hyperactivity disorder and methylphenidate treatment on the adult auditory temporal order judgment threshold. J Speech, Lang Hear Res. 2017;60:2124–8.

31. Tucha O, Mecklinger L, Laufkötter R, Klein HE, Walitza S, Lange KW. Methylphenidate-induced improvements of various measures of attention in adults with attention deficit hyperactivity disorder. J Neural Transm. 2006;113:1575–92. doi:10.1007/s00702-005-0437-7.
32. Korostenskaja M, Kicic D, Kahkonen S. The effect of methylphenidate on auditory information processing in healthy volunteers: a combined EEG/MEG study. Psychopharmacology (Berl). 2008;197:475–86.

33. Panagiotidi M, Overton PG, Stafford T. Attention-Deficit Hyperactivity Disorder-Like Traits and Distractibility in the Visual Periphery. Perception. 2017.

34. Söderlund G, Sikström S, Smart A. Listen to the noise: Noise is beneficial for cognitive performance in ADHD. J Child Psychol Psychiatry Allied Discip. 2007.

35. Overton PG. Collicular dysfunction in attention deficit hyperactivity disorder. Med Hypotheses. 2008;70:1121–7. doi:10.1016/j.mehy.2007.11.016.

36. Stevens AA, Maron L, Nigg JT, Cheung D, Ester EF, Awh E. Increased sensitivity to perceptual interference in adults with attention deficit hyperactivity disorder. J Int Neuropsychol Soc. 2012;18:511–20. doi:10.1017/S1355617712000033.

37. Jung H, Woo YJ, Kang JW, Choi YW, Kim KM. Visual perception of ADHD children with sensory processing disorder. Psychiatry Investig. 2014;11:119–23. doi:10.4306/pi.2014.11.2.119.

38. Grönlund MA, Aring E, Landgren M, Hellström A. Visual function and ocular features in children and adolescents with attention deficit hyperactivity disorder, with and without treatment with stimulants. Eye. 2007.

39. Tannock R, Banaschewski T, Gold D. Color naming deficits and attention-deficit/hyperactivity disorder: A retinal dopaminergic hypothesis. Behav Brain Funct. 2006.

40. Kim S, Chen S, Tannock R. Visual function and color vision in adults with Attention-Deficit/Hyperactivity Disorder. J Optom. 2014.

41. Michalek AMP, Watson SM, Ash I, Ringleb S, Raymer A. Effects of noise and audiovisual cues on speech processing in adults with and without ADHD. Int J Audiol. 2014.

42. McCracken HS, Murphy BA, Glazebrook CM, Burkitt JJ, Karellas AM, Yelder PC. Audiovisual multisensory integration and evoked potentials in young adults with and without Attention-Deficit/Hyperactivity Disorder. Front Hum Neurosci. 2019;13.

43. Jansen I, Philipsen A, Dalin D, Wiesmeier IK, Maurer C. Postural instability in adult ADHD – A pilot study. Gait Posture. 2019.

44. Hove MJ, Zeffiro TA, Biederman J, Li Z, Schmahmann J, Valera EM. Postural sway and regional cerebellar volume in adults with attention-deficit/hyperactivity disorder. Neurolmage Clin. 2015.

45. Luck SJ. Ten simple rules for designing and interpreting ERP experiments. In: Event-Related Potentials: A Methods Handbook. 2005.

46. Polich J. Updating P300: An integrative theory of P3a and P3b. Clinical Neurophysiology. 2007.

47. Light GA, Swerdlow NR, Braff DL. Preattentive sensory processing as indexed by the MMN and P3a brain responses is associated with cognitive and psychosocial functioning in healthy adults. J Cogn Neurosci. 2007.
48. Barry RJ, Clarke AR, McCarthy R, Selikowitz M, Brown CR, Heaven PCL. Event-related potentials in adults with Attention-Deficit/Hyperactivity Disorder: an investigation using an inter-modal auditory/visual oddball task. Int J Psychophysi ol. 2009;71:124–31. doi:10.1016/j.ijpsycho.2008.09.009.

49. NÄÄTÄNEN R, PAAVILAINEN P, TITINEN H, JIANG D, ALHO K. Attention and mismatch negativity. Psychophysiology. 1993.

50. Faraone S V., Biederman J, Spencer T, Wilens T, Seidman LJ, Mick E, et al. Attention-deficit/hyperactivity disorder in adults: An overview. Biological Psychiatry. 2000.

