

[https://doi.org/10.46344/JBINO.2025.v14i03\(a\).09](https://doi.org/10.46344/JBINO.2025.v14i03(a).09)

COGNITIVE LIMITATION DUE TO SIMULTANAGNOSIA SECONDARY TO CHILDHOOD ADVERSITY: A SYSTEMATIC REVIEW

Furlanetto Jr MI¹ & Oliveira MTM²

¹Mario Luiz Furlanetto Junior, Brazilian physician, CRM-SP:134.606, graduated from the University of Marília, has a medical residency in general surgery from the Faculty of Medicine of Marília, residency in vascular and endovascular surgery from Santa Casa de Limeira, specialist in psychiatry, neuropsychology, neurosciences, Occupational Medicine and Hyperbaric Medicine, volunteer researcher of neurosciences applied to Medicine, creator of the Startup Stakehold ZxSy Brasil, and of the methodology of neuropsychological and clinical diagnostic systems.

²Marcelo Tadeu Marques de Oliveira, Graduated in Physical Education from the University of Marília (2003), Specialization in Physical Training and Rehabilitation from Unisaesiano de Lins (2005), Master in Sciences from the University of Campinas - UNICAMP, at the Faculty of Medical Sciences in the Child and Adolescent Health program, research line: Growth and endocrinology of children and adolescents (2018 - 2021).

ABSTRACT

Introduction: Although research on simultanagnosia has focused primarily on its neurological and cognitive aspects, there is an important gap in understanding the psychosocial factors that may influence its development or severity. Exploring this relationship is crucial, since hostile family behaviors may act as primary factors generating this condition through neurobiological mechanisms, and such a problem has never been observed in relation to emotional intelligence, mentalism and cognitive limitation in childhood. **Objective:** The objective of this systematic review is to investigate whether there is evidence to support the association between hostile family behaviors and the development of simultanagnosia as a neuromaladaptive process that limits human intelligence, seeking to understand the possible psychosocial and neurobiological mechanisms underlying its various concepts. **Methods:** This systematic literature review was based on: 1) establishing a guiding question; 2) varied sources for locating studies; 3) establishing inclusion and exclusion criteria; 4) assessment of the methodological quality of the selected studies and 5) descriptive synthesis. **Results:** We consider that simultanagnosia may be the result of damage to an attention system based on objects and values, examining the evidence that patients with simultanagnosia neglect entire objects or perceive objects in a partial way, and thus a maladaptive learning process. Several studies show that patients with simultanagnosia have lesions in the spatial attention system, which can be interpreted as a restricted spatial window of attention. **Discussion:** Empirical observations and the diversity of accumulated data on specific harmful effects according to childhood adversity, the deficit of perception or emotional attention in the first period, together with stressful events present a conditioning effect on the phases of attention and brain processing, which influences the judgment and final perception. **Conclusion:** This systematic review is limited by the impossibility of performing a meta-analysis due to the small number of samples in the included studies. However, it was clear that simultanagnosia is a set of attention and brain processing dysfunctions that influence judgment and final perception, and consequently we deduce the cognitive limitation of simultaneity or phronetic knowledge.

Keywords: Simultanagnosia, cognitive limitation, fluid intelligence, attention

Introduction

Simultanagnosia is a rare neuropsychological condition characterized by the inability to perceive multiple visual elements simultaneously, despite intact vision. It is characterized by a specific failure to recognize a global visual Gestalt, such as a visual scene or complex objects consisting of local elements.¹

In clinical practice, it is characterized by the inability to perceive more than one object at a time, with an inability to conceptualize the entire picture, despite being able to see individual elements within a visual scene, and is divided into dorsal and ventral subtypes.²

Symptoms due to dysfunction of the dorsal or ventral stream often go unrecognized. They can result from any neuropathology affecting the posterior parietal lobes or inferior temporal lobes.²

Dorsal simultanagnosia (DS) is characterized by difficulty perceiving the spatial arrangement of multiple objects: patients with DS can recognize individual objects but have difficulty perceiving how the objects are related.³

Patients with ventral simultanagnosia (VS) can perceive the spatial arrangement of objects but have difficulty recognizing them in unique ways and slowly or piecemeal identification of complex scenes, as in letter-by-letter reading.³

Associative visual agnosia (AVA) is a well-known disorder resulting from damage to the ventral stream and is characterized by the inability to recognize objects yet be able to accurately replicate the objects they

have observed.⁴

The VS and AVA are disorders caused by damage to similar cortical areas: the visual association cortex in the occipital lobe or the occipitotemporal junction within the dominant hemisphere. Specifically, VS is associated with the inferior occipitotemporal region of the dominant hemisphere.⁴

This condition is often associated with bilateral lesions in the superior parietal areas, as observed in Balint syndrome (DS).⁵

In addition to Balint Syndrome, it is described in Anton Babinsky Syndrome and Parietal Hemineglect Syndrome, expressing an absence of perception and identification of situations and objects at a given moment, without presenting visual dysfunctions, characterizing a mechanism of "cortical blindness" and an explicit problem.⁶ Still in some variants of Alzheimer's disease, posterior cortical atrophy is dominated by the progressive degradation of the parieto-occipital gray and white matter and can lead to simultanagnosia with mechanisms of decreased short-term visual memory capacity and visual processing speed.⁷

Although research on simultanagnosia has focused mainly on its neurological and cognitive aspects, there is an important gap in the understanding of the psychosocial factors that can influence its development or severity.⁸

Hostile family factors, such as dysfunctional dynamics, neglect, abuse Emotional disorders, and especially adverse childhood emotions, have been widely studied in relation to other neuropsychiatric conditions.⁹

However, little is known about the impact of these adverse experiences on the manifestation of conditions such as simultanagnosia, albeit in a more subtle form, with mechanisms of dysfunctional perceptions of an emotional nature, and associated with the interruption of the neurodevelopment of fluid intelligence.¹⁰

Exploring this relationship is crucial since hostile family behaviors can act as primary factors generating this condition through neurobiological mechanisms, and such a problem has never been observed in relation to emotional intelligence, mentalism, and cognitive limitation in childhood.¹¹

Understanding

the interactions between preserved object perception, impaired object attention, and disrupted spatial attention will likely provide even more sophisticated insights into the behavioral puzzle that is simultanagnosia.¹⁰ However, grouping mechanisms do not only require spatial integration of visual information but also involve integration processes over time.⁹⁻¹⁰

Studies have shown largely preserved perception of biological motion, while perception of motion-defined shapes is impaired. However, a restriction in overall visual working memory capacity over time has emerged as a general explanation for impaired global shape recognition in patients with simultanagnosia.⁸⁻¹¹

When studying the predictive validity of personality traits, it is important to understand potential overlaps with other constructs. Given a relationship between a trait and an outcome, one may be interested in whether this association can be explained by shared

variance with other constructs, such as cognitive ability.⁸⁻¹¹

Thus, the implications of individual limits of observation, evaluation, and judgment, in relation to the capacities of perception, dissociation, identification, enumeration, interpretation, and individuation of objects in a specific area of human knowledge, allow us to evaluate the reality of dense intelligence, or linear intelligence.⁸⁻¹⁴ According to Ziegler et al. (2012), character strengths can shape the (fluid) domains in which cognitive abilities are invested, but they can also promote the accumulation of knowledge and skills, eventually helping people to promote the virtue of wisdom.

8-14

Practical

wisdom is equivalent to the term *phronetics* by several authors; the *dianoetic* virtue of practical or simultaneous wisdom is the ability to perform objective synthesis and problem-solving using several concepts simultaneously.⁸⁻¹⁵

Objective

The objective of this systematic review is to investigate whether there is evidence to support the association between hostile family behaviors and the development of simultanagnosia as a neuromaladaptive process that limits human intelligence, seeking to understand the possible psychosocial and neurobiological mechanisms underlying its various concepts.

Methods

Study design

This systematic literature review was based on: 1) establishing a guiding question; 2) various sources for locating studies; 3) establishing inclusion and exclusion criteria; 4) evaluating the

methodological quality of the selected studies; and 5) descriptive synthesis.

Databases and search strategies

The studies were searched in three databases: PubMed, Scopus, and Web of Science. To establish the guiding question, "Can simultanagnosia limit human consciousness and intelligence?" the main keywords related to the investigated topics were cross-searched: "simultanagnosia and neurobiology," "Simultanagnosia," "family hostility," and "psychosocial factors".

Broader terms were intentionally used at this point to locate a larger number of articles and avoid inadvertently disregarding any important study.

There is no research on simultanagnosia and adversity, so articles on this topic were selected by convenience, in which they present associated pathophysiology.

Inclusion and exclusion criteria

Only articles with a specific focus on simultanagnosia with a neurobiological basis with fMRI exams, and that did not present an already known underlying disease, were included. No restrictions were imposed on the year of publication, only on the language, so that only articles written in Portuguese, Spanish or English would be included.

Exclusion criteria included books, book chapters, letters from the editor, and other formats that are not subjected to rigorous and peer-reviewed evaluation, as is the case with scientific articles.

Studies with only bibliographic review designs, without approaches to functional neuroimaging exams, were

also excluded, as well as articles that specifically addressed simultanagnosia linked to specific diseases and syndromes such as Balint's DS, Alzheimer's, strokes, and neurosurgeries.

Review procedures

Two researchers conducted the bibliographic search in January 2025 based on the established inclusion criteria. The first stage regarding the selection of studies included reading and analyzing the titles and abstracts of all articles initially selected. In the second stage, the full texts were read, which led to the exclusion of articles that did not meet the criteria.

In the third stage, the main information from the articles was summarized in a table to guide the descriptive and critical analysis of the articles.

Results

The bibliographic search resulted in 529 articles, 369 of which appeared more than once. A total of 449 articles were excluded after applying the inclusion and exclusion criteria, and another 62 articles were excluded after reading the full texts.

The 21 articles that remained comprised the corpus of the review. Figure 1 presents the PRISMA flowchart with the identification, selection, and steps for inclusion of the texts.

Table 1 presents a summary of the main characteristics of the studies. Regarding the methodological characteristics of the studies (Table 1), the sample sizes ranged from 1 to 16 individuals, considering that the studies are primary, observational, and cross-sectional experiments, using third-person neuroscience through clinical data, associated with fMRI exam

evaluations, in a rare neuropsychological condition, and of scarce research.

