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REVIEW

Visuoperceptive Impairments in Severe Alcohol Use Disorder: A Critical Review of Behavioral Studies

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Abstract

The present literature review is aimed at offering a comprehensive and critical view of behavioral data collected during the past seventy years concerning visuoperception in severe alcohol use disorders (AUD). To pave the way for a renewal of research and clinical approaches in this very little understood field, this paper (1) provides a critical review of previous behavioral studies exploring visuoperceptive processing in severe AUD, (2) identifies the alcohol-related parameters and demographic factors that influence the deficits, and (3) addresses the limitations of this literature and their implications for current clinical strategies. By doing so, this review highlights the presence of visuoperceptive deficits but also shows how the lack of in-depth studies exploring the visual system in this clinical population results in the current absence of integration of these deficits in the dominant models of vision. Given the predominance of vision in everyday life, we stress the need to better delineate the extent, the specificity, and the actual implications of the deficits for severe AUD.

Keywords Alcohol-dependence · Vision · Visual perception · Visuospatial

Introduction

Severe alcohol use disorder (AUD) represents a major public health concern worldwide, constituting one of the most prevalent causes of death and being involved in more than 200 diseases (Wittchen et al., 2011). Previously labeled as alcoholism (DSM-III; American Psychiatric Association [APA], 1980) or alcohol-dependence (DSM-IV; APA, 1994), severe AUD (DSM-5; APA, 2013) is centrally characterized by a compulsion to seek and drink alcohol, together with a loss of control over consumption and an inability to reduce drinking, despite the negative consequences. Severe AUD is associated with a broad range of cognitive deficits (Crowe et al., 2019; Oscar-Berman et al., 2014; Stavro et al., 2013) as well as anatomical and functional brain impairments (Sullivan et al., 2010; Zahr, 2014), leading to a continuum ranging from mild cognitive alterations to more severe deficits, and ultimately to alcohol-related neurological complications, including dementia and Korsakoff syndrome (Brion et al., 2014; Pitel et al., 2013). Among these cognitive deficits, visuospatial and visuoperceptive disturbances [1] are frequently reported and often considered as persistent over time. However, this topic has mainly been explored during

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[1] This paper will focus on both visuoperceptive and visuospatial processes, to address both (1) visual recognition processes based on the analysis of basic features such as form, pattern, and color, mostly linked to the ventral visual pathway and temporal cortex, also known as the "what" system, (2) visual-spatial analysis, generally associated with the dorsal visual pathway and parietal cortex, and often called the "where" system (Kreutzer et al., 2017). The term "visuoperception" will be used to refer to visual perception in general, and not specifically to ventral visual processes as little distinction has been made so far between the two visual streams in the severe AUD literature.

the seventies to the nineties of the 20th Century. Since then, far fewer studies have focused on vision, introducing a gap in the literature and increasing the discrepancy between our currently evolving understanding of visuoperceptive processing in healthy individuals and the limited data available in severe AUD. As a result, little is known about the specificity and the implications of visuoperceptive deficits for individuals with severe AUD.

The high relapse rates in severe AUD suggest that our current theoretical models, and associated clinical rehabilitation programs, are insufficient. We propose that the lack of recent studies exploring visual deficits in severe AUD, resulting in the absence of integration of these deficits in the dominant models of vision, hampers the development of a comprehensive description of the disease, with substantial consequences to clinical care. Indeed, evidence stemming from different lines of research (e.g., studies on acute alcohol consumption; new models of the visual system in healthy individuals) suggests that the presence of visuoperceptive deficits should not be overlooked in severe AUD, as (1) alcohol consumption has a deleterious effect on visual performances, and (2) changes in visual perception could influence not only basic perceptual processes but also higher-level cognitive functions, consistent with the current view of vision as a dynamic process that can act in a top-down fashion and need not always precede cognitive (i.e., attention, memory) processes (e.g., Creupelandt et al., 2019; D'Hondt et al., 2014a, 2014b).

While little research has investigated the long-lasting visuoperceptive abilities of individuals with AUD, recent studies focusing on acute alcohol consumption have shown that alcohol can slow down visual search for peripheral targets (Hoyer et al., 2007), restrict visual scanning to central and salient image features (Harvey, 2014, 2016) and impair perceptual judgment (Friedman et al., 2011). Besides, evidence suggests subtle changes in visual contrast sensitivity (Andre et al., 1994; Cavalcanti-Galdino et al., 2014; Pearson & Timney, 1998; Zhuang et al., 2012), motion processing (Andre et al., 1994), short-term visual memory (Schweizer et al., 2006) and depth perception (Nawrot et al., 2004). Even though the effects of acute and chronic alcohol consumption cannot be directly compared, these studies show that alcohol impacts visual processing. People with AUD experience acute alcohol intoxication, often daily, and studies conducted in the '70s and '80s have suggested visual deficits in this population. Therefore, there is a need to explore the presence of long-lasting visual deficits related to severe AUD more systematically.