51. Micoulaud-Franchi J-A, Lopez R, Cermolacce M, Vaillant F, Péri P, Boyer L, et al. Sensory Gating Capacity and Attentional Function in Adults With ADHD: A Preliminary Neurophysiological and Neuropsychological Study. J Atten Disord. 2019;23:1199–209. doi:10.1177/1087054716629716.

52. Holstein DH, Vollenweider FX, Geyer MA, Csomor PA, Belser N, Eich D. Sensory and sensorimotor gating in adult attention-deficit/hyperactivity disorder (ADHD). Psychiatry Res. 2013.

53. Smucny J, Olincy A, Eichman LC, Lyons E, Tregellas JR. Early sensory processing deficits predict sensitivity to distraction in schizophrenia. Schizophr Res. 2013;147:196–200. doi:10.1016/j.schres.2013.03.025.

54. Israel JB, Chesney GL, Wickens CD, Donchin E. P300 and Tracking Difficulty: Evidence For Multiple Resources in Dual-Task Performance. Psychophysiology. 1980.

55. Halperin JM, Schulz KP. Revisiting the role of the prefrontal cortex in the pathophysiology of attention-deficit/hyperactivity disorder. Psychol Bull. 2006.

56. Gonen-Yaacovi G, Arazi A, Shahar N, Karmon A, Haar S, Meiran N, et al. Increased ongoing neural variability in ADHD. Cortex. 2016;81:50–63. doi:10.1016/j.cortex.2016.04.010.

57. Raichle ME, Snyder AZ. A default mode of brain function: A brief history of an evolving idea. NeuroImage. 2007.

58. Cross-Villasana F, Finke K, Hennig-Fast K, Kilian B, Wiegand I, Muller HJ, et al. The Speed of Visual Attention and Motor-Response Decisions in Adult Attention-Deficit/Hyperactivity Disorder. Biol Psychiatry. 2015;78:107–15. doi:10.1016/j.biopsych.2015.01.016.

59. Couperus JW, Alperin BR, Furlong D, Mott K. Visual Selective Attention in Adults with ADHD: Electrophysiological Evidence of Facilitation and Suppression. J Behav Brain Sci. 2014.

60. Hasler R, Perroud N, Meziane HB, Herrmann F, Prada P, Giannakopoulos P, et al. Attention-related EEG markers in adult ADHD. Neuropsychologia. 2016;87:120–33. doi:10.1016/j.neuropsychologia.2016.05.008.

61. Helenius P, Laasonen M, Hokkanen L, Paetau R, Niemivirta M. Impaired engagement of the ventral attentional pathway in ADHD. Neuropsychologia. 2011.

62. Prox V, Dietrich DE, Zhang Y, Emrich HM, Ohlmeier MD. Attentional processing in adults with ADHD as reflected by event-related potentials. Neurosci Lett. 2007.
63. Dockstader C, Gaetz W, Cheyne D, Wang F, Castellanos FX, Tannock R. MEG event-related desynchronization and synchronization deficits during basic somatosensory processing in individuals with ADHD. Behav Brain Funct. 2008;4:8. doi:10.1186/1744-9081-4-8.

64. Dockstader C, Gaetz W, Cheyne D, Tannock R. Abnormal neural reactivity to unpredictable sensory events in attention-deficit/hyperactivity disorder. Biol Psychiatry. 2009;66:376–83. doi:10.1016/j.biopsych.2009.04.010.

65. Biswal B, Zerrin Yetkin F, Haughton VM, Hyde JS. Functional connectivity in the motor cortex of resting human brain using echo-planar mri. Magn Reson Med. 1995.

66. Carmona S, Hoekzema E, Castellanos FX, García-García D, Lage-Castellanos A, Van Dijk KRA, et al. Sensation-to-cognition cortical streams in attention-deficit/hyperactivity disorder. Hum Brain Mapp. 2015;36:2544–57. doi:10.1002/hbm.22790.

67. Chen C, Xiu D, Chen C, Moyzis R, Xia M, He Y, et al. Regional homogeneity of resting-state brain activity suppresses the effect of dopamine-related genes on sensory processing sensitivity. PLoS One. 2015.

