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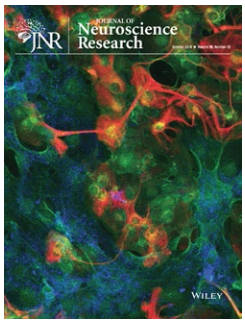
REVIEW



Neural correlates of visuoperceptive changes in severe alcohol use disorder: A critical review of neuroimaging and electrophysiological findings

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Abstract

Visuoperceptive deficits are frequently reported in severe alcohol use disorder (SAUD) and are considered as pervasive and persistent in time. While this topic of investigation has previously driven researchers' interest, far fewer studies have focused on visuoperception in SAUD since the '90s, leaving open central questions regarding the origin and implications of these deficits. To renew research in the field and provide a solid background to work upon, this paper reviews the neural correlates of visuoperception in SAUD, based on data from neuroimaging and electrophysiological studies. Results reveal structural and functional changes within the visual system but also in the connections between occipital and frontal areas. We highlight the lack of integration of these findings in the dominant models of vision which stress the dynamic nature of the visual system and consider the presence of both bottom-up and top-down cerebral mechanisms. Visuoperceptive changes are also discussed in the framework of long-lasting debates regarding the influence of demographic and alcohol-related factors, together stressing the presence of inter-individual differences. Capitalizing on this review, we provide guidelines to inform future research, and ultimately improve clinical care.

KEYWORDS

alcohol use disorder, electrophysiology, neuroimaging, vision, visuospatial

1 | INTRODUCTION

Severe alcohol use disorder (SAUD) constitutes a major health problem, associated with a large variety of psychological and cognitive impairments, among which visuoperceptive deficits (Caneva et al., 2020; Oscar-Berman et al., 2014; Stavro et al., 2013). Visuoperception refers to the ability to process, organize, and interpret visual information. This ability has significant implications for everyday living as it is involved in the monitoring of our environment. It allows the individual to deal with the constant flow of incoming visual cues and to adapt behaviors accordingly. Visuoperception is critical for humans considering not only the temporal precedence of vision in the continuum of cognitive processing but also its interplay with higher-level cerebral systems, including attentional, executive, and emotional ones (Creupelandt et al., 2019). Indeed, current models of vision stress that vision and cognition act together and do not strictly represent two independent and successive steps of cerebral processing (Newen & Vetter, 2017; O'Callaghan et al., 2017). In this framework, the efficiency of visuoperception arises from the integration of both intra-visual connections and communication paths between visual regions and higher-order areas, including, but not limited to, the frontal cortex. In other words, these models suggest that we rely on a cognitive and emotionally laden visual perception of our environment. Considering the simultaneous presence of visuoperceptive and widespread cognitive and emotional deficits in SAUD, such a claim has two implications: (1) patients with SAUD

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might have to base their decisions and judgments on a degraded visual percept (bottom-up deficit); (2) this visual percept might not be computed under the efficient supervision of higher-level cognitive and affective brain regions, partly due to limbic and frontal damage (top-down deficit) (Creupelandt et al., 2019).

While visuoperceptive deficits could play a role in cardinal signs of SAUD, by influencing attentional biases or partly explaining emotional decoding errors for instance (for a review, see Creupelandt et al., 2019), the topic of visuoperception has been rather neglected by researchers working in the field. Most studies targeting visuoperception have been conducted in the '80–'90s and very few were designed to investigate visual-related functions per se. Impaired visuoperceptive abilities were initially spotted via multi-determined behavioral neuropsychological tasks requiring efficient visual analysis such as the Rey-Oestherith Complex Figure copy or the WAIS Block Design and Object Assembly subtests (e.g., Beatty et al., 1997; Fitzhugh et al., 1960, 1965; Jones, 1971; Wilson et al., 1988). More dedicated work confirmed the presence of specific visuoperceptive difficulties by notably reporting reduced chromatic and achromatic vision (Braun & Richer, 1993; de Oliveira Castro et al., 2009; Mergler et al., 1988; Nelson et al., 1977; Reynolds, 1979), motion and speed processing (Chambers & Wilson, 1968; Pillunat et al., 1985; Wegner et al., 2001; Williams, 1984), and peripheral visual field scanning (Bertera & Parsons, 1978). Changes in figure/ground and local/global visual processing, often consistent with impoverished processing of configural cues, usually completed this profile (Beatty et al., 1997; Brandt et al., 1983; Kramer et al., 1989; Robertson et al., 1985). Together, behavioral studies thus identified multiple visuoperceptive deficits in SAUD, potentially implicating the whole retino-geniculo-cortical pathway. They also revealed that some visuoperceptive changes could last for months or even years after drinking cessation (da Cruz et al., 2016; Fein et al., 1990, 2006; Yohman et al., 1985), further justifying the need to consider their long-term consequences. However, cerebral correlates have generally been overlooked, so that the underlying neural changes remain largely unknown. At present, it is still unclear which vision-related brain networks might be disrupted by SAUD, and whether SAUD could influence both the so-called "ventral" and "dorsal" visual streams. Yet, these visual pathways constitute the basic architecture of the human visual system . They are linked to parvocellular and magnocellular retinal ganglion cells and layers from the lateral geniculate nucleus, and associated with the analysis of basic features such as form, pattern, and color (ventral or "what" pathway) and the analysis of spatial relationships and location (dorsal or "where" pathway), respectively (Bullier, 2001; de Haan & Cowey, 2011; Nassi & Callaway, 2009). While behavioral cues tend to promote mixed ventral and dorsal damage (see Creupelandt et al., in press), no comprehensive review has been conducted so far to gather together neuroscience evidence regarding dysregulation of visuoperception in SAUD.

The main objective of the present work is therefore to investigate the neural correlates of visuoperception in SAUD by summarizing evidence stemming from electrophysiological and neuroimaging studies. This review aims to reconnect this literature with recent theoretical advances regarding human vision by: (i) providing the first overview of the anatomo-functional signatures of visuoperceptive changes related to SAUD; (ii) discussing these results in light of central inter-individual demographic and alcohol-related factors (age, gender, SAUD duration, abstinence); (iii) addressing the main limitations of previous work and proposing new research avenues to improve our knowledge in the field. To do so, we reviewed studies comparing detoxified or abstinent individuals with SAUD and healthy controls on visuoperceptive-related cerebral measures. We focused on the temporal and spatial properties of visuoperception through EEG and (f)MRI data, and also covered molecular imagery. Studies needed to refer to the long-term effects of alcoholism (DSM-III; American Psychiatric Association, 1980), alcohol dependence (DSM-III-R, DSM-IV, and DSM-IV-TR; American Psychiatric Association, 1987, 1994, 2000; ICD-10 and ICD-11, World Health Organization, 2004, 2019), or severe alcohol use disorder (DSM-5; American Psychiatric Association, 2013), but not acute alcohol consumption. Exclusion criteria comprised any known ocular or neurological pathology and major organic disease, including cirrhosis. This comprehensive review only included studies using abstract or low-level visual materials as most papers relying on more complex stimuli such as emotional faces, natural scenes, or alcohol-related cues focused on higher-level decision-making or emotional processes. The few papers that did display an explicit interest for early perceptual processing were, however, included to explore the impact of sensory changes on the following processing stages (notably the P3 electrophysiological component). Importantly, our choice to conduct a narrative review was driven by the fact that very few studies, and especially (f)MRI ones, have directly targeted visuoperception in SAUD. As a result, visual-related cerebral regions have often been assessed surreptitiously through broad and relatively aspecific studies rather than been investigated per se. This lack of direct exploration of visuoperception, together with the large methodological heterogeneity across studies, makes it hard to rely on a classical literature search based on abstracts

Significance

Patients with severe alcohol use disorder (SAUD) suffer from cognitive and brain deficits, including visual impairments. However, no integrated view of these visual impairments is available, despite their critical importance in everyday life and their impact on cardinal features of SAUD. To renew researchers' and clinicians' interest in visuoperception and provide them with a solid background to work upon, we review the cerebral correlates of these visuoperceptual changes and show how they involve widespread brain regions. We also stress the limitations of the literature and emphasize new research avenues to understand the roots of the deficits and develop remediation tools.

only, and to conduct a strictly systematic review or meta-analysis. We nevertheless performed an initial systematic literature review search on the PubMed database (see Figure 1) using the following keyword algorithm:(((((((ethanol/adverse effects[MeSH Terms]) OR alcoholics[MeSH Terms]) OR alcoholism[MeSH Terms]) OR alcohol abstinence[MeSH Terms]) OR alcohol-induced disorders[MeSH Terms]) OR alcohol-related disorders[MeSH Terms])) AND (((((visual perception[MeSH Terms]) OR visual disorders[MeSH Terms]) OR visual pathways[MeSH Terms]) OR occipital lobe[MeSH Terms]) OR space perception[MeSH Terms]). The database search identified 1,078 papers published between January 1950 and October 2020, 34 of which were included in the current review. Consistent with the lack of direct investigation of visuoperception, 60 additional relevant records were retrieved through careful screening of the bibliographic references of particularly relevant papers, and additional reading. These preliminary observations underline the urgent need for a renewal of studies in the domain and further encouraged our attempt to provide a solid theoretical background for future research.