The methodological quality assessment was carried out using the CASP-Critical Appraisal Skills Program (2018) checklist, which is summarized in three questions: Are the study results valid? (Section A) What are the results? (Section B) Will the results help locally? (Section C), in which the answers were positive. Simultanagnosia presents different concepts among researchers, but of the included studies and with a neurobiological basis, 11 articles demonstrated perception dysfunction, 1 article demonstrated working memory dysfunction, and 2 articles demonstrated individuation/subitization dysfunction.

In our review, we summarize what is known about the neural substrates of attention in patients with simultanagnosia, without association with known diseases (Baliant, Alzheimer's), and we found that the relationship of simultanagnosia is a related disorder of attention and delayed processing dysfunctions, which causes a deficit in final perception and visual neglect.

We consider that simultanagnosia may be the result of damage to an object-value-based attention system, examining the evidence that patients with simultanagnosia neglect whole objects or perceive objects in a partial way, and thus a maladaptive learning process.

Several studies show that patients with simultanagnosia have damage to the spatial attention system, which can be interpreted as a restricted

spatial window of attention. After analyzing the included articles, we created the PRISMA protocol flowchart (Figure 1) and performed a descriptive synthesis of the clinical and neurobiological data.

Table 1.Included articles and identification of the brain region, main psychological domain dysfunction of simultanagnosia, pathophysiology, and experimental sample.

Authors	Type simultanagnosia	Brain region	Dysfunction	Sample
Huberle E, Rupek P, Lappe M, Karnath HO. (2009)	Restricted visual working memory and impaired visuospatial attention	Bilateral parieto-occipital	Motion-defined shape perception was impaired	2
<u>Huberle, Elisabeth;Karnath, Hans-Otto</u> (2010)	Selective impairment in the simultaneous perception and integration of multiple objects (global perception)	Bilateral parieto-occipital	Global perception increases independently of global object size and depends on the spatial relationship between local elements and the global object.	4
Himmelbach M, Erb M, Klockgether T, Moskau S, Karnath HO (2008)	Global perception	Bilateral lateral and medial inferior parietal cortex	Circular clusters of activity in the right and left primary intermediate sulci and a bilateral cluster in the ventral precuneus.	2
Poncet M, Caramazza A, Mazza V.	Subitization	Parieto-occipital,	Subitization ability correlates with visual working memory capacity during delayed match-to-sample judgments, another task that requires processing and	6

			individuating a small number of objects simultaneously	
Morihara K, Higashiyama Y, Asano S, Matsunaga Y, Takahashi K, Miyake R, Tanaka K, Joki H, Doi H, Takeuchi H, Tanaka F.(2022)	Attentional	Right occipitotemporal	maintaining initial global information perceived for a long period of time during visual presentation	1
Balslev D, Odoj B, Rennig J, Karnath HO. (2014)	Attentional	Bilateral parieto-occipitals	Abnormal allocation of attention around gaze may disrupt the grouping of individual objects into an integrated visual scene.	2
Chechlac M, Rotshtein P, Hansen PC, Riddoch JM, Deb S, Humphreys GW. (2012)	Visual perception Attentional	Bilateral gray matter lesions within the middle frontal area (BA 46), cuneus, calacarina, and parieto-occipital fissures, as well as right hemisphere parietal lesions within the intraparietal and postcentral gyri	Bilateral parieto-occipital white matter disconnections Bilateral damage to major pathways within the visuospatial attention network, including the superior longitudinal fasciculus, the inferior fronto-occipital fasciculus, and the inferior longitudinal fasciculus.	6

Khan AZ, Prost-Lefebvre M, Salemme R, Blohm G, Rossetti Y, Tilikete C, Pisella L. (2015)	Attention	Bilateral lesions of the superior parietal lobule	Spatial integration of separable features (<u>within-object</u> processing), narrowing the attentional field in which a target can be detected. Posterior parietal cortex is crucial for individualizing stimuli by computing their unique spatiotemporal features. Abstraction deficits.	3
Coslett HB, Lie E. (2007)	Perception	Bilateral parietal lesions prevent the binding of preserved object representations to a representation computed by the dorsal visual system.	Posterior parietal cortex is crucial for individualizing stimuli by computing their unique spatiotemporal characteristics.	1
Kranjec A, Ianni G, Chatterjee A. (2013)	Perception	Bilateral occipito-parietal	Abstraction deficit	1
Leek EC, d'Avossa G, Tainturier MJ, Roberts DJ, Yuen SL, Hu M, Rafal R. (2012)	Perception	Posterior occipitotemporal lesions	High-level perception that supports the integration of stored knowledge and visual sensory input.	1
James TW, Culham J,	Visual perception	Visual	Visual form	1

Humphrey GK, Milner AD, Goodale MA.(2003)		occipitotemporal	agnosia is associated with extensive damage to the ventral stream	
Veronelli L, Daini R, Mannino A, Rossetti A, Gilardone G, Corbo M, Primativo S.	Attention	Posterior cortical atrophy	Global processing. Visual-perceptual and/or focal attention mechanisms.	16
Thomas C, Kveraga K, Huberle E, Karnath HO, Bar M. (2012)	Visuospatial attention and perception	Posterior parietal cortex	Impairment in global form perception persists even though the ventral visual pathway, the primary recognition pathway, is intact	1
Suzuki K.	Visual attention is not simply top-down or bottom-up, but is implicitly affected by visual recognition	Callosal disconnection	Impaired ability of participants to visually synthesize and efficiently reproduce Complex Figure stimuli	1
Vialatte A, Yeshurun Y, Khan AZ, Rosenholtz R, Pisella L(2021)	Attentional	Superior parietal lobe	Slower performance than controls in visual search.	8
Duncan J, Bundesen C, Olson A, Humphreys G, Ward R, Kyllingsbæk S, van Raamsdonk M, Rorden C, Chavda S. (2003)	Attentional	Dorsal and ventral	Impairment in global form perception persists even if the ventral visual pathway, the primary recognition pathway, is intact. Extent of	2

			visual attention was dynamically altered depending on the level of visual processing.	
Berry BA, Meyer GJ, O'Gorman ET, Roy M, Sholander LE, Mihura JL. (2024)	Attentional	Predominance of left hemisphere activity	Primary deficit in processing more than one display element, attentional gluing, foveal bias, and global weakness of visual representation.	2
Rennig J, Himmelbach M, Huberle E, Karnath HO. (2015)	Perception	Temporoparietalis direitas	Improvement with training	4
Nestmann S, Wiesen D, Karnath HO, Rennig J.	Perception	Temporoparietalis	Significantly higher BOLD signals during processing of non-canonical objects compared to canonical objects	4
Rennig J, Langenberger C, Karnath HO. (2024)	Global visual perception	Temporoparietal junction	Deficits during recognition of hierarchical stimuli and real-world visual scenes	18

Simultanagnosia and Attention

Visual attention is involved in creating a coherent visual representation of our world, and attributing the fragmented perception of simultanagnosia to a deficient attentional system may be common but underappreciated.¹⁵⁻²⁰

In everyday life, the visual system is constantly stimulated with

information about different objects, and the attentional network captures what it needs to select a subset of this information for further processing.¹⁵⁻²⁰

Attention is the mechanism that allows this selection to occur, but there are competing theories about what guides this selection. One type of attentional selection is object-based: attention is

directed to objects that are defined based on pre-attentional segmentation according to basic grouping principles.²¹⁻²⁷

A second type of attentional selection is spatially based: attention is directed to locations in space independently of objects.²¹⁻²⁷

A third type of attentional dysfunction is conditioning of stimulus-reward associations, which may currently be considered irrelevant, is a phenomenon called "value-driven attentional capture" (VDAC).²¹⁻²⁷

The meaning of the object can influence inattention to objects in simultanagnosia.²¹⁻²⁷

VDACs are representations of stimuli associated with reward that undergo plasticity in the sensory cortex, automatically capturing attention during the initial processing of any lived experience.²¹⁻²⁷

Behavior directed toward goals or associated results presents motivational elements based on cognitive values, which influence decision-making and aid in interpretation in order to distinguish motivational control from value-based processes.²⁸⁻³³ Studies of selective attention typically consider the role of task goals or physical salience, but attention can also be captured by stimuli previously associated with reward or emotional defense.²⁸⁻³³

Many studies show that reward learning induces visual cortical plasticity, which modulates early visual processing to capture attention; learned value modulates spatial signals in visual cortical areas, an effect that correlates with VDAC.²⁸⁻³³

Attentional capture by previously rewarded targets and the modulatory effect of reward on priming, as well as the decoupling of reward history and prior task relevance in value-driven attention.²⁸⁻³³

Studies using magnetic encephalography have investigated modulations by reward learning and have shown that VDAC is supported by learned value signals that modulate spatial selection throughout the visual and posterior parietal cortex, which can still occur in the absence of changes in visual processing in the cortex.³⁴⁻⁴⁰

This value modulation is influenced by the strength of the behavioral VDAC effect and persists in subsequent target processing.³⁴⁻⁴⁰

Recent studies have demonstrated that VDAC is based on Pavlovian conditioning, and behavioral evidence distinguishes VDAC from other established control mechanisms, suggesting a distinct underlying neurobiological process.³⁴⁻⁴⁰ VDAC persists perennially even without reinforcement, unlike other forms of learning, where removal of reinforcement typically leads to extinction.³⁴⁻⁴⁰

Evidence suggests that the time interval between resource and reward is flexible, with some constraint on learning the resource-reward association.³⁴⁻⁴⁰

When these value-signaling stimuli appeared as distractors in the test phase, they continually shaped attentional selection despite their task irrelevance.³⁴⁻⁴⁰

Rule-guided allocation of attention to different stimulus dimensions produced discriminate patterns of activation in the visual cortex, providing a signature of top-down bias in perception.⁴¹⁻⁴⁷

Simultanagnosia is often conceptualized as an object-based disorder, in which attention involved in selecting objects or their features is impaired, and thus there is no effective object processing and perception.⁴¹⁻⁴⁷

An alternative account is that simultanagnosia is a spatially based

attention disorder: if attention can only process information within a restricted location, it may be difficult to select more than one object at a time. ⁴¹⁻⁴⁷