68. Uddin LQ, Kelly AMC, Biswal BB, Margulies DS, Shehzad Z, Shaw D, et al. Network homogeneity reveals decreased integrity of default-mode network in ADHD. J Neurosci Methods. 2008.

69. Salmi J, Salmela V, Salo E, Mikkola K, Leppämäki S, Tani P, et al. Out of focus - Brain attention control deficits in adult ADHD. Brain Res. 2018;1692:12–22. doi:10.1016/j.brainres.2018.04.019.

70. Tian L, Jiang T, Liang M, Zang Y, He Y, Sui M, et al. Enhanced resting-state brain activities in ADHD patients: a fMRI study. Brain Dev. 2008;30:342–8. doi:10.1016/j.braindev.2007.10.005.

71. Johnson JA, Zatorre RJ. Neural substrates for dividing and focusing attention between simultaneous auditory and visual events. Neuroimage. 2006.

72. Ahrendts J, Rüsch N, Wilke M, Philipsen A, Eickhoff SB, Glauche V, et al. Visual cortex abnormalities in adults with ADHD: A structural MRI study. World J Biol Psychiatry. 2011.

73. Berger A, Posner MI. Pathologies of brain attentional networks. Neurosci Biobehav Rev. 2000;24:3–5. doi:10.1016/s0149-7634(99)00046-9.

74. Corbetta M, Shulman GL. Control of goal-directed and stimulus-driven attention in the brain. Nat Rev Neurosci. 2002.

75. Ling S, Carrasco M. When sustained attention impairs perception. Nat Neurosci. 2006.

76. Salmi J, Rinne T, Koistinen S, Salonen O, Alho K. Brain networks of bottom-up triggered and top-down controlled shifting of auditory attention. Brain Res. 2009.

77. Posner MI. Orienting of attention. Q J Exp Psychol. 1980.

78. Luck SJ, Woodman GF, Vogel EK. Event-related potential studies of attention. Trends in Cognitive Sciences. 2000.

79. Lavie N, Tsal Y. Perceptual load as a major determinant of the locus of selection in visual attention. Percept Psychophys. 1994.
80. Lavie N. Perceptual Load as a Necessary Condition for Selective Attention. J Exp Psychol Hum Percept Perform. 1995.

81. Friedman-Hill SR, Wagman MR, Gex SE, Pine DS, Leibenluft E, Ungerleider LG. What does distractibility in ADHD reveal about mechanisms for top-down attentional control? Cognition. 2010;115:93–103. doi:10.1016/j.cognition.2009.11.013.

82. Marco EJ, Hinkley LBN, Hill SS, Nagarajan SS. Sensory processing in autism: A review of neurophysiologic findings. Pediatr Res. 2011.

83. Liss M, Mailloux J, Erchull MJ. The relationships between sensory processing sensitivity, alexithymia, autism, depression, and anxiety. Pers Individ Dif. 2008.

84. Yeap S, Kelly SP, Reilly RB, Thakore JH, Foxe JJ. Visual sensory processing deficits in patients with bipolar disorder revealed through high-density electrical mapping. J Psychiatry Neurosci. 2009.

85. Desseilles M, Balteau E, Sterpenich V, Thien TDV, Darsaud A, Vandewalle G, et al. Abnormal neural filtering of irrelevant visual information in depression. J Neurosci. 2009.

86. Philipsen A. Differential diagnosis and comorbidity of attention-deficit/hyperactivity disorder (ADHD) and borderline personality disorder (BPD) in adults. Eur Arch Psychiatry Clin Neurosci. 2006.

87. Banaschewski T, Mallis C, Oosterlaan J, Roeyes H, Rubia K, Willcutt E, et al. Towards an understanding of unique and shared pathways in the psychopathophysiology of ADHD. Dev Sci. 2005.

88. Micoulaud-Franchi JA, Lopez R, Vaillant F, Richieri R, El-Kaim A, Bioulac S, et al. Perceptual abnormalities related to sensory gating deficit are core symptoms in adults with ADHD. Psychiatry Res. 2015.

89. Koziol LF, Budding D. ADHD and sensory processing disorders: placing the diagnostic issues in context. Appl Neuropsychol Child. 2012;1:137–44. doi:10.1080/21622965.2012.709422.

**Supplementary Files**

This is a list of supplementary files associated with this preprint. Click to download.

- [PRISMA2009checklist.doc](#)