2 | TEMPORAL CHARACTERIZATION OF VISUOPERCEPTION THROUGH ELECTROPHYSIOLOGY

2.1 | Very early neural changes: Results from visual evoked potential studies

Several studies used the Visual Evoked Potential (VEP) technique (Robson et al., 2018) to investigate early cerebral responses to unpatterned luminance changes or pattern changes without luminance variations (Kothari et al., 2016; Pratt, 2012). Unpatterned stimuli most often consist of very brief flashes of white light increasing or decreasing in luminance. These flashes generate a series of electrophysiological components starting with a first negative peak occurring at around 50–70 ms (C1), followed by a positive peak at approximately 100 ms (P1) and two successive negative peaks after 130 (N1) and 200 ms (N2), respectively (Pratt, 2012). Compared to healthy controls, individuals with SAUD generally display reduced and delayed P1/N1 at frontal, central, and occipital locations

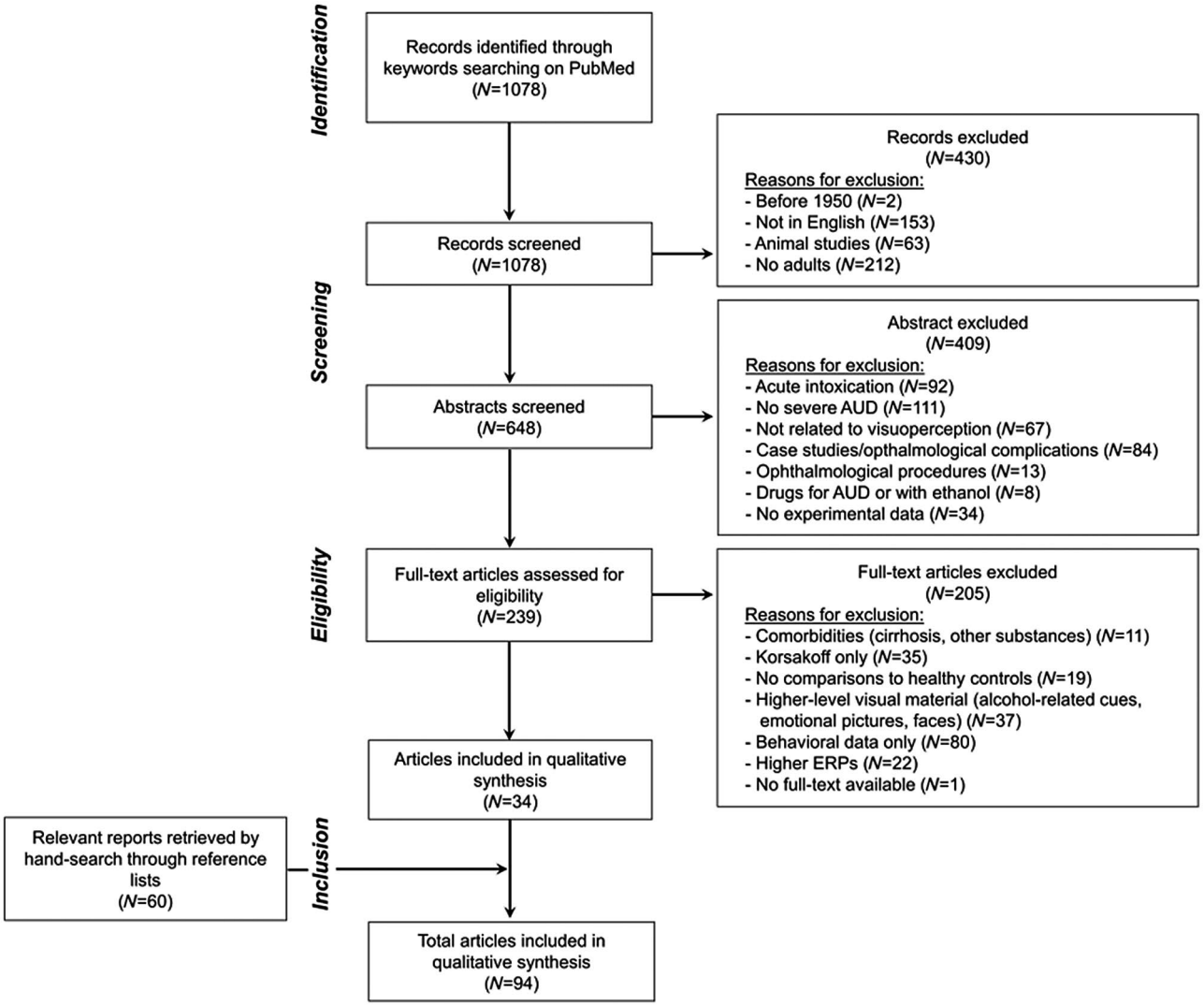

FIGURE 1 Flowchart describing the research strategy and papers included in the review

in response to repetitive flashes (Dustman et al., 1979; Porjesz & Begleiter, 1979; Williams, 1984). It has been hypothesized, based on current source estimations and animal experiments, that they could present impaired inhibitory activity from the thalamus to the visual cortex (P1) and extrastriate functioning (N1) (Kraut et al., 1985). Individuals with SAUD also do not show the typical right frontal dominance generally observed at those latencies (110–190 ms), in line with the idea of a right lateralization of visual-related functions (Coger et al., 1976; Porjesz & Begleiter, 1979). Although it has been suggested that earlier components (<100 ms) could be more resistant to alcohol-related effects (Porjesz & Begleiter, 1982a), an increase in the amplitude of early components has also been documented at parietal locations (Porjesz & Begleiter, 1979), suggesting that earlier sensory processes mediated by the parietal cortex might also be modified. Individuals with SAUD have also been characterized as "augmenters" as they tend to increase, rather than decrease, their averaged VEP responses to flashes of light of increasing intensity (von Knorring, 1976). The maximum amplitude—defined as the distance in microvolts between the highest positive peak and the lowest negative peak occurring between the middle of the visual signal and 150 ms thereafter—was more often raised than lowered with increasing stimulus intensities in individuals with SAUD compared to healthy controls. While often associated with personality traits, the proneness to be an "augmenter" has also been linked to lower brain monoamine oxidase activity (Buchsbaum et al., 1973), implying disruptions of a biological system involved in inhibition. However, it is difficult to draw precise conclusions based on unpatterned VEPs because of their wide range of shapes, sizes, and timings across the general population. Moreover, flashes tend to stimulate the whole retina and thus activate a large portion of the visual cortex (Holder et al., 2010).

Conversely, pattern VEPs have a far less intra- and inter-individual variability and provide more reliable indices of the integrity of the visual primary pathways (Pratt, 2012). Pattern stimuli, defined by their size, contrast, retinal location, and rate of presentation, generally consist of a black and white checkerboard presented in a phase-reversed manner (Brigell, 2001; Holder et al., 2010). The most commonly reported pattern-reversal VEP components are the N75 (or C1), the P100 (P1), and the N145 (N1) (Fishman et al., 2001; Holder et al., 2010), also referred to as the "NPN complex." The P1 has been identified as a robust physiological marker of visual integrity and especially conduction time. It is used as a screening tool for conditions susceptible to influence the transmission of visual sensory information, among which demyelination (Nazliel et al., 2007). In SAUD, the most robust finding is a delayed P1 latency (Ahmed & Hines, 1983; Cadaveira et al., 1991; Chan et al., 1986; Cosi et al., 1986; Nazliel et al., 2007; Porjesz & Begleiter, 1982a; Williams, 1984), sometimes associated with a concomitantly reduced amplitude (Chan et al., 1986; Cosi et al., 1986). This suggests that individuals with SAUD may display alterations in the striate cortex, the dorsal extrastriate cortex of the middle occipital gyrus, or the ventral extrastriate cortex of the fusiform gyrus, all these areas being potential P1 generators (Di Russo et al., 2002, 2005; Holder et al., 2010). Delay and changes in the waveform of the N75 (C1) (Cosi et al., 1986; Devetag, 1988; Kelley et al., 1984) and the N145 (N1) (Devetag, 1988), of striate and extrastriate origin (Di Russo et al., 2002, 2005), respectively, have also been reported, whereas other studies did not reveal any change in pattern-reversal VEP (Bauer & Easton, 1996; Emmerson et al., 1987; Kothari et al., 2018; Meinck et al., 1990). These discrepancies might be due to differences in medication (e.g., use of disulfiram in Kelley et al., 1984), length of abstinence (e.g., a few days/weeks in Cosi et al., 1986; 3 and 6 months in Bauer & Easton, 1996 and Kothari et al., 2018; more than 1 year in Devetag, 1988), or other experimental differences among which potential fluctuations in the locations, sizes, and reversal frequencies of the checkerboards (Holder et al., 2010). According to Cosi et al. (1986) and Devetag (1988), pattern VEP changes in individuals with SAUD, and especially delayed N75 and P100, could reflect a blockage and/or delay of conduction in the optic nerve, with the latency of the N75 expressing a delayed arrival time of the visual input in V1. Because these abnormalities are often asymmetric, with one eye being more preserved, the damage might be located at the optic nerve (Cosi et al., 1986), most likely postchiasmally, or at the retinal level (Chan et al., 1986). The macular bundle might be particularly impaired (Cosi et al., 1986) as VEP changes are usually accentuated for small compared to large checkerboards (e.g., Nicolás et al., 1997; Williams, 1984). Since tobacco–alcohol optic neuropathy and alcohol amblyopia are similarly characterized by impairments of central vision (Chan et al., 1986; Cosi et al., 1986), some authors have proposed that visually asymptomatic individuals with SAUD could display a subclinical form of alcohol amblyopia (Chan et al., 1986). Those impairments could result from a nutritional deficiency and/or from changes in neurotransmitter release or metabolism of cerebral amine caused by ethanol (Chan et al., 1986; Devetag, 1988). Electrolytic disturbances and modifications of the membranes and neuronal structures could especially promote demyelination (Devetag, 1988; Nazliel et al., 2007). Although of more interest than unpatterned VEPs, pattern VEPs also suffer general limitations. In particular, because of the cortical magnification of macular projections, most of the electrical activity collected generally reflects central vision (Brigell, 2001), so that activity in peripheral vision might be overlooked.

As a whole, results from flash and pattern-reversal VEP show that changes in visual processing occur as soon as 75 ms after stimulus onset in SAUD. These modifications could reflect specific damage to the retino-geniculo-cortical pathways, potentially starting at the level of the retina (Devetag, 1988).