The limitation of recognition to single objects in simultanagnosia may suggest reduced ability in object identification, but other alternative concepts suggest preserved object identification, in which patients can recognize single objects, coupled with impaired spatial attention, which restricts the number of objects that can be perceived. ⁴¹⁻⁴⁷ In addition to inattention to whole objects, there is evidence that patients with simultanagnosia perceive individual objects in a fragmented manner, indicating another manifestation of damage to an object-based attention system. ⁴¹⁻⁴⁷

According to Karnath et al. (2000), "local capture" demonstrates this mechanism, as patients identify the local components of an object but fail to see the global aspect of the object, even with unlimited presentation. ⁴⁸⁻⁵²

An fMRI study showed that a patient with simultanagnosia could not make judgments about the triangular spatial relationship between three separate disks but could do so if the solitary triangle was made explicit by adding lines connecting the disks or surface texture to the triangle. ⁴⁸⁻⁵²

Thus, reduced object-based attention results in failure to perceive such objects when the link between the features is deficient. ⁴⁸⁻⁵²

Neural substrates of attention

Following primary processing in the occipital lobes, visual information is transmitted to the posterior parietal lobes (PPLs) via the dorsal stream and to the inferior temporal lobes via the ventral stream. ⁴⁸⁻⁵²

The PPLs unconsciously create a three-dimensional mental map of the environment that guides precise body movements and also facilitates the

focusing of visual attention on the object of interest. ⁵³⁻⁵⁹

The inferior temporal lobes facilitate the recognition of objects, shapes, people, and routes. ⁵³⁻⁵⁹

The PPLs analyze complex visual information and facilitate focusing on objects of interest. Lesions to PPL neurons result in slow, sequential processing of incoming sensory data rather than the usual rapid, parallel processing. ⁵³⁻⁵⁹

In healthy individuals, "change blindness" (the inability to detect even large changes in an object or scene) and "attentional blink" (the failure to consciously perceive a stimulus that quickly follows another stimulus) are mechanisms by which the absence of focused attention can result in a lack of awareness of features of the visual world that are in plain sight, i.e., "looking" but not "seeing." ⁵³⁻⁵⁹

The parallels between neglect and simultanagnosia are striking, leading some to describe simultanagnosia as "bilateral neglect," although distinctions in lesion locations are not. ⁵³⁻⁵⁹

Neglect results from unilateral temporoparietal lesions and is defined by a failure to attend to information on the contralesional (usually left) side of space. ⁵³⁻⁵⁹

Of particular relevance to the present discussion, neglect has been conceptualized in terms of both spatial and object-specific deficits. ⁵³⁻⁵⁹

In the context of neglect, spatial deficits refer to inattention to observer-centered (egocentric) spatial locations, whereas object-specific deficits refer to a failure to attend to object-centered (allocentric) locations (the left side of the object, regardless of where the object is located relative to the observer). ⁶⁰⁻⁶⁴

Spatial attention deficits in neglect have been associated with the

dorsal stream network, particularly the right supramarginal gyrus, whereas object-centered deficits have been associated with more ventral regions, including the posterior inferior temporal, lateral occipital, and posterior middle/inferior temporal regions, suggesting a link between these types of attention and these neuroanatomical regions.⁶⁰⁻⁶⁴

The paucity of neuromorphological studies of lesions across patients makes it difficult to determine the precise neuroanatomical regions that are affected in the disorder or their relationship to object- versus spatial-based deficits.⁶⁰⁻⁶⁴

An fMRI study of patients with simultanagnosia demonstrated that bilateral damage to the medial occipitoparietal junction, cuneus, and inferior intraparietal sulcus, as well as underlying white matter tracts, is critical for producing simultanagnosia.⁶⁰⁻⁶⁴

These studies attributed white matter damage to deficits in processing speed, whereas damage to the occipitoparietal junction was attributed to impaired ability to represent multiple items together and attend to the global aspects of compound shapes.⁶⁰⁻⁶⁴

Himmelbach et al. used fMRI evidence to show that these areas were active when the patient successfully identified the global level of hierarchical stimuli.⁶⁰⁻⁶⁴ Riddoch et al. (2010) provided evidence that these areas are involved in controlling the spatial window or scale of visual attention.⁶⁰⁻⁶⁴

Michel and Henaff (2004) attributed the deficits of their simultanagnosia patients, which resulted from bilateral and relatively symmetrical lesions between the superior calcarine fissure and the occipitoparietal sulcus, extending into the white matter in the frontoparietal areas, to deficits and restrictions of the attentional visual field.⁶⁰⁻⁶⁴ Thus, we consider how certain

behaviors in simultanagnosia are in the nature of the attentional impairments that define the disorder.⁶⁰⁻⁶⁴

Evidence for object-based deficits

Object-based theories suggest that attention is directed to candidate objects that are then selected for further processing.⁶⁵⁻⁶⁹

The limitation in attentional capacity is seen in terms of the number of separate objects that can be perceived at once, with patients ignoring some objects in their entirety and seeing other objects in parts.⁶⁵⁻⁶⁹

Simultanagnosia, the inability to see more than one object at a time, is therefore understood as an abnormal limitation of this attentional capacity: "It is whole objects that are neglected, not spatially determined parts of objects; and the objects that are neglected may occupy the same spatial coordinates as an object that is seen."⁶⁵⁻⁶⁹

Better recognition was also found for pairs of line drawings that were semantically related (both animals) than those that were unrelated (animal and tool).⁶⁵⁻⁶⁹ In both cases, the spatial locations of the stimuli remain constant, thus pointing to object-based effects that may modulate the attentional deficit of simultanagnosia, although it does not conclude that the deficit itself is object-based.⁶⁵⁻⁶⁹

Part-based object perception (individuation)

In the inability to identify multiple objects, patients with simultanagnosia can enumerate up to 2-3 elements as efficiently as healthy individuals (the so-called "subitization" phenomenon).⁷⁰⁻⁷⁴

This intriguing observation is one of the first results to support the existence of an "object individuation" mechanism that can spatially mark a limited set of objects simultaneously and resonates with recent research on the brain dynamics of enumeration in healthy

individuals.⁷⁰⁻⁷⁴

The latter distinction has been the subject of recent neuroimaging research that has provided refined insights into the spatial and temporal aspects of object individuation and recognition.⁷⁰⁻⁷⁴

The lessons learned from neuropsychological research on exact enumeration in simultanagnosia can be generalized to the normal functioning of the human mind and have provided insightful clues for cognitive neuroscience.⁷⁰⁻⁷⁴

Evidence for spatially-based deficits

According to Posner et al. (1980), spatially-based attention is the direction of attention to specific locations and objects, and the parietal lobes are central to visual attention and spatial processing.⁷⁵⁻⁸⁰

According to a systematic review, the ventral stream, which extends from early visual areas through the inferotemporal cortex, is responsible for processing information about objects, whereas the dorsal stream, which extends from early visual areas through the parietal cortex, is responsible for processing information about space.⁷⁵⁻⁸⁰

Goodale and Miller proposed an integrated account of object and spatial processing, suggesting that both visual streams were implicated in the processing of object and spatial information and that both streams are influenced by attention.⁷⁵⁻⁸⁰ Chechlacz et al. (2012a) showed that simultanagnosia results from bilateral parieto-occipital white matter disconnections within the visuospatial attention network, explicitly linking simultanagnosia with current understandings of the anatomical locus of spatial-based attention.⁷⁵⁻⁸⁰

Studies have highlighted other behavioral dysfunctions that are associated with simultanagnosia, with impaired spatial processing.⁷⁵⁻⁸⁰

Spatial-based behavioral deficits

Although features can be processed preattentively, focused attention is required to combine features in the perception of an object. Patients with simultanagnosia experience a large number of illusory conjunctions, incorrectly linking features of different objects.⁷⁵⁻⁸⁰

The increased number of illusory conjunctions in simultanagnosia is evidence of impaired spatial processing or limited spatial attention, coupled with incorrect localization of object features.⁷⁵⁻⁸⁰

More direct evidence of spatial processing deficits is evident in other tasks, such as abstraction and synthesis.⁷⁵⁻⁸⁰

Despite these explicit spatial deficits, simultanagnosia patients can still correctly process spatial information implicitly.⁷⁵⁻⁸⁰

In 1987, Rizzo and Hurtig proposed the existence of an attentional dysfunction in simultanagnosia that allows spatial information to be partially processed, enough to guide eye movements but not enough for conscious perception.⁷⁵⁻⁸⁰

Object-based modulation of spatial-based deficits

Object properties can influence the competition between local and global perception in a situation where spatial attention capacity is restricted. One such factor is familiarity with the elements.⁸¹⁻⁸⁶

Shalev et al. describe attention between object components: whichever component attracts attention first will draw attention to that area or spatial scale.⁸¹⁻⁸⁶ This suggests that the allocation of attention to space is critically dependent on the identity of the object itself and its components, and thus disturbances in attentional capture for emotional or physical/financial survival value are a hypothesis worth considering.