In this context, our main aim is to provide the first comprehensive and critical review of previous behavioral studies comparing visuoperceptive processing in abstinent individuals with severe AUD and healthy controls. To do so, this paper recalls the context of research on visuoperception in severe AUD and tracks its unfolding while distinguishing the precise visuoperceptive findings that punctuated the field from the more general results stemming from broader cognitive testings. It discusses the role that inter-individual factors (alcohol consumption characteristics, demographic variables, and abstinence) may play in visuoperceptive difficulties. Special attention is also given to the status of visuoperception in the overall cognitive profile of individuals with severe AUD, in an attempt to propose a first integrated description of the deficits. Finally, central limitations and several potential associated research perspectives are addressed, as well as the clinical implications for the field.

Methods

Research Question, Articles Identification and Selection Procedure

We specified our research question and inclusion criteria through the PICOS procedure (Population, Intervention, Comparator, Outcome, Setting; Liberati et al., 2009). Regarding the population, we considered studies including human participants defined as alcoholics (DSM-III; APA, 1980), alcohol-dependent (DSM-III-R, DSM-IV, DSM-IV-TR; APA, 1987, 1994, 2000; ICD-10, ICD-11; World Health Organization, 2004, 2019), or suffering from severe alcohol use disorders (DSM-5; APA, 2013). Considering the number of old studies, we also included papers referring to "alcoholism" without further diagnostic criteria. However, we excluded studies focusing on cirrhotic patients or patients with other major comorbid organic diseases. Concerning intervention and exposure, we included studies exploring the long-term effects of chronic alcohol consumption, but not its acute effects. Therefore, participants had to be abstinent at testing time. Regarding the comparator, we included studies offering a reliable comparison between an experimental alcohol-related group and a healthy light social drinkers or non-drinkers control group. Articles that did not include a control group but referred to norms from the general population were considered. The outcome was focused on studies that proposed at least one visuoperceptive-related measure as a dependent variable. Studies had to explore visuoperception with abstract or low-level visual materials, except for some tasks or subtests using pictures of everyday objects. Papers explicitly exploring the processing of more complex visual stimuli (e.g., emotional faces, natural scenes, alcohol-related stimuli) were not included as they were focused on higher-level decision-making or emotional judgment processes, and thus exceeded our scope. Finally, regarding the setting, we included studies related to any design based on comparisons between groups or experimental conditions (i.e., interventional, observational,

cross-sectional), and thus excluded single-case or case series studies, as well as papers not reporting experimental data (i.e., review, meta-analysis, reply, commentary, erratum, conference proceedings).

We performed a literature review via the PubMed database 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 articles included in this review were peer-reviewed articles written in English and published between January 1950 and June 2019. A second search round was performed upon abstract reading, and bibliographic references were reviewed to identify additional publications.

Conducting a strictly systematic review was not appropriate for our purpose because visuoperceptive disorders have rarely been at the heart of a specific research theme in severe AUD and have, therefore, not been thoroughly discussed as the main finding in most studies. A classical systematic search strategy would not have made it possible to identify all the relevant papers, given that the results concerning visuoperception are not necessarily described in the abstract. Such a methodology would thus have prevented us from retracing the history and complexity of the visuoperceptive results obtained in very different studies. Accordingly, we decided to adopt a more flexible method allowing for additional inclusions based on the type of neuropsychological tests used and also targeted papers that addressed visuoperception in their discussion. This strategy explains why a large part of the papers included in this review was found by a careful study of the bibliographic references of the most relevant papers. It was impossible to carry out a meta-analysis due to the heterogeneity of the studies selected, notably regarding methodological rigor and experimental tasks. Hence, a critical narrative review was the most appropriate format.