2.2 | Implications for the following cognitive event-related potentials

Beyond changes in VEPs, which rely on the passive viewing of abstract visual stimuli, visuoperceptive deficits can also be observed in cognitive tasks requiring a response from the participant. Most cognitive Event-Related Potential (ERP) studies conducted in SAUD

have focused on the P3 complex, and particularly the P3b subcomponent, a centro-parietal positivity reflecting high-level decisional processing that is classically elicited 300 to 500 ms after the presentation of infrequent but task-relevant target stimuli during oddball paradigms (Polich, 2011). Studies have typically found that individuals with SAUD show reduced amplitude and delayed latency for the P3b compared to healthy controls (Fein & Chang, 2006; Maurage et al., 2007, 2008) and that this decrease in amplitude is more prominent in visual rather than auditory tasks (Euser et al., 2012). However, these studies often neglected the possibility that changes in amplitude and latency of earlier components such as the P100 and N100 may mirror poorer prior sensory analysis and/or introduce a time-lag affecting the latency of the following ERP components, including the P3b. In that event, individuals with SAUD may have to make a decision based on a degraded visual percept, resulting in a weakened decisional process (reduced P3b amplitude) (Maurage et al., 2007, 2008). This led to the proposal that deficits at early visual stages may last for the whole cognitive continuum, impacting later processing steps (D'Hondt et al., 2014; D'Hondt, Lepore & Maurage, 2014; Fein et al., 2009; Maurage et al., 2008). Several studies confirmed the presence of early visual disturbances in cognitive paradigms requiring to be actively engaged in a task. Relying on oddball experiments, some reported an increase in P100 amplitude (Glenn et al., 1996, but see also Begleiter et al., 1980), whereas others observed an increase in P100 latency in the absence of any significant amplitude change (Maurage et al., 2007). The increased latency of the P100 was further replicated in a visual judgment task requiring to identify the gender and emotion expressed by faces (Maurage et al., 2008). Many studies also documented a reduced N100 amplitude (Glenn et al., 1996; Olbrich et al., 2000; Parsons et al., 1990; Patterson et al., 1987; Porjesz, Begleiter, & Garozzo, 1980; Porjesz, Begleiter, & Samuelly, 1980). Even though the activity of this negative wave has frequently been studied in the context of attentional processes, it is also influenced by low-level visual properties such as luminance (Johannes et al., 1995) and therefore represents an additional potential marker of perceptual changes. This claim is further supported by the presence of a depressed N100 amplitude for both task-relevant and task-irrelevant stimuli, implying a deficit in the neural substrates of basic visual processing rather than in the selective attention network (Patterson et al., 1987). A significant latency delay has also been documented for the N170 (Maurage et al., 2008), a negative posterior wave specifically elicited by faces (Eimer, 2011; Rossion, 2014). Importantly, changes in amplitude and latency at early perceptual (P100) and face-processing (N170) stages appear to be specific to SAUD and cannot be explained by comorbid depressive states (Maurage et al., 2008; but see Hoffman et al., 2019). In addition, they have been found to correlate with the amplitude and latencies of the P300 (Maurage et al., 2007), therefore providing support to the notion that early perceptual deficits could have cumulative consequences for cognitive processing in SAUD. Finally, analyses of scalp current density topographic maps also disclosed reduced right occipito-temporal activity and increased bilateral frontal activity in individuals with SAUD during a visuospatial working memory task (Zhang et al., 1997), suggesting the presence of compensatory mechanisms based on greater frontal recruitment.

Altogether, results from ERP studies thus confirm the conclusions of VEP investigations by showing that individuals with SAUD display similar early ERP disturbances affecting the P100, N100, and N170, with potential consequences for the following cognitive processing stages and notably the P300 complex.

2.3 | Impaired synchronization between local and distant visual-related areas: results from event-related oscillations and resting-state EEG studies

Recent studies show a growing interest for Event-Related Oscillation (ERO) (Campanella et al., 2009; Pandey et al., 2012), which reflect the basic processes underpinning neural communication during cognitive tasks, and especially associative and integrative mechanisms (Rangaswamy & Porjesz, 2008). Slow frequencies are thought to mirror synchronized neuronal activity throughout distant cerebral regions, while fast frequencies correspond to the synchronization of groups of neurons in more restricted brain areas, a pattern of activity generally associated with sensory processing (Von Stein & Sarnthein, 2000). Whereas the most common finding in SAUD is a decrease in delta (1–3 Hz) and theta (3.5–7.5 Hz) EROs, thought to reflect the high-level processes underlying the P3 ERP component (e.g., Andrew & Fein, 2010; Pandey et al., 2012; Porjesz & Begleiter, 2003; Rangaswamy & Porjesz, 2014), changes in the gamma band (28.5–50 Hz) have also been highlighted (Padmanabhapillai et al., 2006; Sion et al., 2020). Interestingly, gamma oscillations have been linked to vision and integrative functions such as "feature binding" (e.g., shape and color) as well as top-down interactions with frontal regions during sensory processing (Başar et al., 1999; Porjesz & Begleiter, 2003). In healthy controls, locked gamma activity is usually increased in response to attended rather than unattended stimuli in oddball paradigms, consistent with the P3 ERP component (Başar et al., 1999). The opposite profile is, however, observed in individuals with SAUD, with lower early gamma power (0–150 ms) in response to target stimuli (Padmanabhapillai et al., 2006). Top-down processing is particularly important when the mapping between sensory inputs, thoughts, and actions is weakened (Miller & Cohen, 2001), as it is most probably happening in SAUD. This lower gamma power, also observed at rest (Sion et al., 2020), combined with the potential decrease in communication between distant areas indexed by lower responses in the delta and theta range, might suggest impaired frontal-visual connectivity in SAUD. Such a proposal is consistent with results from resting-state EEG documenting lower spectral power in the alpha frequency range (7.5–12 Hz) in bilateral occipital areas of individuals with SAUD, although specifically in men (Ehlers & Phillips, 2007). It also converges with recent findings showing that the spectral entropy features that best discriminate the visual ERPs of individuals with SAUD and healthy controls in a specific gamma sub-band (30–55 Hz) during a visual object recognition task are located in the frontal, fronto-parietal, and occipital regions (Padma

Shri & Sriraam, 2016, 2017). According to the authors (Padma Shri & Sriraam, 2017), activity in these regions reflects the specificity of individual with SAUD's sensory control, attentional, and visual processes. Finally, and while not directly applicable to SAUD, a magnetoencephalographic study focusing on the occipital activity of heavy drinkers during a visual-spatial processing task requiring to detect the location of black and white checkerboards also revealed reduced activity of the alpha band in lateral visual association cortices (Lew et al., 2020). This change was found in the absence of any differences in theta and gamma oscillations in the medial primary visual cortices and was also interpreted as a sign of impaired top-down visual processing in extra-striate areas.

In sum, ERO studies suggest modifications in both low and high spectral frequencies that could be related to disturbances in neural synchronization between local and distant cerebral areas, and especially impaired communication between occipital and frontal regions.

3 | SPATIAL CHARACTERIZATION OF VISUOPERCEPTION THROUGH NEUROIMAGING

3.1 | Impoverish white and gray matter integrity: Results from structural MRI studies

Anatomical studies in SAUD generally point toward the presence of widespread volume reduction of white and gray matter, most commonly associated with a specific vulnerability of frontal regions as well as dorsal striatal/insular and cingulate damage (Bühler & Mann, 2011; Rolland et al., 2020; Xiao et al., 2015). Nevertheless, and while they are generally less explored, visual-related regions also appear to suffer structural damage.

Gray matter volume reductions have been reported in parts of the occipital lobe (Durazzo et al., 2015, 2017; Fein et al., 2009; Mackey et al., 2019). Both data-driven region-by-region comparisons (Fortier et al., 2011) and comparisons based on a priori ROI computation (Uhlmann et al., 2018) show that SAUD is associated with decreased occipital cortical thickness. Although occipital changes might not be systematic (Cardenas et al., 2007; Durazzo et al., 2015; Sullivan et al., 2018), volume reductions in other broad cerebral areas involved in visual processing such as the parietal and temporal cortex are also often observed (Durazzo et al., 2015, 2017; Fein et al., 2009; Sullivan et al., 2018). These regions notably include the inferior and superior parietal cortex as well as the inferior, middle, and superior temporal gyri and the precuneus (Mackey et al., 2019). Changes in gray matter volumes have also been observed in more restricted structures known to subserve better defined visual functions, among which the cuneus (Rando et al., 2011; Wang, Fan, et al., 2018), lingual gyrus (Zhu et al., 2018), and fusiform gyrus (Mackey et al., 2019). The lingual gyrus is involved in the early perception of the global form of a visual stimulus (Frith et al., 1992), whereas the cuneus closely interacts with V1 and contributes to modify the quantity and quality of information reaching later visual processing stages (Vanni et al., 2001). The fusiform gyrus, together with the lingual gyrus, also contributes to various high-level visual recognition and categorization mechanisms and is often considered to be face selective (Weiner & Zilles, 2016). In the same line, recent reports of increased rightward asymmetry of gray matter in the lingual gyrus and cerebellum of individuals with SAUD (Zhu et al., 2018) support damage of the visual network, and notably V1.These changes might be linked to the microstructural damage of the corpus callosum frequently reported in SAUD (e.g., Pfefferbaum et al., 2000). Importantly, smaller gray matter volumes in medial frontal as well as parietal-occipital regions (including the precuneus, cuneus, and posterior cingulate regions) are predictive of shorter time to any alcohol use and heavy drinking relapse at 3-month follow-up (Rando et al., 2011; Wang, Fan, et al., 2018). Because no visuospatial tests were used in these studies, no conclusion can be drawn in terms of direct behavioral correlates. However, other researchers did find a negative association between severity of parietal gray matter shrinkage and individuals with SAUD's performances on computerized and paper-pencil visuospatial tasks including the WAIS Block Design subtest (Fein et al., 2009).