81-86

Although healthy individuals are still able to report local and global components, with a latency advantage, the limited attentional capacity of patients with simultanagnosia means that only one component is functional.⁸¹⁻⁸⁶

Healthy individuals are better at making two judgments about a single object compared to a single judgment about two objects.⁸¹⁻⁸⁶

One might predict that this effect would be enhanced in simultanagnosia patients because they see only one object at a time and would be impaired at making judgments/synthesis/conclusions about multiple aspects of an object, in addition to suffering from anticipation in processing phases, or processing delays.⁸¹⁻⁸⁶

Shalev and Humphreys (2002) explained these findings in terms of a restricted spatial zoom lens of attention, showing that patients can move the narrow lens from object to object to make two-object judgments but struggle to expand this zoom lens to go from object parts to whole objects to make within-object judgments. Stored representations may facilitate the expansion of the zoom lens, explaining why patients show an advantage for within-object judgments with familiar objects and between-object judgments with unfamiliar objects.⁸¹⁻⁸⁶

Preserved object-based attention may also bias spatial-based attention in patients with simultanagnosia.⁸¹⁻⁸⁶

Humphreys and Riddoch suggested that in lesions of the spatial selection system, as in simultanagnosia, preserved object processing exerts more influence on selection, controlling the spatial attention system more weakly.⁸¹⁻⁸⁶

Thus, in the reverse direction, impaired spatial attention may disrupt the perception of objects, and an early

judgment may occur as in negligent judgment.⁸¹⁻⁸⁶

Simultanagnosia as a Reduced “Spatial Window of Attention”

One approach that is particularly relevant to discussions of simultanagnosia is the idea of attention as a “spotlight.” This proposes that, like a spotlight, attention can be moved and directed to various locations in space.⁸⁶⁻⁹⁰

The spotlight is often described as flexible: it can zoom out to cover a large spatial area or zoom in to cover a smaller spatial area.⁸⁶⁻⁹⁰

Furthermore, just as a spotlight becomes brighter as the beam is narrowed, attentional acuity can increase as the spatial extent of an attended area is reduced in size.⁸⁶⁻⁹⁰

The concept of simultanagnosia as reflecting a restricted window (or “spotlight”) of attention is apparent from descriptions used in the literature.⁸⁶⁻⁹⁰

Michel and Henaff (2004) explored the simultanagnosia deficit in terms of a shrinking visual attentional field.⁸⁶⁻⁹⁰

Shalev et al. (2004) reported priming results consistent with a flexible reduction of a spatial attentional window in simultanagnosia.⁸⁶⁻⁹⁰

The ability of patients with simultanagnosia to perceive global levels of facial stimuli may require an expanded window of attention in the presence of stimuli with highly salient global properties in which there is strong binding between component features.⁸⁶⁻⁹⁰

Interestingly, one interpretation of researchers who have observed the consequent failure to see local elements when global form is perceived is that the expansion of the window occurs at the expense of attentional acuity: they can see a face but cannot resolve its details.⁸⁶⁻⁹⁰

There is data showing that information can still be processed to a degree outside the restricted window of attention that supports conscious perception of stimuli.⁸⁶⁻⁹⁰ Thus, a small processing window, restricted in visual or attentional capacity, may impair the relative localization and spatial integration of local elements necessary to infer global form.⁸⁶⁻⁹⁰

Some simultanagnostic effects cannot be easily explained by a reduced spatial window of attention or at least suggest additional modifications of the latter. The illusory conjunctions mentioned earlier are an example.⁸⁶⁻⁹⁰

A reduced window might be expected to allow processing of a single object falling within it and promote correct binding of object features, but patients frequently show binding errors.⁸⁶⁻⁹⁰

Such illusory conjunctions can also occur in healthy individuals with brief viewing under conditions of divided attention.⁸⁶⁻⁹⁰

However, it may also be that processing stimuli at two locations requires some expansion of the narrowed attentional window, which has a consequent reduction in the spatial resolution of attention in simultanagnostic patients, allowing binding errors to occur.⁸⁶⁻⁹⁰

Rizzo and Hurtig speculate that this reflects cortical fatigue, which prevents sustained processing of stimuli at a conscious level, consistent with Pavlov's hypothesis that the visual deficits in simultanagnosia were related to "low arousal tone" in the visual cortex.⁸⁶⁻⁹⁰

One possibility is that with cortical fatigue, the window of attention closes completely. Another explanation is that processing of information within the window of attention may be fatigued.⁸⁶⁻⁹⁰

The spatial Stroop experiment demonstrated implicit processing of location information in simultanagnosia despite the absence of explicit knowledge of location.⁸⁶⁻⁹⁰ Thus, visual stimuli outside the boundaries of the narrow spatial window of attention are not subject to complete failure of representation, but rather to a weaker degree of representation than normal.⁸⁶⁻⁹⁰

Simultanagnosia and Neuromaladjustment

A systematic review showed that threat exposures and deprivation exposures were associated with differences in regional thickness or volume within single neural networks.⁹¹⁻⁹⁶

Patients with bilateral parieto-occipital brain damage may show intact processing of single objects, while their perception of multiple objects is concomitantly impaired.⁹¹⁻⁹⁶

The right hemisphere is involved in emotion perception, and functional impairments are due to interhemispheric transfer dysfunction (functional commissurotomy model).⁹¹⁻⁹⁶ The ventral visual pathway, which projects along the inferior temporal cortex (IT) and is primarily involved in visual object recognition, and the dorsal visual pathway, which projects along the parietal cortex and is primarily involved in visuospatial processing in the service of visually guided action.⁹¹⁻⁹⁶

The implications of individual limits of observation, evaluation and judgment, in relation to the capacities of perception, dissociation, identification, enumeration, interpretation and individuation of objects in a given specific area of human knowledge, allow us to assess only the reality of dense intelligence, or linear intelligence of knowledge.⁹¹⁻⁹⁶

The

distribution of attention in extrapersonal space is coordinated by a large-scale network built around the frontal eye fields, the intraparietal sulcus, and the cingulate gyrus.⁹¹⁻⁹⁶

The frontal eye fields and posterior parietal cortex act as a network in spatial attention tasks, the middle temporal and temporopolar cortices for face and object recognition, the amygdala and hippocampal-entorhinal complex for the emotion-memory network, Wernicke's area and Broca's area for the language network, and the prefrontal cortex and posterior parietal cortex for the executive function network of working memory.⁹¹⁻⁹⁶

The inferior longitudinal fasciculus (ILF) is the long association system of the ventral visual pathways in the occipitotemporal cortices. Visual agnosia and prosopagnosia are two clinical conditions that can arise from damage to the ILF.⁹¹⁻⁹⁶ The fronto-occipital fasciculus (FOF) connects the parieto-occipital region with the premotor and dorsal prefrontal cortices and is a long association system of the dorsomedial aspects of the dorsal visual stream, being fundamental to the anatomical substrates involved in peripheral vision and in the processing of visual spatial information.⁹¹⁻⁹⁶

Discussion

Although simultanagnosia is a specific term, there are variations in concepts and causes, but the mechanisms involved that stand out in the included studies are deficits in perception and attention.⁹⁷⁻¹⁰³

Studies classify it into two subtypes, in which one is due to impairment of early visual attention, while a second is related to a later impairment of the link between object and spatial information (processing).⁹⁷⁻¹⁰³

Therefore, a restricted visual window in simultanagnosia would

qualify as an impairment of visual attention in an early stage. Attention is complex and multifaceted, and the attentional defect in simultanagnosia may mirror this complexity.⁹⁷⁻¹⁰³

The proposal of a limited spatial window of attentional processing has a long history in our concepts of simultanagnosia, and recent modeling work with restricted windows of visual processing in healthy individuals shows that it has considerable explanatory power for many behavioral effects in simultanagnosia.⁹⁷⁻¹⁰³

If a spatially based attentional restriction can explain some of the phenomena frequently seen with simultanagnosia, it is worth considering whether it can also explain any of the findings that have previously been interpreted as evidence for object-based attentional deficits.¹⁰⁴⁻¹⁰⁸

Riddoch and Humphreys (2004) have argued that the fact that basic Gestalt principles such as connectivity, axis alignment, collinearity, and closure still affect patient performance indicates that "fundamental aspects of object coding continue to operate in simultanagnosia."¹⁰⁴⁻¹⁰⁸

It has been argued that simultanagnosia is a misnomer because "agnosia" implies a failure of recognition, whereas patients with simultanagnosia can typically recognize the objects they see.¹⁰⁴⁻¹⁰⁸

One scenario in which object perception requires preserved representation of spatial relations of parts, but there are more complex ways in which spatial processing is important for object perception.¹⁰⁴⁻¹⁰⁸

With this relationship between space and object perception in mind, space-based theories can explain how whole objects are neglected in simultanagnosia.¹⁰⁴⁻¹⁰⁸ Robertson et al.

argue that damage to a spatially based system may be responsible for findings that appear to indicate object-based effects, such as those demonstrating that patients with simultanagnosia extinguish objects based on object properties, regardless of space. ¹⁰⁴⁻¹⁰⁸

In the case of dysfunctional spatial input, objects may be selected based on object properties, and this explains why some objects are extinguished at the expense of others in simultanagnosia. ¹⁰⁴⁻¹⁰⁸

The basic simultanagnosia phenomenon of seeing only one object at a time can easily be explained by impaired spatial selection, with preserved object processing allowing the remaining object to be identified. ¹⁰⁴⁻¹⁰⁸

This is consistent with the conceptualization of patients with simultanagnosia as "object detectors." Indeed, there is some evidence for priority for object selection over spatial selection in simultanagnosia, implying preservation of the former and deficits in the latter. ¹⁰⁴⁻¹⁰⁸

There are theoretical accounts explaining how impairment of spatial processing can disrupt object perception. There are two types of object recognition systems: a spatial one that defines the object in terms of shape and location, and a second one as a retinotopic representation of the object that is activated by top-down information from the first system. ¹⁰⁴⁻¹⁰⁸

Disruption of this spatial system leaves the shape and size of an object unclear, a conclusion supported by computer simulations and consistent with the fact that patients with simultanagnosia sometimes have inaccurate perception of individual objects. ¹⁰⁹⁻¹¹¹ These lesions are supported by the effect of hostile

behaviors in early childhood, which occur interhemispherically as disconnection (commissurectomy) and right parietotemporal exclusion, which consequently excludes the activities of this region, such as creativity, synthesis capacity, processing speed, emotional intelligence, and cognitive flexibility. ¹⁰⁹⁻¹¹¹

Thus, with the empirical practice associated with the deduction that the fluidity of intelligence is associated with the intelligence of wisdom, or phronetics, in which it is characterized by the ability of synthesis or judgment using several knowledge at the same time. ¹⁰⁹⁻¹¹¹

Avey et al. (2012) reported small positive associations between the character strengths attributed to the virtue of wisdom and knowledge (creativity, curiosity, love of learning, judgment, and perspective) and performance on a verbal creativity task. ¹⁰⁹⁻¹¹¹ However, this pathophysiological process that occurs in childhood may be occurring silently, with intergenerational transmission, causing intelligence limitations in children who are exposed to ACE, in addition to the absence of the development of emotional and fluid intelligence. ¹¹²

Thus, we hypothesize a harmful and subtle neuromaladjustment mechanism for future approaches in new research on human intelligence.