Methodological Quality Assessment

The methodological quality of each study was assessed via an adapted version of the "quality assessment tool for observational cohort and cross-sectional studies", developed by the National Heart, Lung and Blood Institute (NHLBI, 2014). This 14-item scale is appropriate for the cross-sectional studies considered here. We made minor modifications to focus the quality assessment on the visuoperceptive content of each paper, as many papers actually had a broader scope and did not necessarily focus on visuoperception only. Accordingly, the resulting quality assessment rating does not generalize to the whole paper but does reflect the behavioral visuoperceptive outcomes. More precisely, we slightly adapted the first item initially focused on the clarity of the main objective of the paper to instead evaluate the presence of any visuoperceptive-related explicit objective. We also split in two the content of items related to participants' selection (items 4a-b) and statistical analyses (items 5a-b) and added two complementary sub-items checking the presence of any visual screening before testing (item 4c) and complementary exposure measures (item 10b). This procedure resulted in a final methodological assessment scale composed of 18 items with a binary answer (Yes/No), with a maximum score of 18. Supplementary 1 Table 1 reports the score obtained by each study on each item, as well as a global score (i.e., number of items with a "Yes" answer divided by 18).

Data Extraction and Synthesis

A systematic data extraction procedure determined the main characteristics of each paper regarding six categories (adapted from the PICOS procedure): (1) Purpose (main objective, primary focus on visuoperception or not); (2) Population (sample size, age, gender ratio, medication, exclusion criteria); (3) Exposures (psychiatric diagnosis, alcohol-consumption measures, comorbidities); (4) Comparator (control group sample size and characteristics, matching variables); (5) Experimental design (processes measured, tasks and stimuli); (6) Outcomes (main results, reported limitations, key conclusions). Supplementary 1 Table 1 provides a comprehensive synthesis of the data extracted from each study regarding visuoperceptive processes. As for the methodological quality assessment scale, this synthesis focuses on the visuoperceptive content of each paper. The "key conclusions" section, however, allows the reader to get a slightly more general idea of the overall conclusion of the paper when relevant to the purpose of the present review.

Results and Discussion

The initial search yielded 1064 potentially eligible articles. Relevance screening led to the exclusion of 429 records based on the criteria of date (before 1950), language (not in English), human research (animal), and age (not adults). The remaining 635 records were assessed for eligibility based on the abstract and full-text, and 77 papers were eligible according to the inclusion criteria mentioned above. We also examined the references of the most comprehensive studies, identifying 71 additional articles. Following this search strategy (Fig. 1), 148 articles were included in the review