Beyond gray matter changes, white matter impairments have also been documented in different cerebral regions, including the fronto-occipital fasciculus, namely the long associative white matter fiber tract connecting parieto-occipital regions with dorsolateral premotor and prefrontal areas (Schmahmann & Pandya, 2007). Measures of low fractional anisotropy and high mean diffusivity in fronto-occipital fasciculus bundles have been found to correlate with individuals with SAUD's performances in behavioral tasks requiring to copy different shapes and figures (Bagga, Sharma, et al., 2014). Also, compromised myelin integrity in the occipital forceps and left occipital cortex (Pandey et al., 2018; Rosenbloom et al., 2009), suggest the presence of concomitant intra-occipital impairments in visual transmission. These intra-occipital degradations also correlated with individuals with SAUD's copy accuracy in analogous drawing tasks, making them additional potential useful markers of behavioral performances. Besides changes in (fronto-)occipital regions, degradation of each major node of the frontocerebellar circuitry has also been reported (Sullivan, 2003). Cerebellar hemispheric white matter volume predicted individuals with SAUD's performance at the Hidden Figure Test (i.e., a test measuring the ability to extract figures from complex backgrounds, Gottschaldt, 1926), stressing the role of the cerebellum in the regulation and automatization of recently learned abilities, among which visuospatial skills. Consistent with gray matter results, white matter alterations of the splenium of the corpus callosum have also been reported in SAUD and are considered to contribute to an inefficient inter-hemispheric transfer of visual information. For instance, callosal degradation has been linked to impaired hemispheric asymmetry in individuals with SAUD (Schulte et al., 2010), as indexed by different patterns of visuomotor advantages depending on the spatial location of a target (left/right) and the hand used to react to it (left/right). It has also been associated with slower visuospatial and visuomotor responses in classical neuropsychological tasks such as the WAIS Digit Symbol

subtest (Pfefferbaum et al., 2006) or the part A of the Trail Making Test (Pfefferbaum & Sullivan, 2002). Finally, callosal microstructural integrity in SAUD does not show the expected relationship with the congruency and interference effects triggered by hierarchical visual stimuli such as Navon letters (i.e., a big letter composed of similar or dissimilar small letters) (Müller-Oehring et al., 2009). While these effects depend on posterior callosal regions in healthy individuals—in line with the role of posterior regions in early stimulus perception and identification, as well as bilateral cerebral communication—they are tought to rely upon frontal regions, and especially bilateral fronto-parietal attentional systems in individuals with SAUD, implying the recruitment of higher-order cerebral areas.

Other works also support the presence of potential compensatory mechanisms in SAUD. Their exact directionality remains unclear though, as some results suggest the involvement of additional frontal regions, while others promote the recruitment of more posterior cerebral circuits to take over impaired frontal functioning. For instance, it has been proposed that the inferior cingulate bundle that connects frontal regions with medial temporal regions, including episodic memory systems, could serve as an alternative pathway to the compromised frontal circuitry during visuoperceptive tasks (Rosenbloom et al., 2009). In individuals with SAUD, the integrity of this structure correlated with copy strategy and recall performances on the Rey–Osterrieth complex figure. Pure copy scores, which constitute a more direct index of visuoconstruction abilities, were also associated with the integrity of the occipital forceps, external capsule, and superior longitudinal fasciculus, implying a role for the close interface between frontal and visual regions. Among healthy controls, copy scores were associated with the occipital forceps, the external capsule, but also the frontal forceps rather than the superior longitudinal fasciculus. Apart from cortical compensatory mechanisms, individuals with SAUD could also potentially recruit other brain regions, including the cerebellum, as suggested by results showing that performance at the Hidden Figure Test is better predicted by the integrity of the cerebellum rather than the parietal cortex in individuals with SAUD (Sullivan, 2003).

From a structural point of view, individuals with SAUD present with both gray and white matter alterations in various regions involved in visuoperception, including temporal, parietal, and occipital areas, but also cerebellar and callosal structures. Compromised myelin integrity within occipital areas, as well as between frontal and occipital regions could contribute to lower individuals with SAUD's behavioral performances, notably by impairing the fronto-occipital fasciculus. Depending on the preserved brain regions, different compensation mechanisms could coexist.

3.2 | Reduced activity in key nodes of the visual system: Results from fMRI studies

Lower fMRI activity has been measured in the striate (V1) and extra-striate (V2–V5) visual cortices of individuals with SAUD in response to passive visual stimulation (Hermann et al., 2007) and during reaction time tasks measuring visuomotor speed for unilateral or bilateral visual targets (Schulte et al., 2010). Broad changes in connectivity have also recently been documented in ventral and dorsal areas, usually associated with the temporal and parietal cortices. Chen et al. (2019), in particular, observed impairments in the ventral visual pathway-cerebellar circuit of individuals with SAUD. They showed that measures of functional connectivity density within specific regions of the visual association cortex, encompassing the fusiform, lingual, and mid-occipital gyri, allowed to discriminate between healthy controls and individuals with SAUD with a higher degree of specificity and sensitivity (both around 92%) than that of frontal areas. This result, reflecting less correlated activity in functional hubs of the ventral stream responsible for the processing of different properties such as shape, color, and size, raised the possibility of reduced network communication, with potential implications for central adaptive abilities (e.g., the ability to distinguish relevant information from irrelevant one). This disorganization of information transmission within ventral visual areas also correlated positively with SAUD severity. Complementary results from graph theory applied to the resting-state fMRI data of individuals with SAUD also revealed a decrease in global efficiency of brain networks associated with reduced nodal efficiency in the right fusiform gyrus, right superior temporal gyrus, right inferior occipital gyrus, and left insula (Wang et al., 2018). Consistent with impaired occipito-frontal communication, individuals with SAUD exhibited hypo-connectivity between the right dorsolateral prefrontal cortex and the right superior occipital gyrus. Reduced nodal efficiency was measured in the right orbitofrontal cortex (a region lying at the intersection of sensory, memory, and affective processing, Rolls & Grabenhorst, 2008) while an increased nodal efficiency was observed in the left part of the same structure. As a whole, the authors concluded toward a lack of efficient communication between a series of multi-sensory modalities brain areas (Wang, Zhao, et al., 2018), including the face-voice integration network, lending support to previous evidence of reduced functional connectivity between areas of the ventral visual stream and frontal structures (Maurage et al., 2013). Conversely, other findings rather support the presence of an impaired dorsal network involving the superior parietal and dorsolateral prefrontal cortex (Pfefferbaum, Desmond, et al., 2001). For instance, Karamzadeh et al. (2015) found changes in frontal, anterior frontal, centro-parietal, parieto-occipital, and occipital lobes, especially in the right cerebral hemisphere, of individuals with SAUD compared to healthy controls during an active task requiring to judge whether two successive object pictures were similar. The analysis of spontaneous fluctuations in BOLD-fMRI signal intensity of the resting brain also disclosed increased intensity in the right inferior parietal lobule and right supplementary motor area of individuals with SAUD, in combination with decreased intensity in the left precuneus and bilateral cerebellum posterior lobe (Liu et al., 2018). These areas could also predict membership (SAUD vs. healthy control) with high sensitivity and specificity. Finally, and consistently with results from structural MRI, reduced functional connectivity between regions surrounding the parieto-occipital sulcus, including the cuneus and

prefrontal areas, has been found to predict future relapse (Wang, Fan, et al., 2018). Resting-state investigations additionally confirmed that synchronization within the bilateral middle occipital gyrus could predict relapse independently of regions comprising the reward and executive control networks (Camchong et al., 2013). Recent data collected during a visual attentional task requiring to track and judge sequences of moving balls agree upon the presence of spatially diffuse cerebral damage involving both ventral and dorsal areas, as well as prefrontal cortices, reconnecting together previous fragmented results (Zehra et al., 2019).

Results from fMRI also indicate the presence of potential compensatory mechanisms. While scarce, evidence suggests differences in ventral/dorsal activation in individuals with SAUD. For example, while healthy controls rely on a dorsal circuit comprising superior parietal and dorsolateral prefrontal regions to perform a spatial working memory task, individuals with SAUD appear to rely on a more ventral circuit comprising medial temporal and ventral prefrontal areas to achieve a similar level of behavioral performance (Pfefferbaum, Desmond, et al., 2001). This change in response was interpreted as a way to counterbalance dorsal impairment and is consistent with reduced BOLD response observed in parietal regions in another spatial working memory task (Tapert et al., 2001). Besides, the brain activity of individuals with SAUD measured at rest is also characterized by an increase in short-range functional connectivity density in the left precentral gyrus, right salience network, and right cingulate gyrus combined with a decreased connectivity in ventral visual regions (Chen et al., 2019). Chen et al. (2019) advanced two explanations for these results: (1) a compensatory proposal based on the gathering of additional resources to maintain the same performance level; (2) an explanation in terms of enhanced neural effort to counter the neurotoxicity of alcohol on cerebral structures. Importantly, increased nodal efficiency has also been spotted in the left orbitofrontal cortex of individuals with SAUD (Wang, Zhao, et al., 2018), implying a role for higher-order integrative regions. Considering that the activity of the orbitofrontal cortex is closely linked to the associative content of visual information and contributes to the efficient computation of visuo-affective predictions through top-down visual regulation (Chaumon et al., 2014), it has been speculated that individuals with SAUD could enhance early orbitofrontal activity to increase their top-down control over visual information (Wang, Zhao, et al., 2018). However, this compensatory mechanism might be largely hampered by the severity of individuals with SAUD's brain damage, and especially impaired fronto-occipital connectivity, leading to genuinely reduced visual processing skills. Whatever the exact underlying processes, caution is advised as the recruitment of additional or alternative cerebral regions and spatially expanded connectivity might not always express functional compensation. Müller-Oehring et al. (2015) underscore that such extended activity might also reflect a failure to keep neural coherence confined to relevant and specialized networks, and might, therefore, be viewed as a form of "network dedifferentiation" associated with lower performances. Nonetheless, their resting-state fMRI results rather support an explanation in terms of "compensatory ability", at least concerning visual perception. Indeed, expanded and greater connectivity between the dorsolateral prefrontal cortex and the cuneus was associated with normal performances in individuals with SAUD (Müller-Oehring et al., 2015). Given that the authors only used the block span test to assess visuospatial functioning offline, and that this test comprises an important working memory component, one could, however, question the generalizability of their results. More globally speaking, precise conclusions are precluded by the low number of studies directly exploring the functional correlates of specific visuoperceptive abilities in SAUD.

In sum, individuals with SAUD present patterns of both enhanced and reduced functional activity/connectivity within and between essential components of the visual system. Results tend to support primary dysregulation of ventral visual pathways, although dorsal damage cannot be excluded. Consistent with structural findings, they also ascribe a role to frontal regions, and fronto-occipital routes, in the efficient monitoring of visuoperception in SAUD.