Conclusion

Our systematic review is limited by the impossibility of performing a meta-analysis due to the small number of samples in the included studies. However, there is evidence of neurological mechanisms associated with states of simultanagnosia without a defined primary cause. It is a disorder that affects attention and thus brain processing, which influences judgment and final

perception.

We observed that less simultanagnostic states may be related to individuals with phronetic or simultaneous knowledge, and consequently, the worse their intensity, the greater the cognitive limitation, due to the restriction of capturing information from an object. ¹⁰⁹⁻¹¹¹

Another limitation is that this is the first study to propose simultanagnosia as a neuromaladaptive process, which is a new practical concept based on clinical neuroscience, and which should be better identified and observed by new experimental and population research.

Conflict of interest statement

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Bibliographic References

1. Neitzel J, Ortner M, Haupt M, Redel P, Grimmer T, Yakushev I, Drzezga A, Bublak P, Preul C, Sorg C, Finke K. Neurocognitive mechanisms of simultanagnosia in patients with posterior cortical atrophy. *Brain*. 2016 Dec;139(Pt 12):3267-3280. doi: 10.1093/brain/aww235. Epub 2016 Oct 3. PMID: 27702740.

2. Pehere NK, Dutton GN, Mankad K. Simultanagnosia as a cause of visual disturbance following Posterior Reversible Encephalopathy Syndrome (PRES): A case report. *Indian J Ophthalmol*. 2020 Jan;68(1):254-256. doi: 10.4103/ijo.IJO_807_19. PMID: 31856545; PMCID: PMC6951156.

3. Dalrymple KA, Barton JJ, Kingstone A. A world unglued: simultanagnosia as a

spatial restriction of attention. *Front Hum Neurosci*. 2013 Apr 17;7:145. doi: 10.3389/fnhum.2013.00145. PMID: 23616758; PMCID: PMC3627977.

4. Barton JJ. Disorder of higher visual function. *Curr Opin Neurol*. 2011 Feb;24(1):1-5. doi: 10.1097/WCO.0b013e328341a5c2. PMID: 21102334.

5. Himmelbach M, Erb M, Klockgether T, Moskau S, Karnath HO. fMRI of global visual perception in simultanagnosia. *Neuropsychologia*. 2009 Mar;47(4):1173-7. doi: 10.1016/j.neuropsychologia.2008.10.025. Epub 2008 Nov 6. PMID: 19038276.

6. Huberle E, Rupek P, Lappe M, Karnath HO. Perception of global gestalt by temporal integration in simultanagnosia. *Eur J Neurosci*. 2009 Jan;29(1):197-204. doi: 10.1111/j.1460-9568.2008.06559.x. PMID: 19120445.

7. Coslett HB, Lie E. Simultanagnosia: effects of semantic category and repetition blindness. *Neuropsychologia*. 2008;46(7):1853-63. doi: 10.1016/j.neuropsychologia.2007.08.024. Epub 2007 Sep 2. PMID: 18514677; PMCID: PMC2494704.

8. Veronelli L, Daini R, Mannino A, Rossetti A, Gilardone G, Corbo M, Primativo S. Global Processing Deficit in Amnesic Mild Cognitive Impairment. *J Alzheimers Dis*. 2024;101(4):1151-1165. doi: 10.3233/JAD-240375. PMID: 39302364.

9. James TW, Culham J, Humphrey GK, Milner AD, Goodale MA. Ventral occipital lesions impair object recognition but not object-directed grasping: an fMRI study. *Brain*. 2003 Nov;126(Pt 11):2463-75. doi:

10.1093/brain/awg248. Epub 2003 Sep 23. PMID: 14506065.

10.Kranjec A, Ianni G, Chatterjee A. Schemas reveal spatial relations to a patient with simultanagnosia. *Cortex*. 2013 Jul-Aug;49(7):1983-8. doi: 10.1016/j.cortex.2013.03.005. Epub 2013 Mar 27. PMID: 23643246.

11.Khan AZ, Prost-Lefebvre M, Salemm R, Blohm G, Rossetti Y, Tilikete C, Pisella L. The Attentional Fields of Visual Search in Simultanagnosia and Healthy Individuals: How Object and Space Attention Interact. *Cereb Cortex*. 2016 Mar;26(3):1242-54. doi: 10.1093/cercor/bhv059. Epub 2015 Apr 2. PMID: 25840422.

12.Leek EC, d'Avossa G, Tainturier MJ, Roberts DJ, Yuen SL, Hu M, Rafal R. Impaired integration of object knowledge and visual input in a case of ventral simultanagnosia with bilateral damage to area V4. *Cogn Neuropsychol*. 2012;29(7-8):569-83. doi: 10.1080/02643294.2012.752724. PMID: 23521054.

13.Chechlacz M, Rotshtein P, Hansen PC, Riddoch JM, Deb S, Humphreys GW. The neural underpinnings of simultanagnosia: disconnecting the visuospatial attention network. *J Cogn Neurosci*. 2012 Mar;24(3):718-35. doi: 10.1162/jocn_a_00159. Epub 2011 Nov 8. PMID: 22066584.

14.Poncet M, Caramazza A, Mazza V. Individuation of objects and object parts rely on the same neuronal mechanism. *Sci Rep*. 2016 Dec 7;6:38434. doi: 10.1038/srep38434. PMID: 27924910; PMCID: PMC5141436.

15.Morihara K, Higashiyama Y, Asano S, Matsunaga Y, Takahashi K, Miyake R, Tanaka K, Joki H, Doi H, Takeuchi H, Tanaka F. Seen by a Glance, But Not by a

Stare-A Case Study of a Patient With Simultanagnosia. *Arch Clin Neuropsychol*. 2022 May 16;37(4):865-871. doi: 10.1093/arclin/acab088. PMID: 34664637.

16.Balslev D, Odoj B, Rennig J, Karnath HO. Abnormal center-periphery gradient in spatial attention in simultanagnosia. *J Cogn Neurosci*. 2014 Dec;26(12):2778-88. doi: 10.1162/jocn_a_00666. Epub 2014 Jun 4. PMID: 24893736.

17.Thomas C, Kveraga K, Huberle E, Karnath HO, Bar M. Enabling global processing in simultanagnosia by psychophysical biasing of visual pathways. *Brain*. 2012 May;135(Pt 5):1578-85. doi: 10.1093/brain/aws066. Epub 2012 Mar 14. PMID: 22418740; PMCID: PMC3338926.

18.Suzuki K. [Neuropsychological approach to visual attention]. *No To Shinkei*. 2007 Jan;59(1):23-30. Japanese. PMID: 17228775.

19.Duncan J, Bundesen C, Olson A, Humphreys G, Ward R, Kyllingsbæk S, van Raamsdonk M, Rorden C, Chavda S. Attentional functions in dorsal and ventral simultanagnosia. *Cogn Neuropsychol*. 2003 Dec 1;20(8):675-701. doi: 10.1080/02643290342000041. PMID: 20957589.

20.Vialatte A, Yeshurun Y, Khan AZ, Rosenholtz R, Pisella L. Superior Parietal Lobule: A Role in Relative Localization of Multiple Different Elements. *Cereb Cortex*. 2021 Jan 1;31(1):658-671. doi: 10.1093/cercor/bhaa250. PMID: 32959044.

21.Nestmann S, Wiesen D, Karnath HO, Rennig J. Temporo-parietal brain regions are involved in higher order object perception. *Neuroimage*. 2021 Jul 1;234:117982. doi: 10.1016/j.neuroimage.2021.117982. Epub 2021 Mar 21. PMID: 33757908.

22.Rennig J, Langenberger C, Karnath HO. Beyond visual integration: sensitivity of the temporal-parietal junction for objects, places, and faces. *Behav Brain Funct.* 2024 Apr 18;20(1):8. doi: 10.1186/s12993-024-00233-2. PMID: 38637870; PMCID: PMC11027340.

23.Rennig J, Himmelbach M, Huberle E, Karnath HO. Involvement of the TPJ area in processing of novel global forms. *J Cogn Neurosci.* 2015 Aug;27(8):1587-600. doi: 10.1162/jocn_a_00809. Epub 2015 Mar 26. PMID: 25811709.

24.Berry BA, Meyer GJ, O'Gorman ET, Roy M, Sholander LE, Mihura JL. Simulating Local Biases in Visual Attention in Neurocognitive Performance. *Percept Mot Skills.* 2024 Feb;131(1):135-160. doi: 10.1177/00315125241228128. Epub 2024 Jan 22. PMID: 38253419.

25. Dalrymple KA, Birmingham E, Bischof WF, Barton JJ, Kingstone A. Opening a window on attention: documenting and simulating recovery from simultanagnosia. *Cortex.* 2011 Jul-Aug;47(7):787-99. doi: 10.1016/j.cortex.2010.07.005. Epub 2010 Jul 24. PMID: 20719307.

26. Barrett AY, Cheng TW, Flannery JE, Mills KL, Fisher PA, McCann CF, Pfeifer JH. Comparing the multivariate relationships of conceptual adversity models and structural brain development in adolescent girls: A registered report. *Dev Psychol.* 2024 May;60(5):858-877. doi: 10.1037/dev0001684. Epub 2024 Feb 15. PMID: 38358662; PMCID: PMC11332272.

27. Kretschmar A, Wagner L, Gander F, Hofmann J, Proyer RT, Ruch W. Character strengths and fluid intelligence. *J Pers.* 2022 Dec;90(6):1057-1069. doi: 10.1111/jopy.12715. Epub 2022 Apr 8. PMID: 35303763; PMCID: PMC9790612.

28. Hao L, Mat Ludin AF, Ahmad M, Meng X, Zhong Lei H. The prevalence and its

associated factors of psychological stress among middle school students in China: pooled evidence from a systematic scoping review. *Front Public Health.* 2024 Apr 17;12:1358210. doi: 10.3389/fpubh.2024.1358210. PMID: 38694991; PMCID: PMC11062323.

29.Cui Y, Liu Y, Yang C, Cui C, Jing D, Zhang X, Chen Y, Li B, Liang Z, Chen K, Zhang Z, Wu L. Brain structural and functional anomalies associated with simultanagnosia in patients with posterior cortical atrophy. *Brain Imaging Behav.* 2022 Jun;16(3):1148-1162. doi: 10.1007/s11682-021-00568-8. Epub 2021 Nov 17. PMID: 34787788; PMCID: PMC9107404.