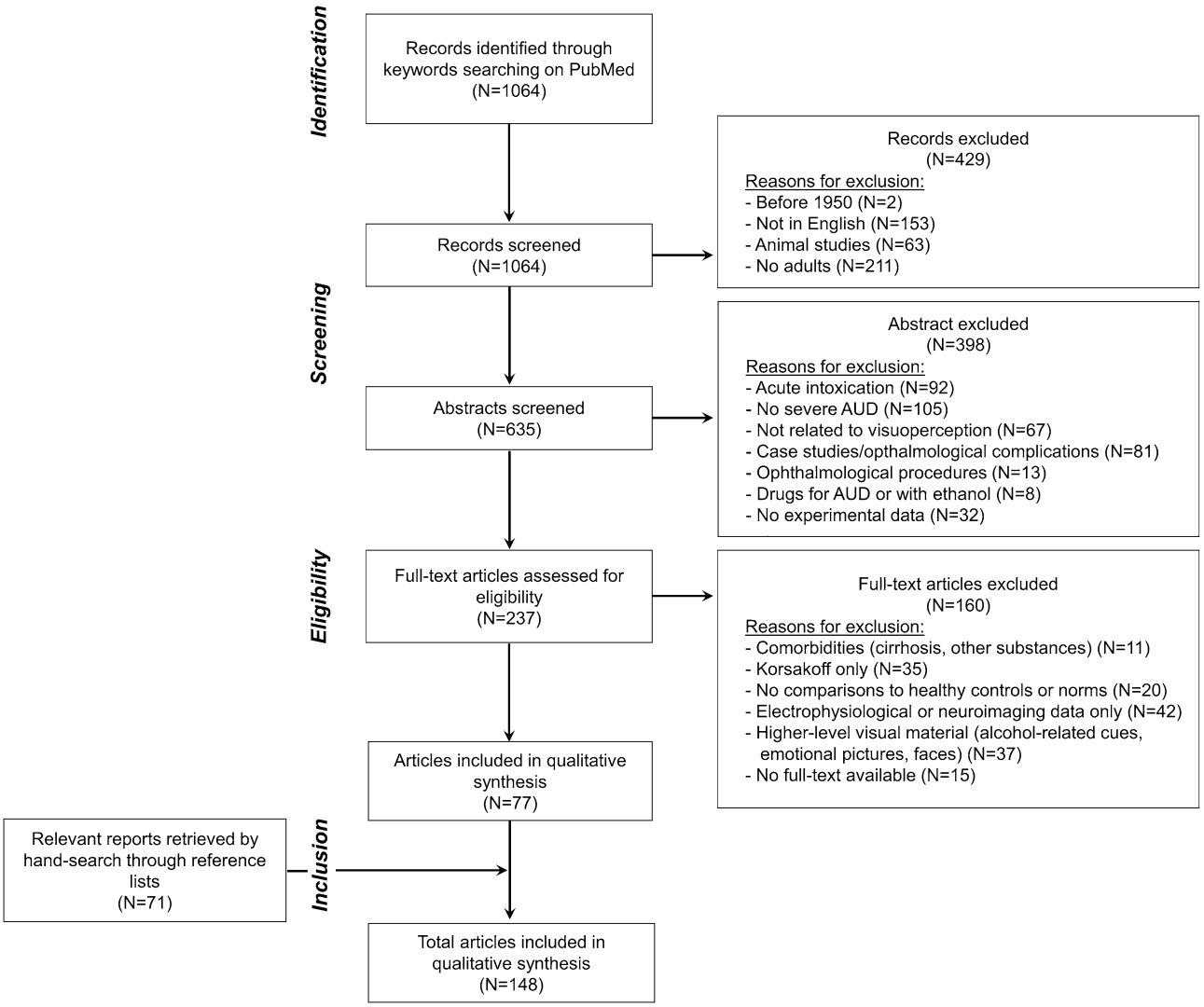

Fig. 1 Flowchart describing the research strategy and papers included

(Supplementary 2 Table 1). All papers are cross-sectional, and 21 include a test–retest measure. Figure 2 shows their distribution across time.

Brief Historical Background

Visuoperception in severe AUD started driving researchers' interest in the '60-'70s, in line with the observation that individuals with severe AUD generally display higher verbal than nonverbal performances. This research notably led to the emergence of the "right-hemisphere hypothesis". Considering that visuospatial and verbal functions depend predominantly on the left and right hemispheres respectively, this theory posited that individuals with severe AUD present with selective damage to right-hemisphere functions (Ellis & Oscar-Berman, 1989). However, the first studies exploring this proposal with broad neuropsychological batteries (especially intellectual tests) obtained mixed results. While some did show stronger deficits for nonverbal abilities (Di Sclafani et al., 1995; Fitzhugh et al., 1960, 1965; Jones, 1971; Jones & Parsons, 1972; Obaldia et al., 1981), others found generalized cognitive impairments (Drake et al., 1990; Errico et al., 1992; Gudeman et al., 1977; Molina et al., 1994; Parsons et al., 1990a, b; Whipple et al., 1988). Studies exploring visuoperceptive memory (Beatty et al., 1996; Bowden et al., 1992; Errico et al., 2002; Mulhauser et al., 2018; Quaglino et al., 2015; Wilson et al., 1988) or comparing verbal and nonverbal memory (Acker et al., 1984; Adams et al., 1980; Butters et al., 1973; Cutting, 1978, 1979; Franceschi et al., 1984; Haxby et al., 1983; Hill, 1980; Leber et al., 1981; Mann et al., 1999; Nixon et al., 1987; Riege, 1977; Samuelson et al., 2006; Selby & Azrin, 1998; Zinn et al., 2004) also found inconsistent findings, even with tests designed to maintain a similar structure and level of