3.3 | Metabolic changes: Results from molecular imagery

Lower brain glucose metabolism has been reported in portions of the occipital cortex of individuals with SAUD (Tomasi et al., 2019; Volkow et al., 1992; Wik et al., 1988), together with decreased levels of $GABA_A$/benzodiazepine receptor bindings in occipital (Abi-Dargham et al., 1998; Behar et al., 1999) and parietal (Lingford-Hughes et al., 1998) regions (but see Lingford-Hughes et al., 2000; Mason et al., 2006). Signs of cerebral inflammation, indexed by changes in histaminergic neurotransmission (Alakarppa et al., 2003) have also been observed in the occipital cortex, along with a tendency to express more annexin IV compared to healthy controls in occipital but not frontal regions (Sohma et al., 2001). Annexins, a family of proteins binding to Ca^{++} and phospholipids, are increased in other pathologies such as ischemia and Alzheimer's disease, thereby constituting another biomarker of interest for SAUD. Other signs of neurotransmitter dysregulations have also been documented, among which higher serotonin synthesis in the occipital lobe and left superior temporal gyrus, concomitantly with lower serotonin synthesis in frontal areas in the absence of brain volume change (Nishikawa et al., 2009). Considering that patients had only been abstinent for a few hours, this last result must be viewed with care. Changes in serotonin levels were especially located in Brodmann area 19, namely a visual area with multimodal integrative properties, and could thus be partly explained by withdrawal effects (e.g., visual hallucinations, even if unreported in the study). Regarding behavioral correlates, individuals with SAUD's ability to draw and reproduce patterns of cubes have been found to correlate with changes in N-acetyl-aspartate, glutamate–glutamine, and choline, considered as a marker of neuronal viability, an excitatory neurotransmitter, and an indicator of myelination and cell membrane metabolism, respectively, within the primary visual cortex (Bagga, Khushu, et al., 2014). It has been proposed that changes in metabolite ratios could reflect

cellular membrane damage, a potential cause of reduced connectivity within occipital regions and, in fine, lead to decreased visual performances (Bagga, Khushu, et al., 2014; Modi et al., 2011). Finally, beyond intra-visual changes, and consistent with potential fronto-occipital disconnections, a single-photon emission computed tomography study showed that reduced regional blood flow in the occipital cortex of individuals with SAUD was accompanied by an hypo-perfusion in the frontal lobe (Nicolás et al., 1993).

Taken together, molecular imaging studies revealed a series of metabolic disruptions in the occipital lobe involving several important inhibitory and excitatory neurotransmitters.

4 | HOW DO DEMOGRAPHIC AND ALCOHOL-RELATED VARIABLES INFLUENCE VISUOPERCEPTION?

4.1 | Do women with SAUD display more severe visuoperceptive alterations?

The gender vulnerability effect refers to the hypothesis that women might be more sensitive to the deleterious effects of alcohol, notably due to their slower metabolization of ethanol promoting higher blood alcohol concentrations (Acker, 1985). So far, very few studies have investigated gender differences in visuoperception in SAUD.

Only limited electrophysiological results showed a specific gender interaction, and these results rather support a reverse effect. For instance, resting-state EEG revealed lower spectral power in the alpha frequency range in bilateral occipital areas in men but not women with SAUD (Ehlers & Phillips, 2007). Likewise, altered visual N1 amplitudes have been observed in men but not women with SAUD compared to gender-matched healthy controls in a visual oddball paradigm (Parsons et al., 1990). Interestingly, this dissociation appeared in contradiction with the pattern of behavioral results since women were more impaired than men on a composite visuospatial score based on the WAIS Block Design, Digit Symbol, and figural memory tasks, among others. This raises the possibility that gender differences might depend on the type of measurement. The fact that no ERP alteration emerged in women even after equating years of SAUD duration across genders further led the authors to suggest that: (1) gender differences cannot be explained by variations in SAUD duration; and (2) ERP measures might not always be causally related to neuropsychological performances (Parsons et al., 1990). By contrast, no gender difference emerged in a study exploring the N75 (or C1) and P1 components generated by pattern-reversal visual stimuli (Bauer & Easton, 1996). The authors only observed a trend toward shorter latencies in women with SAUD. However, this analysis collapsed SAUD patients with previous cocaine users, hence reducing the specificity of the results. As a whole, electrophysiological studies thus do not support the presence of more severe visuoperceptive deficits in women with SAUD.

At the anatomical level, results differ according to the study focus. When comparing the overall cerebral volumes of women and men with SAUD and adjusting for intracranial sizes, some researchers found the magnitude of differences in brain volumes compared to healthy controls to be greater in women than men, particularly for gray matter (Hommer et al., 2001), while other found cortical volume deficits in men only (Pfefferbaum et al., 2001). Explorations of more defined cerebral regions also go against the hypothesis of a higher toxicity of alcohol on women's brains. For example, Fein et al. (2009) reported a specific gender by group interaction wherein men with SAUD displayed the most observable reductions in orbitofrontal as well as primary visual and somatosensory gray matter volumes. Changes in glucose metabolism are also more consistently found in the occipital, parietal, and temporal brain regions of men (Martin et al., 1992; Volkow et al., 1992, 1994) than women with SAUD (Wang et al., 1998). Results are less straightforward for other relevant regions such as the corpus callosum. While direct gender comparisons suggest that the size of this structure is reduced in women but not men with SAUD (Hommer, 1996), separate gender analyses uncover significant volume changes in men (Pfefferbaum et al., 1996) but not women (Pfefferbaum et al., 2002). Men and women could display distinct microstructural changes in the absence of broad macrostructural differences: women could suffer more severe white matter deficits in central callosal regions whereas men could present abnormalities in the splenium in the absence of gender difference in total callosal size (Pfefferbaum & Sullivan, 2002).

It is difficult to conclude to a total absence of gender difference given the limited number of studies specific to visuoperception and the mixed results obtained. However, several electrophysiological and neuroimaging work suggest that men could be more severely impaired so that a higher vulnerability in women seems unlikely.

4.2 | Is there premature aging of visuoperceptive functions?

Age has also continuously raised researchers' interest, notably through the idea that SAUD could induce premature cognitive aging. Two slightly different models have been put forward to explain the similarities between individuals with SAUD's and elderly non-drinking individuals' profiles (Evert & Oscar-Berman, 2001). According to the "accelerated aging model," SAUD accelerates the cognitive effects of normal aging of both young and old individuals with SAUD (Eckardt et al., 1980; Noonberg et al., 1985). By contrast, the "increased vulnerability model" posits that individuals with SAUD only experience premature aging after the first expected manifestations of normal aging began (Goldman et al., 1983; Jones & Parsons, 1971).

Although these hypotheses have not been developed specifically in relation to visuoperception, parallelisms between individuals with SAUD and elderly people's patterns of VEP have already been proposed 40 years ago by Dustman et al. (1979) who documented similar amplitude and latency disturbances of the N1/P1 components in these two populations. In their study, the cerebral responses of young individuals with SAUD differed from those of age-matched

healthy controls and mimicked those of elderly healthy controls, supporting the "accelerating aging" proposal. Similar steeper age-related increases in latency of the C1 have also been reported (Kelley et al., 1984), suggesting that the effects of alcohol on very early visual processes could increase with age. On the side of the "increased vulnerability model," evidence suggests that individuals with SAUD who display significantly longer P1 latencies are older than those who demonstrate normal VEP (Nicolás et al., 1997). However, P1 latencies were solely predicted by the total amount of alcohol intake of individuals with SAUD, and not by their age, so that these visual changes might reflect longer periods of SAUD rather than increased vulnerability with older age. Even though researchers have not yet come with a definitive consensus on the matter, individuals with SAUD and elderly healthy controls' electrophysiological disturbances could differ in their nature. Closer examination of the data of Dustman et al. (1979) suggests the presence of dissimilarities in the pattern of VEP between elderly healthy controls and individuals with SAUD for very early cerebral waves (~60–80 ms)—reflecting initial reception of sensory stimuli—as well as for the P1/N1 components when recorded at central rather than occipital locations (Porjesz & Begleiter, 1982a). Even though elderly people's and individuals with SAUD's occipital P1 and N1 variations looked alike, Porjesz and Begleiter (1982a) argue that such dissociation between occipital and frontal locations would not be expected if chronic alcohol consumption was indeed able to accelerate the aging process. In their view, the premature aging effect should not differentially affect the activity of primary sensory areas. They stress that the delayed P100 observed in individuals with SAUD is not consistently documented in older healthy controls and consider that many methodological differences between studies prevent meaningful comparisons and speculations about the physiopathology underlying this delay in both aging and SAUD. Regarding the common P3 disturbances reported in visual oddball paradigms, Porjesz and Begleiter (1982b) also comment that individuals with SAUD generally display a reduction in amplitude associated with a lack of differentiation between relevant and irrelevant stimuli, while P3 deficits in older healthy controls are most often characterized by prolonged latencies. These qualitative differences suggest that individuals with SAUD may present with impaired "sensory-filtering" and "probability-matching" mechanisms, leading to undifferentiated cerebral responses for different targets, whereas elderly people could still successfully evaluate targets, but at the expense of speed.

Inconclusive structural neuroimaging results also emerged since Courville (1955) first wrote that individuals with SAUD and normal elderly people could present strikingly analogous cortical atrophy. A large-scale longitudinal study exploring the accelerated aging hypothesis (Sullivan et al., 2018) recently concluded that age-alcohol interactions could induce frontal cortical damage in older individuals with SAUD but that such interactions could not be observed in occipital and parietal cortices. However, other findings suggest that individuals with SAUD could exhibit lower regional volumes than age-related healthy controls in different areas of interest for vision such as the precuneus and the superior parietal gyrus (Guggenmos et al., 2017). Drawing on these results, researchers calculated that the brain age of individuals with SAUD could be increased up to 11.7 years (Guggenmos et al., 2017). Age-alcohol interaction has also been observed in other regions of interest, such as the corpus callosum, with smaller volumes and higher diffusivity in the genu and splenium of old individuals with SAUD compared to younger ones (Pfefferbaum et al., 2006). Based on purely structural resemblances, the possibility that some visual abilities might endure an accelerated aging process thus cannot be ruled out. Behavioral correspondences are nonetheless drastically lacking to demonstrate significance for actual visual functioning.