30.Skipper JL. Uma voz sem boca não mais: A neurobiologia da linguagem e da consciência. *NeurosciBiobehav Rev.* 2022 Sep;140:104772. doi: 10.1016/j.neubiorev.2022.104772. Epub 2022 Jul 11. PMID: 35835286.

31.Rho HJ, Kim JH, Lee SH. Function of Selective Neuromodulatory Projections in the Mammalian Cerebral Cortex: Comparison Between Cholinergic and Noradrenergic Systems. *Front Neural Circuits.* 2018 Jun 22;12:47. doi: 10.3389/fncir.2018.00047. PMID: 29988373; PMCID: PMC6023998.

32.Schmahmann JD, Smith EE, Eichler FS, Filley CM. Cerebral white matter: neuroanatomy, clinical neurology, and neurobehavioral correlates. *Ann N Y Acad Sci.* 2008 Oct;1142:266-309. doi: 10.1196/annals.1444.017. PMID: 18990132; PMCID: PMC3753195.

33.Septien S, Rubin MA. Transtornos da Consciência: Questões Éticas de Diagnóstico, Tratamento e Prognóstico. *SeminNeurol*. 2018 Out;38(5):548-554. doi: 10.1055/s-0038-1667384. Epub 2018 15 de outubro. PMID: 30321893.

34.Graham M, Doherty CP, Naci L. Usando a Neuroimagem para Detectar Consciência Encoberta e Determinar o Prognóstico de Pacientes em Coma: Informando os Tomadores de Decisão Substitutos sobre os Resultados Individuais dos Pacientes. *SeminNeurol*. 2018 Out;38(5):555-560. doi: 10.1055/s-0038-1667385. Epub 2018 15 de outubro. PMID: 30321894.

35.Graham M, Wallace E, Doherty C, McCann A, Naci L. Da Consciência ao Prognóstico: Implicações Éticas de Descobrir Consciência Oculta em Pacientes Comportamentalmente Não Respondentes. *Ética em Saúde Camb Q*. 2019 Oct;28(4):616-631. doi: 10.1017/S0963180119000550. PMID: 31526429.

36.Di Perri C, Bahri MA, Amico E, Thibaut A, Heine L, Antonopoulos G, Charland-Verville V, Wannez S, Gomez F, Hustinx R, Tshibanda L, Demertzi A, Soddu A, Laureys S. Correlativos neurais de consciência em pacientes que emergiram de um estado minimamente consciente: um estudo de imagem multimodal transversal. *Lancet Neurol*. 2016 Jul;15(8):830-842. doi: 10.1016/S1474-4422(16)00111-3. Epub 2016 27 de abril. PMID: 27131917.

37.Calabrò RS, Bramanti P, Naro A. Rumo a novos métodos de diagnóstico em distúrbios de consciência. *Lancet Neurol*.

2016 Out;15(11):1114-5. doi: 10.1016/S1474-4422(16)30202-2. PMID: 27647635.

38.McFadyen J, Mattingley JB, Garrido MI. An afferent white matter pathway from the pulvinar to the amygdala facilitates fear recognition. *Elife*. 2019 Jan 16;8:e40766. doi: 10.7554/eLife.40766. PMID: 30648533; PMCID: PMC6335057.

39.Throuvala MA, Pontes HM, Tsaousis I, Griffiths MD, Rennoldson M, Kuss DJ. Exploring the Dimensions of Smartphone Distraction: Development, Validation, Measurement Invariance, and Latent Mean Differences

40.Anderson ED, Barbey AK. Investigating cognitive neuroscience theories of human intelligence: A connectome-based predictive modeling approach. *Hum Brain Mapp*. 2023 Mar;44(4):1647-1665. doi: 10.1002/hbm.26164. Epub 2022 Dec 20. PMID: 36537816; PMCID: PMC9921238.

41.Di Perri C, Thibaut A, Heine L, Annen J, Laureys S. Rumo a novos métodos de diagnóstico em transtornos de consciência - Resposta dos autores. *Lancet Neurol*. 2016 Oct;15(11):1115-6. doi: 10.1016/S1474-4422(16)30205-8. PMID: 27647636.

42.Jox RJ, Bernat JL, Laureys S, Racine E. Distúrbios da consciência: estamos prontos para uma mudança de paradigma? - resposta dos autores. *Lancet Neurol*. 2013 Fev;12(2):132. doi: 10.1016/S1474-4422(12)70286-7. Epub 2012 21 de dezembro. PMID: 23260191.

43. Septien S, Rubin MA. Transtornos da Consciência: Questões Éticas de Diagnóstico, Tratamento e Prognóstico. *SeminNeurol*. 2018 Out;38(5):548-554. doi: 10.1055/s-0038-1667384. Epub 2018 15 de outubro. PMID: 30321893.

44. Shu J, Qiang Q, Yan Y, Wen Y, Ren Y, Wei W, Zhang L. Distinct Patterns of Brain Atrophy associated with Mild Behavioral Impairment in Cognitively Normal Elderly Adults. *Int J Med Sci*. 2021 Jun 11;18(13):2950-2956. doi: 10.7150/ijms.60810. PMID: 34220322; PMCID: PMC8241773.

45. Byram AC, Lee G, Owen AM, Ribary U, Stoessl AJ, Townson A, Illes J. Considerações Éticas e Clínicas na Interseção de Neuroimagem Funcional e Transtornos da Consciência. *Ética em Saúde Camb Q*. 2016 Oct;25(4):613-22. doi: 10.1017/S0963180116000347. PMID: 27634713.

46. Farisco M, Laureys S, Evers K. Externalização da consciência. Possibilidades científicas e implicações clínicas. *Curr Top BehavNeurosci*. 2015;19:205-22. doi: 10.1007/7854_2014_338. PMID: 25146416.

47. Dollberg DG, Hanetz-Gamliel K, Levy S. COVID-19, problemas de comportamento infantil e ansiedade e mentalização da mãe: um modelo de moderação mediada. *CurrPsychol*. 2021 Nov9:1-12. doi: 10.1007/s12144-021476-y. Epub antes da impressão. PMID: 34776719; PMCID: PMC8577641.

48. Dollberg DG, Hanetz-Gamliel K. Ligações de Mediação-Moderação Entre ECAs das Mães, Sintomas de

Psicopatologia de Mães e Crianças e Mentalização Materna Durante a COVID-19. *Psiquiatria Frontal*. 2022 Mar 17;13:837423. doi: 10.3389/fpsy.2022.837423. PMID: 35370808; PMCID: PMC8968198.

49. Behrendt HF, Scharke W, Herpertz-Dahlmann B, Konrad K, Firk C. Como mãe, como criança? Determinantes maternos do desenvolvimento socioemocional precoce das crianças. *Saúde Infantil J*. 2019 Mar;40(2):234-247. doi: 10.1002/imhj.21765. Epub 2019 7 de fevereiro. PMID: 30731022.

50. Brazeau N, Reisz S, Jacobvitz D, George C. ENTENDENDO A CONEXÃO ENTRE O TRAUMA DO APEGO E A AUTOEFICÁCIA MATERNA EM MÃES DEPRIMIDAS. *Saúde Infantil J*. 2018 Jan;39(1):30-43. doi: 10.1002/imhj.21692. Epub 2017 27 de dezembro. PMID: 29281747; PMCID: PMC5814850.

51. Park M, Brain U, Grunau RE, Diamond A, Oberlander TF. As trajetórias da depressão materna desde a gravidez até 3 anos após o parto estão associadas ao comportamento das crianças e às funções executivas aos 3 e 6 anos. *Arch WomensMent Health*. 2018 Jun;21(3):353-363. doi: 10.1007/s00737-017-0803-0. Epub 2018 16 de janeiro. PMID: 29340801.

52. Kingston D, Kehler H, Austin MP, Mughal MK, Wajid A, Vermeyden L, Benzie K, Brown S, Stuart S, Giallo R. Trajetórias de sintomas depressivos maternos durante a gravidez e os primeiros 12 meses pós-parto e comportamento de externalização e internalização infantil aos três anos. *PLoS Um*. 2018 Abr 13;13(4):e0195365. doi:

10.1371/journal.pone.0195365. PMID: 29652937; PMCID: PMC5898728.

53.Aoyagi SS, Tsuchiya KJ. A depressão pós-parto materna afeta os resultados de desenvolvimento das crianças? J ObstetGynaecol Res. 2019 Sep;45(9):1809-1820. doi: 10.1111/jog.14064. Epub 2019 18 de julho. PMID: 31321836.

54.Whittle S, Simmons JG, Hendriksma S, Vijayakumar N, Byrne ML, Dennison M, Allen NB. Maus-tratos na infância, psicopatologia e o desenvolvimento de sub-regiões hipocampais durante a adolescência. Comportamento Cerebral. 2016 Nov 30;7(2):e00607. doi: 10.1002/brb3.607. PMID: 28239518; PMCID: PMC5318361.

55.Donges US, Suslow T. Alexitimia e processamento automático de estímulos emocionais: uma revisão sistemática. RevNeurosci. 2017 1 de abril;28(3):247-264. doi: 10.1515/revneuro-2016-0049. PMID: 28099136.

56.Ghosh S, Maunsell JHR. Neuronal correlates of selective attention and effort in visual area V4 are invariant of motivational context. Sci Adv. 2022 Jun 10;8(23):eabc8812. doi: 10.1126/sciadv.abc8812. Epub 2022 Jun 10. PMID: 35687684; PMCID: PMC9187239.

57.Li SW, Williams ZM, Báez-Mendoza R. Investigating the Neurobiology of Abnormal Social Behaviors. Front Neural Circuits. 2021 Nov 30;15:769314. doi: 10.3389/fncir.2021.769314. PMID: 34916912; PMCID: PMC8670406.

58.Poort J, Wilmes KA, Blot A, Chadwick A, Sahani M, Clopath C, Mscic-Flogel TD, Hofer SB, Khan AG. Learning and

attention increase visual response selectivity through distinct mechanisms. Neuron. 2022 Feb 16;110(4):686-697.e6. doi: 10.1016/j.neuron.2021.11.016. Epub 2021 Dec 13. PMID: 34906356; PMCID: PMC8860382.