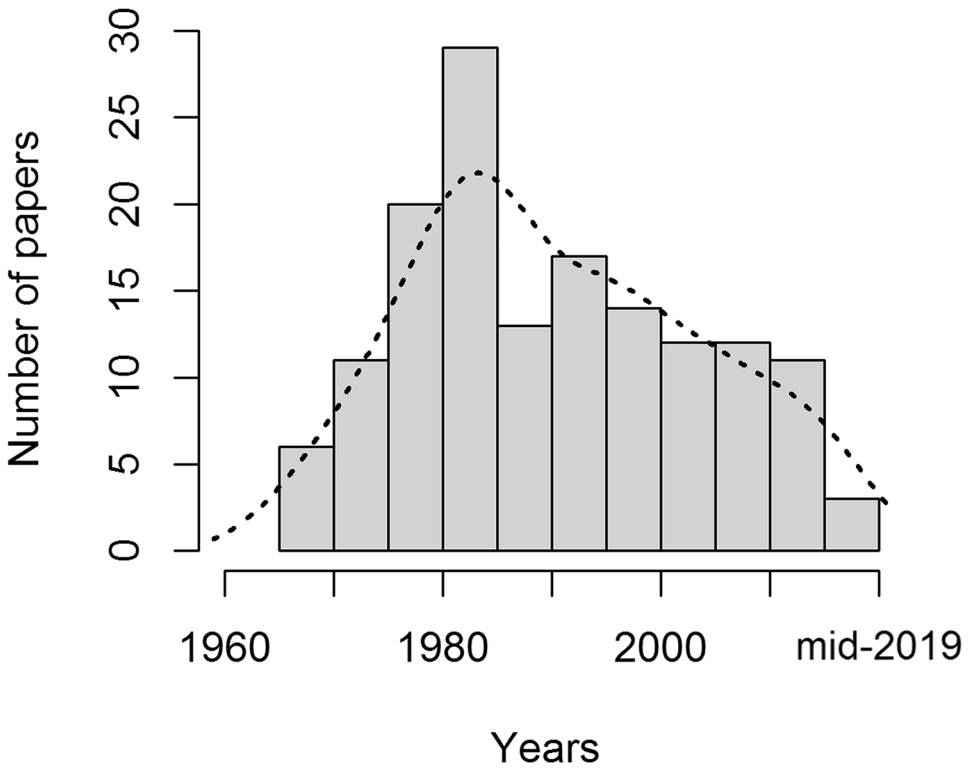

Fig. 2 Distribution over the years of the papers included in the review (1960–2019)

difficulty across verbal and visual modalities (Burnett et al., 1996; Butters et al., 1977; Miglioli et al., 1979; Shelton et al., 1984). Other researchers focused more directly on visuoperceptive skills and compared visual scanning (Beatty et al., 1996; Bertera & Parsons, 1978), visual detection thresholds (Kostandov et al., 1982; Oscar-Berman et al., 1973; Oscar-Berman & Weinstein, 1985; Reshchikova, 1985; Roberts & Bauer, 1993) or perceptual judgments (Tsagareli, 1995) in the right and left visual fields, but failed to evidence a systematic pattern of right cerebral lateralization of the deficits. As a whole, no clear-cut distinction could be made between right and left hemispheres functioning (Oscar-Berman & Schendan, 2000), and the right-hemisphere hypothesis is now considered outdated. Nevertheless, this early literature provided the first evidence of visuoperceptive deficits in severe AUD.

A few years later, researchers began investigating visuoperceptive functioning in the context of the premature aging hypothesis. Based on the observation that individuals with severe AUD and elderly people share neuropsychological similarities, among which visuoperceptive deficits, this theory suggested that the progressive decline expected in normal aging appears earlier in individuals with severe AUD (Ellis & Oscar-Berman, 1989; Ryan & Butters, 1984). Two alternative models were put forward (Evert & Oscar-Berman, 2001): the accelerated aging model postulated that severe AUD accelerates age-related cognitive decline regardless of the age at which excessive drinking begins (Eckardt et al., 1980; Noonberg et al., 1985); the increased vulnerability model posited that vulnerability to severe AUD-related brain damage is increased in older individuals, i.e., after the expected manifestations of aging began (Goldman et al., 1983; Jones & Parsons, 1971). The parallel between visuoperceptive functioning in normal aging and severe AUD has been investigated with a large variety of nonverbal neuropsychological tests, including broad intellectual (Blusewicz et al., 1977a, b; Hamblin et al., 1984) and memory tasks (Ryan & Butters, 1980), and appeared particularly marked for the tasks composing the "Brain-Age Quotient" (Reitan, 1967), namely the Tactual Performance Test, the Halstead Category Test, the Trail-Making Test and WAIS Block Design and Digit Symbol subtests (Hochla & Parsons, 1982), which all comprised a perceptual component. However, studies directly comparing groups of individuals with severe AUD and healthy controls of varying age never provided strong evidence in favor of either the accelerated aging or increased vulnerability model. Whereas some findings suggested that individuals with severe AUD could perform similarly to 5–20 years older healthy controls (Blusewicz et al., 1977a; Hochla & Parsons, 1982; Noonberg et al., 1985; Ryan & Butters, 1980) in the visuoperceptive domain, others studies did not replicate this finding (Blusewicz et al., 1977b; Brandt et al., 1983; Grant et al., 1979, 1984; Oscar-Berman & Bonner, 1985; Shelton et al., 1984). Higher performances were even

sometimes observed in old compared to young individuals with severe AUD, suggesting that the magnitude of severe AUD-related visuoperceptive decline decreased with aging (Evert & Oscar-Berman, 2001). Contradictory results were thus found for the premature aging proposal, which is considerably less investigated nowadays, at least from a behavioral perspective.