Results from functional neuroimaging studies provide more convincing arguments against the premature aging of vision in SAUD by showing that the pattern of functional connectivity of individuals with SAUD and elderly healthy controls do not relate similarly to visual performances. Indeed, while the cerebral activity of both groups is characterized by less specific and distinctive activity in frontal and posterior brain regions, as well as modified inter-connectivity between these areas, these changes might not have the same significance in both groups, at least for visuoperception. In elderly people, they are associated with reduced visuoperceptive performances, and most probably reflect a reduced ability to confine neural activity within specialized networks and to synchronize it with additional brain regions (Goh, 2011). By comparison, these connectivity changes show a positive link with visuoperceptive scores in individuals with SAUD, indicating that they might be better understood as a neural compensatory mechanism allowing them to reach higher performances. Results from resting-state functional brain networks investigations especially showed that expanded calcarine-inferior orbitofrontal connectivity in individuals with SAUD correlates positively with visuospatial working memory (measured with the block span task), suggesting that this spatial expansion might indeed enhance their performances (Müller-Oehring et al., 2015). Such a relationship is network specific as it does not apply to the default mode, executive control, and salience networks in which greater and more spatially distributed connectivity is associated with lower performances (Müller-Oehring et al., 2015). In other words, elderly people may experience functional dedifferentiation in the visual network whereas individuals with SAUD may rely on more or less efficient compensatory mechanisms to support functions sustained by this network. According to the compensatory hypothesis (e.g., Ham & Parsons, 1997; Tracy & Bates, 1994), the gradual rather than sudden expression and onset of alcohol-related deleterious effects could favor the reorganization of damaged functions, and this reorganization might occur more frequently in younger individuals with SAUD due to higher cerebral plasticity.

Taken together, neuroscience results tend to refute the idea of premature aging of visual functions. Despite structural similarities, changes in visual-related areas do not seem to share the same implications in the aging and SAUD populations.

4.3 | Is visuoperception influenced by SAUD duration and amount of alcohol consumed?

The causal role of alcohol constitutes another major cross-dimensional research question in SAUD. Due to the lack of longitudinal data allowing causal attributions, researchers generally examine the association between the extent of alterations and SAUD duration/severity to assess whether cerebral changes might be linked to the neurotoxic effects of alcohol.

Mixed results are found for the P100 electrophysiological component, with researchers reporting a correlation between the delayed latency and the lifetime dose of ethanol consumed (Nicolás et al., 1997) and others failing to find any link (Meinck et al., 1986; Nazliel et al., 2007). Changes in EROs do not appear to correlate with alcohol-related characteristics, as illustrated by the absence of association between the lower spectral power in the alpha frequency measured at occipital regions in individuals with SAUD and alcohol-related characteristics, including quantity and frequency of drinking (Ehlers & Phillips, 2007). Structural white matter changes and gray matter volumes fluctuations in visuospatial-related regions such as the occipital or parietal cortex do not appear to vary according to lifetime doses and drinking severity either in most studies (Cardenas et al., 2007; Rando et al., 2011; Sullivan, 2003; Tomasi et al., 2019; Uhlmann et al., 2018; Zhu et al., 2018). There are, however, exceptions, with studies showing a significant relationship between: (i) gray matter shrinkage in parietal (but not occipital) regions and alcohol doses (Fein et al., 2009); (ii) white matter alteration of the superior longitudinal fasciculus and lifetime alcohol consumption (Pfefferbaum et al., 2009). This second result reconnects with the idea that alcohol could deteriorate long association fiber tracts binding distant cerebral areas but will need to be further confirmed as another study found no link between the integrity of the inferior fronto-occipital fasciculus fiber bundles and SAUD duration, age at first drinking, age at drinking onset, and AUDIT score (Bagga, Sharma, et al., 2014). At the molecular level, except for an association between total lifetime alcohol and cerebral glucose metabolic rate (Tomasi et al., 2019), no relation has been found between alcohol consumption and changes in metabolites (Bagga, Khushu, et al., 2014), $GABA_A$/benzodiazepine receptor bindings (Abi-Dargham et al., 1998), and serotonin synthesis (Nishikawa et al., 2009) measured at occipital regions. By contrast, the integrity of other regions, and especially the corpus callosum, appears more consistently related to lifetime alcohol intake (Pfefferbaum et al., 2006; Schulte et al., 2005; Yeh et al., 2009; but see also Konrad et al., 2012). However, behavioral performances in visual tasks requiring efficient inter-hemispheric transfer generally do not correlate with lifetime consumption data, rendering the overall dynamic between alcohol, behavior, and cerebral correlates less clear (Kramer et al., 1989; Müller-Oehring et al., 2009; but see also Schulte et al., 2010).

Based on this limited number of correlational studies, the link between visuoperceptive-related cerebral deficits and alcohol consumption thus appears very tenuous.

4.4 | Do visuoperceptive deficits recover with prolonged abstinence?

Finally, another key question pertains to the stability of visuoperceptive deficits, which have often been considered as persistent in time and thus resistant to long-term abstinence. Electrophysiological and neuroimaging studies assessing visuoperceptive recovery following abstinence are scarce and provide mixed evidence. While alterations of the P100 component could return to normal after 6 months of abstinence (Chan et al., 1986), other ERP changes, and especially the reduced visual P3b amplitude, could still be observed after 6 years of abstinence (Fein & Chang, 2006). Considering the very small sample of abstinent individuals with SAUD whose P100 was examined and the fact that P3b disturbances can be considered as a vulnerability factor rather than a direct consequence of SAUD (Porjesz et al., 2005), little is known about the recovery of visuospatial functioning at the electrophysiological level.

Neuroimaging findings are more consistent and suggest that structural changes in posterior brain regions could improve over time but generally remain impaired long after drinking cessation. Regarding gray matter volumes, persistent shrinkage has been documented within the occipital and parietal cortex of individuals with SAUD abstinent from 1 month (Rando et al., 2011) up to more than 6 years (Fein et al., 2009). Recovery of occipital and parietal gray matter does not follow a linear pattern over time, with volumes increases being greater over 1 week to 1 month than from 1 month to 7.5 months of abstinence (Durazzo et al., 2015). Consistently, whole-brain tissue volumes have been found to increase, and concomitant cerebrospinal fluid to decrease, more quickly during the first month of abstinence than during the following 5–8 months (Gazdzinski et al., 2005). In this study, changes mainly occurred around the lateral, third and fourth ventricles, at the limit with frontal, parietal and superior temporal lobes, as well as in the cerebellum, pons, and hippocampi, and were most rapid in individuals with SAUD who had the strongest brain shrinkage and highest drinking severity at baseline. Variations nevertheless exist in recovery and changes in some cerebral regions might be more reversible than others. For instance, a recent study documented gray matter volume increase in the parietal lobe of individuals with SAUD between 1 and 4 weeks of abstinence, but could not spot similar improvement in the occipital lobe (Durazzo et al., 2017). Contrary to their expectations, the authors were also unable to find any difference in parietal and occipital volume changes between individuals who remained abstinent for 1 month and those who resumed drinking within that same period. They proposed that these inconsistencies across studies might be due to the lack of specificity of the cerebral regions explored and emphasized the need to target specific components of well-established neural circuitries. As discussed in Sections 3.1 and 3.2, the cuneus could be of particular interest since its volume and activity have been linked to relapse. Based on signs of persistent gray matter volume reduction in the cuneus and precuneus of heroin-dependent individuals who had been free of drugs for multiple years (Wang et al., 2016), it has been proposed that gray matter volumes in the cuneus might not return to normal levels as easily as other brain regions (Wang, Fan, et al., 2018).

TABLE 1 Most robust visuoperceptive-related cerebral changes identified in individuals with SAUD

Findings	Implications	Supporting studies
Reduced and/or delayed P1 and N1	Blockage and/or delay of conduction in the optic nerve; Impaired inhibitory activity from the thalamus to the visual cortex; Alteration in the striate cortex, the dorsal extrastriate cortex of the middle occipital gyrus or the ventral extrastriate cortex of the fusiform gyrus	Ahmed and Hines (1983), Cadaveira et al. (1991), Chan et al. (1986), Cosi et al. (1986), Dustman et al. (1979), Glenn et al. (1996); Maurage et al. (2007, 2008), Nazliel et al. (2007), Olbrich et al. (2000), Parsons et al. (1990), Patterson et al. (1987), Porjesz, Begleiter, and Garozzo (1980), Porjesz, Begleiter, and Samuelly (1980), Porjesz and Begleiter (1979, 1982a); Williams (1984)
Changes in the gamma band (28.5–50 Hz)	Alteration of integrative functions (e.g., "feature binding"); Impaired top-down interactions with frontal regions during sensory processing	Padmanabhapillai et al. (2006), Padma Shri and Sriraam (2016, 2017), Sion et al. (2020)
Gray matter volume reduction in the occipital lobe, including the cuneus and lingual gyri, as well as in the fusiform gyrus	V1 damage; Potential consequences for visual information filtering, perception of global forms, and high-level visual recognition and categorization; Association with relapse	Durazzo et al. (2015, 2017), Fein et al. (2009), Fortier et al. (2011), Mackey et al. (2019), Rando et al. (2011), Uhlmann et al. (2018), Wang, Fan, et al. (2018), Zhu et al. (2018)
White matter impairment within the occipital cortex and the fronto-occipital fasciculus	Reduced intra-visual connections and cortico-cortical top-down connections; Association with behavioral figure copy scores	Bagga, Sharma, et al. (2014), Pandey et al. (2018), Rosenbloom et al. (2009), Schmahmann and Pandya (2007)
Reduced connectivity in the cuneus, precuneus, lingual, fusiform, mid-occipital and parieto-occipital gyri, and between prefrontal and occipital/parietal regions	Alteration of functional hubs of the ventral stream involved in the processing of basic visual properties (e.g., color, shape, size); Lack of communication between multi-sensory modalities brain areas; Association with relapse	Chen et al. (2019), Karamzadeh et al. (2015), Liu et al. (2018), Pfefferbaum, Desmond, et al. (2001), Wang, Fan, et al. (2018), Wang, Zhao, et al. (2018), Zehra et al. (2019)

By contrast to gray matter volumes, parietal (but not occipital) white matter volumes tend to increase linearly over the course of maintained abstinence, with proportionate increases between 1 week, 1 month, and 7.5 months (Durazzo et al., 2015). The increase in fractional anisotropy in occipital regions is also accompanied by a decrease in mean diffusivity during the first month of abstinence (Gazdzinski et al., 2010). Changes in occipital and parietal white matter volumes have, however, less frequently been reported in SAUD (Durazzo et al., 2017), and changes in mean diffusivity and fractional anisotropy can be observed in the absence of any detectable white matter atrophy (Gazdzinski et al., 2010). So far, no study has been conducted to explore how the integrity of long associative white matter fibers such as the fronto-occipital fasciculus evolves and potentially recovers with abstinence, despite the central role that these fiber tracks could play in both bottom-up and top-down visual processes (see Section 3.1).