59.Wetzel N, Widmann A, Scharf F. Distraction of attention by novel sounds in children declines fast. Sci Rep. 2021 Mar 5;11(1):5308. doi: 10.1038/s41598-021-83528-y. PMID: 33674634; PMCID: PMC7935912.

60.Han Y; COVID-Dynamic Team; Adolphs R. A shared structure for emotion experiences from narratives, videos, and everyday life. iScience. 2024 Jun 24;27(7):110378. doi: 10.1016/j.isci.2024.110378. PMID: 39100924; PMCID: PMC11296042.

61.Mair RG, Francoeur MJ, Krell EM, Gibson BM. Where Actions Meet Outcomes: Medial Prefrontal Cortex, Central Thalamus, and the Basal Ganglia. Front Behav Neurosci. 2022 Jul 5;16:928610. doi: 10.3389/fnbeh.2022.928610. PMID: 35864847; PMCID: PMC9294389.

62.Mair RG, Miller RL, Wormwood BA, Francoeur MJ, Onos KD, Gibson BM. The neurobiology of thalamic amnesia: Contributions of medial thalamus and prefrontal cortex to delayed conditional discrimination. Neurosci Biobehav Rev. 2015 Jul;54:161-74. doi: 10.1016/j.neubiorev.2015.01.011. Epub 2015 Jan 20. PMID: 25616180

63.Phillips JM, Kambi NA, Redinbaugh MJ, Mohanta S, Saalman YB. Disentangling the influences of multiple thalamic nuclei on prefrontal cortex and cognitive

control. *Neurosci Biobehav Rev.* 2021 Sep;128:487-510. doi: 10.1016/j.neubiorev.2021.06.042. Epub 2021 Jun 30. PMID: 34216654; PMCID: PMC8393355..

64.Saalmann YB. Intralaminar and medial thalamic influence on cortical synchrony, information transmission and cognition. *Front Syst Neurosci.* 2014 May 9;8:83. doi: 10.3389/fnsys.2014.00083. PMID: 24847225; PMCID: PMC4023070.

65.Varela C, Moreira JVS, Kocaoglu B, Dura-Bernal S, Ahmad S. A mechanism for deviance detection and contextual routing in the thalamus: a review and theoretical proposal. *Front Neurosci.* 2024 Feb 29;18:1359180. doi: 10.3389/fnins.2024.1359180. PMID: 38486972; PMCID: PMC10938916.

66.Hallett M, Aybek S, Dworetzky BA, McWhirter L, Staab JP, Stone J. Functional neurological disorder: new subtypes and shared mechanisms. *Lancet Neurol.* 2022 Jun;21(6):537-550. doi: 10.1016/S1474-4422(21)00422-1. Epub 2022 Apr 14. Erratum in: *Lancet Neurol.* 2022 Jun;21(6):e6. doi: 10.1016/S1474-4422(22)00179-X. PMID: 35430029; PMCID: PMC9107510.

67.Oegema R, Barakat TS, Wilke M, Stouffs K, Amrom D, Aronica E, Bahi-Buisson N, Conti V, Fry AE, Geis T, Andres DG, Parrini E, Pogledic I, Said E, Soler D, Valor LM, Zaki MS, Mirzaa G, Dobyns WB, Reiner O, Guerrini R, Pilz DT, Hehr U, Leventer RJ, Jansen AC, Mancini GMS, Di Donato N. International consensus recommendations on the diagnostic work-up for malformations of cortical development. *Nat Rev Neurol.* 2020

Nov;16(11):618-635. doi: 10.1038/s41582-020-0395-6. Epub 2020 Sep 7. PMID: 32895508; PMCID: PMC7790753.

68.Rogers JC, De Brito SA. Cortical and Subcortical Gray Matter Volume in Youths With Conduct Problems: A Meta-analysis. *JAMA Psychiatry.* 2016 Jan;73(1):64-72. doi: 10.1001/jamapsychiatry.2015.2423. PMID: 26650724.

70.Jure R. Autism Pathogenesis: The Superior Colliculus. *Front Neurosci.* 2019 Jan 9;12:1029. doi: 10.3389/fnins.2018.01029. PMID: 30686990; PMCID: PMC6334746.

71.Koller K, Rafal RD, Platt A, Mitchell ND. Orienting toward threat: Contributions of a subcortical pathway transmitting retinal afferents to the amygdala via the superior colliculus and pulvinar. *Neuropsychologia.* 2019 May;128:78-86. doi: 10.1016/j.neuropsychologia.2018.01.027. Epub 2018 Feb 3. PMID: 29410291.

72.Sosa M, Gillespie AK, Frank LM. Neural Activity Patterns Underlying Spatial Coding in the Hippocampus. *Curr Top Behav Neurosci.* 2018;37:43-100. doi: 10.1007/7854_2016_462. PMID: 27885550; PMCID: PMC5628163.

73.Dricu M, Frühholz S. A neurocognitive model of perceptual decision-making on emotional signals. *Hum Brain Mapp.* 2020 Apr 15;41(6):1532-1556. doi: 10.1002/hbm.24893. Epub 2019 Dec 23. PMID: 31868310; PMCID: PMC7267943.

74.Rolls ET, Deco G, Huang CC, Feng J. The human posterior parietal cortex: effective connectome, and its relation to

function. *Cereb Cortex*. 2023 Mar 10;33(6):3142-3170. doi: 10.1093/cercor/bhac266. PMID: 35834902; PMCID: PMC10401905.

75.Schmahmann JD, Smith EE, Eichler FS, Filley CM. Cerebral white matter: neuroanatomy, clinical neurology, and neurobehavioral correlates. *Ann N Y Acad Sci*. 2008 Oct;1142:266-309. doi: 10.1196/annals.1444.017. PMID: 18990132; PMCID: PMC3753195.

76.Rolls ET. Hippocampal spatial view cells for memory and navigation, and their underlying connectivity in humans. *Hippocampus*. 2023 May;33(5):533-572. doi: 10.1002/hipo.23467. Epub 2022 Sep 7. PMID: 36070199; PMCID: PMC10946493.

77.Marchman VA, Ashland MD, Loi EC, Munevar M, Shannon KA, Fernald A, Feldman HM. Associações entre eficiência precoce no processamento da linguagem e resultados cognitivos e da linguagem em crianças nascidas a termo e pré-termo: semelhanças e diferenças. *Neuropsicol Infantil*. 2023 Aug;29(6):886-905. doi: 10.1080/09297049.2022.2138304. Epub 2022 2 de novembro. PMID: 36324057; PMCID: PMC10151433.

78.Marchman VA, Loi EC, Adams KA, Ashland M, Fernald A, Feldman HM. A Velocidade da Compreensão da Linguagem aos 18 Meses de Idade Prevê Resultados Relevantes para a Escola aos 54 Meses de Idade em Crianças Nascidas Pré-termo. *J DevBehavPediatr*. 2018 Abr;39(3):246-253. doi: 10.1097/DBP.00000000000000541. PMID: 29309294; PMCID: PMC5866178.

79.Geyer S, Luppino G, Ekamp H, Zilles K. O lobulo parietal inferior do macaco: citoarquitetura e padrão de distribuição dos locais de ligação à serotonina 5-HT1A. *Anat Embryol (Berl)*. 2005 Dez;210(5-6):353-62. doi: 10.1007/s00429-005-0026-4. PMID: 16180022.

80.Seitz, Benjamin M et al. "Condicionamento de Ordem Superior e Dopamina: Traçando um Caminho a Seguir." *Fronteiras na neurociência comportamental* vol. 15 745388. 4 de outubro de 2021, doi:10.3389/fnbeh.2021.745388

81.Mai JK, Majtanik M. Myeloarchitectonic maps of the human cerebral cortex registered to surface and sections of a standard atlas brain. *TranslNeurosci*. 2023 Dec 26;14(1):20220325. doi: 10.1515/tnsci-2022-0325. PMID: 38152094; PMCID: PMC10751573.

82.Brazeau N, Reisz S, Jacobvitz D, George C. ENTENDENDO A CONEXÃO ENTRE O TRAUMA DO APEGO E A AUTOEFICÁCIA MATERNA EM MÃES DEPRIMIDAS. *Saúde Infantil J*. 2018 Jan;39(1):30-43. doi: 10.1002/imhj.21692. Epub 2017 27 de dezembro. PMID: 29281747; PMCID: PMC5814850.

83.Dollberg DG, Hanetz-Gamliel K, Levy S. COVID-19, problemas de comportamento infantil e ansiedade e mentalização da mãe: um modelo de moderação mediada. *CurrPsychol*. 2021 Nov 9:1-12. doi: 10.1007/s12144-021476-y. Epub antes da impressão. PMID: 34776719; PMCID: PMC8577641.

81.Kingston D, Kehler H, Austin MP, Mughal MK, Wajid A, Vermeyden L, Benzie K, Brown S, Stuart S, Giallo R. Trajetórias de sintomas depressivos maternos durante a gravidez e os primeiros 12 meses pós-parto e comportamento de externalização e internalização infantil aos três anos. PLoS Um. 2018 Abr 13;13(4):e0195365. doi: 10.1371/journal.pone.0195365. PMID: 29652937; PMCID: PMC5898728.

82.Vaeuver MS, Pedersen IE, Smith-Nielsen J, Tharner A. A depressão pós-parto materna é um fator de risco para a variabilidade emocional infantil aos 4 meses. Saúde Infantil J. 2020 Jul;41(4):477-494. doi: 10.1002/imhj.21846. Epub 2020 14 de fevereiro PMID: 32057136.

83.Aoyagi SS, Tsuchiya KJ. A depressão pós-parto materna afeta os resultados de desenvolvimento das crianças? J ObstetGynaecol Res. 2019 Sep;45(9):1809-1820. doi: 10.1111/jog.14064. Epub 2019 18 de julho. PMID: 31321836.

84.Hamann S. Diferenças de sexo nas respostas da amígdala humana. Neurocientista. 2005 Ago;11(4):288-93. doi: 10.1177/1073858404271981. PMID: 16061516.

85.Biederman J, Petty CR, Dolan C, Hughes S, Mick E, Monuteaux MC, Faraone SV. O curso longitudinal de longo prazo do transtorno desafiador de oposição e transtorno de conduta em meninos com TDAH: resultados de um estudo de acompanhamento longitudinal prospectivo controlado de 10 anos. Psicol Med. 2008 Jul;38(7):1027-

36. doi: 10.1017/S0033291707002668. Epub 2008 Jan 21. PMID: 18205967.

86.Moinho J, Petronis A. Riscos ambientais pré e perinatais para transtorno de hiperatividade por déficit de atenção (TDAH): o papel potencial dos processos epigenéticos na mediação da suscetibilidade. J Psiquiatria Psicológica Infantil. 2008 Oct;49(10): 1020-30. doi: 10.1111/j.1469-7610.2008.01909.x.Epub 2008 19 de maio. PMID: 18492038.

87.Millichap JG. Classificação etiológica do transtorno de déficit de atenção/hiperatividade. Pediatria. 2008 Fev;121(2):e358-65. doi: 10.1542/peds.2007-1332. PMID: 18245408.

88.Spencer TJ, Biederman J, Mick E. Transtorno de déficit de atenção/hiperatividade: diagnóstico, vida útil, comorbidades e neurobiologia. J PediatrPsychol. 2007 Jul;32(6):631-42. doi: 10.1093/jpepsy/jsm005. Epub 2007 7 de junho. PMID: 17556405.

89.Ollendick TH, Jarrett MA, Grills-Taquechel AE, Hovey LD, Wolff JC. Comorbidade como preditor e moderador do resultado do tratamento em jovens com transtorno de ansiedade, transtorno afetivo, déficit de atenção/hiperatividade e transtorno de oposição/comportamento. ClinPsychol Rev. 2008 Dec;28(8):1447-71. doi: 10.1016/j.cpr.2008.09.003. Epub 2008 17 de setembro. PMID: 18973971.

90.Tao H, Shao T, Ni L, Sun Y, Yan S, Gu C, Cao H, Huang K, Tao F, Tong S. [A relação entre os sintomas emocionais maternos durante a gravidez e problemas emocionais e comportamentais em crianças pré-escolares: um estudo de

coorte de nascimento]. Zhonghua Yu Fang Yi Xue Za Zhi. 2016 Fev;50(2):129-35. Chinês. doi: 10.3760/cma.j.issn.0253-9624.2016.02.006. PMID: 26926720.

91.Dricu M, Frühholz S. A neurocognitive model of perceptual decision-making on emotional signals. Hum Brain Mapp. 2020 Apr 15;41(6):1532-1556. doi: 10.1002/hbm.24893. Epub 2019 Dec 23. PMID: 31868310; PMCID: PMC7267943.

92.Šimić G, Tkalčić M, Vukić V, Mulc D, Španić E, Šagud M, Olucha-Bordonau FE, Vukšić M, R Hof P. Understanding Emotions: Origins and Roles of the Amygdala. Biomolecules. 2021 May 31;11(6):823. doi: 10.3390/biom11060823. PMID: 34072960; PMCID: PMC8228195.

93.Shin M, Jeon HA. A Cortical Surface-Based Meta-Analysis of Human Reasoning. Cereb Cortex. 2021 Oct 22;31(12):5497-5510. doi: 10.1093/cercor/bhab174. PMID: 34180523; PMCID: PMC8568011.

94.Fitz H, Hagoort P, Petersson KM. Neurobiological Causal Models of Language Processing. Neurobiol Lang (Camb). 2024 Apr 1;5(1):225-247. doi: 10.1162/nol_a_00133. PMID: 38645618; PMCID: PMC11025648.

95.Zhen S, Yu R. Neural correlates of recursive thinking during interpersonal strategic interactions. Hum Brain Mapp. 2021 May;42(7):2128-2146. doi: 10.1002/hbm.25355. Epub 2021 Jan 29. PMID: 33512053; PMCID: PMC8046141.

96.Willbrand EH, Jackson S, Chen S, Hathaway CB, Voorhies WI, Bunge SA, Weiner KS. Sulcal variability in anterior

lateral prefrontal cortex contributes to variability in reasoning performance among young adults. bioRxiv [Preprint]. 2023 Sep 2:2023.02.10.528061. doi: 10.1101/2023.02.10.528061. Update in: Brain Struct Funct. 2024 Mar;229(2):387-402. doi: 10.1007/s00429-023-02734-8. PMID: 36798378; PMCID: PMC9934691.

97.Degré-Pelletier J, Danis É, Thérien VD, Bernhardt B, Barbeau EB, Soulières I. Differential neural correlates underlying visuospatial versus semantic reasoning in autistic children. Cereb Cortex. 2024 May 2;34(13):19-29. doi: 10.1093/cercor/bhae093. PMID: 38696600; PMCID: PMC11065103.

98.Tooley UA, Park AT, Leonard JA, Boroshok AL, McDermott CL, Tisdall MD, Bassett DS, Mackey AP. The Age of Reason: Functional Brain Network Development during Childhood. J Neurosci. 2022 Nov 2;42(44):8237-8251. doi: 10.1523/JNEUROSCI.0511-22.2022. Epub 2022 Oct 3. PMID: 36192151;

99.Gellersen HM, McMaster J, Abdurahman A, Simons JS. Demands on perceptual and mnemonic fidelity are a key determinant of age-related cognitive decline throughout the lifespan. J Exp Psychol Gen. 2024 Jan;153(1):200-223. doi: 10.1037/xge0001476. PMID: 38236240; PMCID: PMC10795485.

100.Spalding KN, Jones SH, Duff MC, Tranel D, Warren DE. Investigating the Neural Correlates of Schemas: Ventromedial Prefrontal Cortex Is Necessary for Normal Schematic Influence on Memory. J Neurosci. 2015 Nov 25;35(47):15746-51. doi: 10.1523/JNEUROSCI.2767-15.2015. PMID: 26609165; PMCID: PMC4659831.

101.Cho I, Leger KR, Valoumas I, Mair RW, Goh JOS, Gutchess A. Effects of Age on Cross-Cultural Differences in the Neural Correlates of Memory Retrieval. *bioRxiv* [Preprint]. 2024 Apr 28:2024.04.25.591227. doi: 10.1101/2024.04.25.591227. PMID: 38712235; PMCID: PMC11071622.

102.Masís-Obando R, Norman KA, Baldassano C. Schema representations in distinct brain networks support narrative memory during encoding and retrieval. *Elife*. 2022 Apr 8;11:e70445. doi: 10.7554/eLife.70445. PMID: 35393941; PMCID: PMC8993217.

103.Abe N, Fujii T, Suzuki M, Ueno A, Shigemune Y, Mugikura S, Takahashi S, Mori E. Encoding- and retrieval-related brain activity underlying false recognition. *Neurosci Res*. 2013 Aug;76(4):240-50. doi: 10.1016/j.neures.2013.05.006. Epub 2013 May 28. PMID: 23726799.

104.Webb CE, Turney IC, Dennis NA. What's the gist? The influence of schemas on the neural correlates underlying true and false memories. *Neuropsychologia*. 2016 Dec;93(Pt A):61-75. doi: 10.1016/j.neuropsychologia.2016.09.023. Epub 2016 Sep 30. PMID: 27697593; PMCID: PMC5354939

105.Dennis NA, Bowman CR, Vandekar SN. True and phantom recollection: an fMRI investigation of similar and distinct neural correlates and connectivity. *Neuroimage*. 2012 Feb 1;59(3):2982-93. doi: 10.1016/j.neuroimage.2011.09.079. Epub 2011 Oct 7. PMID: 22001165.

106.Carpenter CM, Webb CE, Overman AA, Dennis NA. Within-category similarity

negatively affects associative memory performance in both younger and older adults. *Memory*. 2023 Jan;31(1):77-91. doi: 10.1080/09658211.2022.2123524. Epub 2022 Sep 21. PMID: 36131610; PMCID: PMC9991946.

107.Naspi L, Hoffman P, Devereux B, Morcom AM. Perceptual and Semantic Representations at Encoding Contribute to True and False Recognition of Objects. *J Neurosci*. 2021 Oct 6;41(40):8375-8389. doi: 10.1523/JNEUROSCI.0677-21.2021. Epub 2021 Aug 19. PMID: 34413205; PMCID: PMC8496201.

108.Naspi L, Stensholt C, Karlsson AE, Monge ZA, Cabeza R. Effects of Aging on Successful Object Encoding: Enhanced Semantic Representations Compensate for Impaired Visual Representations. *J Neurosci*. 2023 Nov 1;43(44):7337-7350. doi: 10.1523/JNEUROSCI.2265-22.2023. Epub 2023 Sep 6. PMID: 37673674; PMCID: PMC10621770.

109.Murty VP, Tomparay A, Adcock RA, Davachi L. Selectivity in Postencoding Connectivity with High-Level Visual Cortex Is Associated with Reward-Motivated Memory. *J Neurosci*. 2017 Jan 18;37(3):537-545. doi: 10.1523/JNEUROSCI.4032-15.2016. PMID: 28100737; PMCID: PMC5242406.

110.Ezzyat Y, Inhoff MC, Davachi L. Differentiation of Human Medial Prefrontal Cortex Activity Underlies Long-Term Resistance to Forgetting in Memory. *J Neurosci*. 2018 Nov 28;38(48):10244-10254. doi: 10.1523/JNEUROSCI.2290-17.2018. Epub 2018 Jul 16. PMID: 30012697; PMCID: PMC6262147.

111.Tompary A, Davachi L. Consolidation Promotes the Emergence of Representational Overlap in the Hippocampus and Medial Prefrontal Cortex. *Neuron*. 2017 Sep 27;96(1):228-241.e5. doi: 10.1016/j.neuron.2017.09.005. Erratum in: *Neuron*. 2020 Jan 8;105(1):199-200. doi: 10.1016/j.neuron.2019.12.020. PMID: 28957671; PMCID: PMC5630271.

112.Dixsaut L, Gräff J. The Medial Prefrontal Cortex and Fear Memory: Dynamics, Connectivity, and Engrams. *Int J Mol Sci*. 2021 Nov 9;22(22):12113. doi: 10.3390/ijms222212113. PMID: 34830009; PMCID: PMC8619965.