Altogether, regional lobar white matter volumes could thus increase in a rather linear fashion, whereas gains in gray matter volumes could be significantly greater over the first weeks of abstinence than the following months and years (Durazzo et al., 2015). Residual cerebral changes in visual-related areas remain, however, noticeable despite prolonged abstinence, suggesting potential, but not systematic, long-term behavioral manifestations. The paucity of studies considering behavioral performances makes it hard to conclude. As with the impact of SAUD duration, researchers generally report a lack of significant correlation between abstinence length and cerebral correlates (Fein et al., 2009; Sullivan, 2003). Moreover, complex profiles of correlations are sometimes described, such as scenarios in which duration of abstinence correlates with the magnitude of structural brain changes (e.g., parietal gray matter shrinkage) but not behavioral scores (e.g., score at the WAIS Block Design and Microcog Clocks and Tic Tac subtests) despite a significant link between these structural changes and behavioral performance (Fein et al., 2009). This suggests that we might sometimes overlook part of the underlying deleterious processes, or that deficits might not especially express similarly across time at the cerebral and behavioral level. In this regard, it is also especially likely that compensatory mechanisms operate in parallel (see Sections 3.1 and 3.2).

5 | SUMMARY OF THE FINDINGS

As a whole, neuroscience studies promote an explanation of visuoperceptive changes in SAUD that involves the dysregulation of an extensive visual network. Results from this review support the idea that both the ventral and dorsal visual pathways, broadly associated with temporal and parietal regions, as well as their communication channels with distant nodes of the visual system, might be impaired.

To sum up (see Table 1 for a summary of the most robust findings), electrophysiological studies demonstrate the presence of visual disturbances very early on in the cerebral processing continuum. Changes in VEPs and perceptual ERPs are thought to reflect potential modifications at the level of the retina and/or the optic nerve as well as cortical damage. Neuroimaging work shows that these changes are accompanied by structural and functional modifications in various visual-related areas located in the occipital lobe, including the cuneus, as well as in the temporal and parietal cortex. In line with recent studies exploring EROs, connectivity analyses also reveal reduced communication within occipital regions and between occipital and long-distant areas, among which frontal regions. Finally, results from molecular imagery complement these outcomes by showing that part of this reduced connectivity might be due to specific occipital metabolic changes.

The association between visuoperceptive changes and frontal damage, and especially disrupted occipito-frontal connectivity, is probably one of the key results of this review. Indeed, not only does this finding allows to better understand how deficits in very distinct and distant brain structures influence visuoperception, but it also centrally reconnects with the new models of vision that promote tights links between vision and other higher-level cerebral networks. In line with these models, this review disclosed evidence that the integrity of the fronto-occipital fasciculus is associated with the visuoperceptive behavioral performances of individuals with SAUD. It also stressed that recordings of reduced early EEG gamma power could be indicative of degraded top-down interactions with frontal regions during sensory processing. The importance of such long-distance connections is also supported by the fact that: (1) connectivity changes observed in areas surrounding the parieto-occipital sulcus, as well as different ventral visual pathway hubs and prefrontal regions, are linked to relapse; (2) the same functional changes allow to efficiently distinguish individuals with SAUD from healthy controls. In other words, it might no longer be relevant to address the visuoperceptive changes of individuals with SAUD in terms of either low- or high-level deficits, but rather dynamic ones. Likewise, they do not correspond to spatially circumscribed cerebral alterations. Both basic perceptual and more evolved executive deficits most probably interact, potentially very early on, so that individuals with SAUD might especially struggle when the bottom-up and top-down connections between anterior and posterior cerebral regions are disrupted (see Figure 2).

In keeping with the idea of visuoperception as a dynamic process, we also reviewed fMRI studies suggesting that individuals with SAUD could rely on relatively "high-level" cognitive strategies and circuitries to counteract their rather "low-level" sensory-perceptive deficits. Such compensatory processes often rely on the close interplay between anterior and posterior brain regions and could also operate, more or less effectively, within the visual network. Indeed, we showed that individuals with SAUD could recruit more ventral visual regions to overcome potential dorsal alterations and achieve a normal level of performance. Importantly, the compensatory nature of these changes in cerebral activity and connectivity is acknowledged by their association with improved and potentially normalized

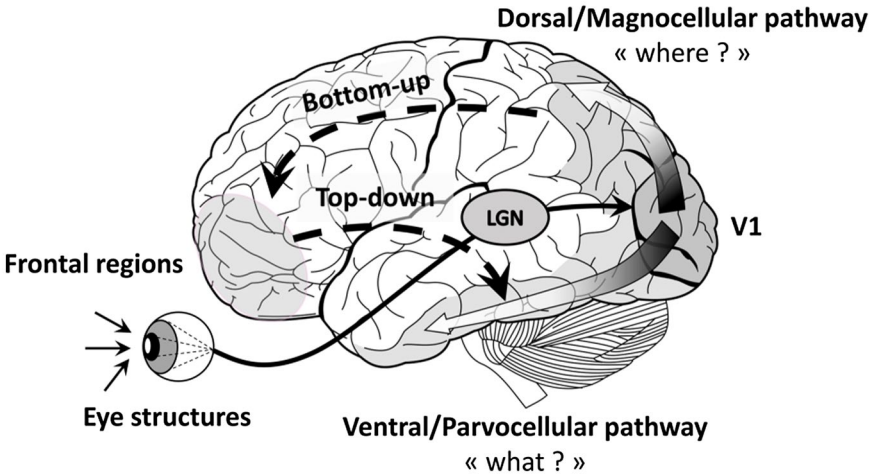

FIGURE 2 Schematic representation of the classical retino-geniculate-striate visual pathway. Visual information arriving at the retina is sent to the primary visual cortex (V1) through the lateral geniculate nucleus (LGN) before projecting either to the dorsal or ventral visual streams. Besides, visual information, especially of dorsal origin, is sent in a bottom-up fashion to frontal regions. These higher-order areas, including the orbitofrontal cortex, ensure efficient top-down control over visual processing, notably to promote robust visual recognition within the ventral pathway. In SAUD, the processing of incoming visual information could be disrupted at different stages of the retino-geniculate-striate visual pathways and is especially characterized by reduced connectivity between occipital and frontal regions, implying weakened bottom-up and top-down mechanisms (dotted arrows)

visuoperceptive performances. These compensatory mechanisms may capture part of the specificity of neural changes associated with SAUD and may help to spot their singularity compared to cerebral changes occurring in other populations, among which the aging population. Indeed, similar changes in connectivity generally mirror dysfunctional cerebral diffusion rather than functional compensation in elderly people, in whom they are associated with lower rather than higher visuoperceptive performances.

Besides describing the cerebral changes associated with visuoperception in SAUD, another objective of this review was to examine how they vary according to demographic and alcohol-related inter-individual factors, as well as how they evolve through time with maintained abstinence. We found very inconsistent results regarding gender differences, and especially the hypothesis that women may suffer more severe visuoperceptive-related cerebral alterations, so that the opposite pattern might be as plausible. Concerning age, fMRI results revealed that changes in the visual network do not reflect the same underlying processes in individuals with SAUD and aging healthy controls despite sensibly similar structural profiles. Even though the proposal of premature aging of cognitive functioning in SAUD continues to be adopted by some researchers, this parallel might not apply to visuoperception. The fact that individuals with SAUD probably do not experience a real "premature aging" of visuoperception does not, however, preclude any influence of age on the severity of the deficits. Finally, the limited number of studies exploring the relationship between alcohol and visuoperception found little link between signs of cerebral damage and alcohol-related consumption indices. While this interrogates the role of alcohol in the development of these changes, a series of biases probably influence the ability to properly grasp this relationship. While no consensus emerged regarding the influence of age, gender, and alcohol consumption, our results appear to agree upon the idea that visuoperception could constitute one of the most long-lasting domains of deficit in SAUD. Electrophysiological studies exploring recovery at early perceptual stages are very scarce but suggest that the P100 component could normalize within 6 months of abstinence. By contrast, neuroimaging findings more consistently show that structural changes in posterior brain regions can improve over time but remain impaired long after drinking cessation. Indeed, lower gray matter occipital and parietal volumes have been documented after several months (and sometimes even years) of abstinence. How compensatory mechanisms may act in parallel to substantial functional and structural recovery, and whether distinct cerebral areas may be differentially sensitive to the neurotoxic effects of alcohol, remains, however, unknown.

Altogether, visuoperceptive deficits in SAUD are probably of multiple and widespread origins, implicating both low-level sensory and high-level cognitive cerebral circuits. By offering a more integrated picture of the deficits and binding two central domains of impairments in SAUD, namely visuoperceptive and frontal-executive deficits, neuroscience studies allow us to better apprehend their joint role in the pathology and converge toward the need to develop a more complex model of visuo-cognitive interactions in SAUD. Such a model would help, for instance, to better understand how the integrity of visual regions, which has long been neglected, can be directly or indirectly linked to relapse. Current research also tends to discuss the consequences of visuoperceptive deficits on the allocation of one's limited cognitive resources and argues that individuals with SAUD's attempt to exert executive control to compensate for their visuoperceptive deficits might leave little attentional resources available to face additional demands (Fama et al., 2004; Wang, Fan, et al., 2018). As a result, they might not be able to efficiently monitor their behavior when facing highly motivational visual stimuli, typically alcohol-related cues, which may contribute to early relapse. However, to date, the lack of systematic examination of each distinct component of the visual system, and consideration of the successive cerebral processing steps, makes it difficult to formulate more precise proposals and draw a proper spatiotemporal map of the deficits (Creupelandt et al., 2019). We thus hope to renew researchers' interest in visuoperception in order for future studies to reduce the gap between the SAUD literature and current dynamic models of vision.

6 | LIMITATIONS AND FUTURE PERSPECTIVES

A series of limitations related to the available studies constrain the conclusions and implications of this review, among which the large heterogeneity of diagnostic criteria for SAUD and degree of screening for comorbidities, but also types of measures and techniques used. The main general limitations inherent to the evaluation of SAUD also apply: Besides the inevitable presence but little consideration of inter-individual differences in age, gender, or family history across studies, the lack of longitudinal measures often hampers the examination of the causal role of alcohol in the deficits and limits the possibility to control for any deficit before drinking onset. Considering the absence of objective monitoring of alcohol consumption, studies also rely on self-reported consumption data that suffer biases (Del Boca et al., 2014) and largely influence correlations. Besides, patients are often tested after different abstinence durations, which has significant implications for the nature of the observations, as distinct visuoperceptive properties and related cerebral regions might recover at various speeds. Depending on the time points selected, not every single impairment might be properly detected, not to mention the interactions with compensatory mechanisms that would require more in-depth qualitative analyses of the results. What is more, selecting treated samples of individuals with SAUD might limit the generalization of the results to the whole SAUD population. To limit these methodological issues and facilitate comparison across studies, future work should adopt a systematic definition of SAUD and consider the heterogeneity of profiles, especially since the variations in outcomes underlined in this review suggest that visuoperceptive deficits may only concern part of the SAUD population. To better capture inter-individual differences, studies should also: (i) explore gender differences while equating the number of drinks and duration of SAUD to dissociate actual

physiological differences from uneven consumption patterns; (ii) assess whether functional reorganization of visual networks in SAUD systematically reflects neural compensation in groups of individuals with SAUD of varying ages, or whether compensatory mechanisms may only concern younger individuals.

Neuroimaging and electrophysiological techniques are also subject to a series of specific flaws that limit our current understanding of visuoperception in SAUD. On the one hand, each method has its limitations, especially in terms of spatial and temporal resolution, so that diverse measures have to be collected and combined to get a clear picture of the deficits. On the other hand, and more importantly, how neuroimaging and electrophysiological data have been gathered and bind together so far about visuoperception in SAUD can also easily be criticized as very few neuroimaging studies have directly focused on precise visual-related regions. As a result, both structural and functional results are often based on unspecific investigations that did not directly investigate visual perception, further increasing the risk of biases. For instance, even though we focused on regions known to be associated with visuoperception, the ventral and dorsal visual streams also interact and potentially overlap with other cerebral networks, including attentional ones, making the distinction between perceptual and attentional processes difficult to draw. Moreover, results are not systematically linked to behavioral cues, and when they are, they are often associated with either very basic (e.g., passive viewing or oddball paradigm) or very multidetermined (e.g., copy and recall of a visual figure) cognitive tasks that do not target well defined visual processes and often comprise a higher-level executive and/or mnemonic component. Similarly, electrophysiological studies in SAUD often did not look at the early sensory activity and rather focused on higher-level cognitive processes, except for VEPs that are elicited by very standardized and invariant visual events rarely renewed or manipulated, and thereby provide only limited new information. The lack of correspondence between cerebral and behavioral measures raises two main issues. First, this necessarily constrains conclusions regarding impairments, as we cannot ascertain that neural differences necessarily reflect impairment per se. Unless there is a behavioral impact, it might be more cautious to refer to dysregulation or cerebral change. Second, very little is known about the integrity of specific visual structures and especially the two main visual pathways in SAUD, which would deserve further examination. Investigating the structural and functional integrity of the ventral and dorsal visual pathways would be all the more relevant considering that the new models of vision ascribe distinct, yet complementary, roles to these two pathways and consider that visual impairment could have a significant impact on key adaptative behaviors (Bar, 2003, 2007, 2009; Bar et al., 2006; Barrett & Bar, 2009).

To trigger and measure specific visual activity, future studies should exploit the distinct low-level visual properties of each visual pathway through carefully controlled tasks. The ventral and dorsal pathways, generally considered of primary parvocellular and magnocellular origins, differ in terms of spatial and temporal sensitivity, as well as contrast (Livingstone & Hubel, 1988). Spatial frequencies refer to a basic property of the visual system which analyses any visual input as a series of variations in luminance occurring at various frequencies across space (Boothe, 2002; Goldstein, 2010). Low spatial frequencies (LSF) correspond to large-scale luminance variations and allow the detection of coarse visual information (Loftus & Harley, 2004). High spatial frequencies (HSF), on the opposite, refer to small-scale luminance variations and enable the processing of small details and sharp edges (Loftus & Harley, 2004). LSF are preferentially mediated by magnocellular cells and parvocellular cells are preferentially tuned to HSF, so that the manipulation of the spatial frequency content of a visual stimulus can influence the pathway processing it (Boothe, 2002; Goldstein, 2010). In terms of temporal frequencies, magnocellular cells are best suited to process rapidly flickering stimuli as well as changes in speed and motion parameters compared to parvocellular ones. Depending on the spatial frequency content of a stimulus, and its static or dynamic presentation, it is thus possible to bias its processing toward one or the other pathway. Likewise, it is possible to manipulate contrast, that is, the magnitude of the difference between the alternating variations of luminance (i.e., bright and dark bars), to promote parvocellular or magnocellular processing (Alexander et al., 2004; Merigan & Maunsell, 1993; Pokorny & Smith, 1997; Pokorny, 2011). Examples of tasks relying on these basic properties include thresholds measurement for spatial frequencies, contrast, and temporal frequencies using sinusoidal gratings (i.e., Gabor patches) or detection, scanning and judgments of filtered stimuli composed of a specific spatial frequency range displayed at different contrast and temporal levels (see Creupelandt et al., 2019). From a neuroscience perspective, such tasks will help to explore early perceptual ERPs more systematically, especially since magnocellular and parvocellular cells may contribute differently to their occurrence and appearance. For instance, it has been shown that the early P1 could be primarily elicited by magnocellular and mixed magnocellular/parvocellular stimuli, whereas the somewhat later N1 could be primarily elicited by parvocellular and magnocellular/parvocellular mixed stimuli (Schechter et al., 2006). Individuals with SAUD might show a different pattern of cerebral activation than healthy controls in response to ventral and dorsal biased visual information. Coarse-to-fine visual sequences, namely series of SF-filtered pictures that initially contain only LSF information and progressively shift toward more HSF information, could especially prove useful to spot changes in the time course of visual processing. In healthy controls, coarse-to-fine visual sequences generally increase activity in the orbitofrontal cortex and temporo-parietal areas in response to LSF cues before enhancing HSF analysis in the primary visual cortex and more ventral regions (Peyrin et al., 2010). In individuals with SAUD, this efficient information extraction mechanism might be dysregulated due to reduced intra-visual and fronto-occipital connectivity.

Finally, exploring visuoperception has clinical implications as individuals with SAUD may initially better benefit from remediation focusing on the improvement of their visual skills rather than remediation immediately centered on more complex cognitive tasks. In fact, from the moment practitioners suspect impaired

visuoperceptive analysis, it is likely that individuals with SAUD may base their judgment on degraded and incomplete visual information. As a result, targeting high-level cognitive deficits might not be sufficient. Introducing a visuoperceptive rehabilitation program before the training of executive functions may have a positive impact on the first sensory stages of the cognitive continuum and promote, in turn, better recovery of the following visual-related attentional, memory and decisional cognitive steps. Such a proposal is notably supported by the fact that: (1) individuals with SAUD can rely on compensatory strategies and alternative cerebral circuitries to put up with their difficulties, implying that they still display recovery opportunities (Fama et al., 2004; Müller-Oehring et al., 2009; Pfefferbaum, Desmond, et al., 2001; Tapert et al., 2001; Zhang et al., 1997); (2) evidence of efficient visuoperceptive learning of basic visual aptitudes (e.g., motion perception, Zhang & Yang, 2014; spatial frequency processing, Peters et al., 2017; contrast sensitivity, Deveau et al., 2014) exists in healthy controls. Exploring the role of visuoperception would also provide interesting clues on how to improve already existing training programs using high-level stimuli of direct relevance for individuals with SAUD, such as alcohol-related cues or emotional faces. It would be of interest to assess the benefit of using visually modified stimuli such as LSF or HSF filtered faces in such remediation tools. Centrally, this visuoperceptive rehabilitation program would have to be scheduled on an individual basis, considering the profile of impairments that would have been previously identified through the above-mentioned low-level visuoperceptive tasks. Since all individuals with SAUD might not present with visuoperceptive impairments, the priority in cognitive remediation must be individualized. Visuoperceptive learning should be part of a well-considered program engaging other cognitive processes and skills susceptible to influence individuals with SAUD's training and progress, among which, in particular, top-down attentional control (see Byers & Serences, 2012 for a review).

7 | CONCLUSION

This paper offers the first comprehensive review of the cerebral correlates of visuoperceptive alterations in SAUD and provides strong evidence of structural and functional damage in various brain regions involved in visual perception. It centrally shows that the cerebral changes are very widespread and involve not only damage of the primary visual cortex and other occipital structures, but also impaired fronto-occipital connectivity, leading to a weakened bottom-up sensory processing and top-down visual control. While some neural markers of the deficits last for years, individuals with SAUD could experience partial recovery with prolonged abstinence and could rely on alternative compensatory neuronal circuitries within the visual system and beyond. Although these deficits are thus well established, very little effort has been directed toward their integration so that no satisfactory and up-to-date anatomo-functional model of visuoperception in SAUD currently exists. Future research should therefore adopt a more defined research framework, and notably explore the ventral and dorsal visual pathways more thoroughly, to unveil how the seemingly scattered cerebral deficits of individuals with SAUD might interact. Considering the omnipresence of vision, exploring and training visuoperceptive abilities in SAUD could constitute an additional and important gateway toward a better understanding and therapeutic care of this condition.

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CONFLICT OF INTEREST

The authors declare that they have none.

AUTHOR CONTRIBUTIONS

Conceptualization, C.C., P.M., and F.D.; *Methodology*, C.C., P.M., and F.D.; *Investigation*, C.C.; *Validation*, C.C.; *Writing – Original Draft*, C.C.; *Supervision*, F.D. and P.M.; *Writing – Review and Editing*, F.D. and P.M.; *Project Administration*, P.M.

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