As a whole, studies investigating either the right hemisphere hypothesis or the premature aging hypothesis suffer a major limitation related to the use of multi-determined visuoperceptive tasks. Consequently, most initial studies exploring visuoperception in severe AUD did not properly isolate the low-level perceptual processes involved in the tasks from high-level cognitive abilities, leaving open the central question of the origin of the deficits.

Towards more Specific Visuoperceptive Measures

Given the little specificity of the early literature mentioned above, additional studies tried to better discern the presence of a defective visuoperceptive component. First, several studies addressed methodological shortfalls, notably by ruling out the risk of retinotopic confound through the use of different rather than identical stimuli in delayed matching-to-sample tasks (e.g., Zhang et al., 1997) or by selecting more basic low-level "meaningless" visual material in mnemonic tasks, thus preventing the use of verbal encoding strategies (e.g., Wegner et al., 2001).

Second, these studies distinguished between the influence of low-level perceptual and high-level memory or executive cognitive components, by showing that individuals with severe AUD exhibit slower visually guided movement but preserved simple reaction speed, indicative of selective impairment of perceptual motor-coordination rather than pure motor deficits (Vivian et al., 1973; Wilson et al., 1988). Researchers also assessed the impact of instructions emphasizing speed or accuracy and showed that these instructions had very little influence on lower perceptual skills, excluding a major role for impulsivity on differences in speed-accuracy thresholds in the deficits (Glenn & Parsons, 1991). Another set of studies checked that impaired performances on different visuoperceptive tasks usually found deficient in individuals with severe AUD, such as the WAIS Digit Symbol subtest (Acker et al., 1984; Brandt et al., 1983; Gudeman et al., 1977), were linked to an impoverished visuoperceptive analysis, rather than weaker psychomotor skills or reduced perceptual memory and learning capacities solely (Glosser et al., 1977; Kapur & Butters, 1977, but see Sassoon et al., 2007 for opposite results). These studies showed that the performance of individuals with severe AUD not only correlated with the ability to memorize the digit-symbol code but also with perceptual scores on the Embedded Figure Test (Witkin et al., 1971). They also underlined the use of different perceptual strategies in individuals with severe AUD compared to healthy controls (Kapur & Butters, 1977). These findings were complemented by studies showing that impoverished constructional skills and organizational strategies contributed to the weaker performances recorded in other classical visuoperceptive tasks based on the copy of drawings (e.g., Rey-Osterrieth Complex Figure, Beatty et al., 1996; Bender-Gestalt test, Snortum, 1965).

These results paved the way for the more specific examinations of perceptual analysis in severe AUD, which will be described in the section below. They also highlighted how tenuous the link between low-level and high-level skills could be. Indeed, organizational strategies can also draw on executive skills, and other studies suggested that visuoperceptive deficits could be sustained by a weakened ability to temporally organize and sustain goal-directed behaviors (i.e., "cognitive impersistence", Alterman et al., 1984), poor problem-solving skills (Dawson & Grant, 2000), inadequate planning (Munro et al., 2000), lower motor control, attention, and motivation (Ceccanti et al., 2015) or reduced incidental learning abilities (Bowden, 1988). The fact that some studies documented impaired recall of the Rey-Osterrieth Complex Figure despite satisfying initial copy performances also led some authors to doubt the presence of any visuoperceptive (rather than memory) deficits at all (Errico et al., 2002; Zinn et al., 2004). However, researchers also found recall scores on the Rey-Osterrieth Complex Figure to be strongly correlated with relapse rates, unlike other memory and executive scores (Desfosses et al., 2014), indicating that visuoperceptive deficits could influence drinking behavior, beyond executive or memory functioning alone (but see also the inconclusive results of Moriyama et al., 2002).

Third and finally, a few experiments also explored the presence of compensatory mechanisms in severe AUD. They assessed the use of differential cognitive strategies to understand how, for instance, one can exhibit normal perceptual learning of incomplete pictures despite basic visuoperceptive deficits in a simple embedded figure task (Fama et al., 2004, 2006). Results demonstrated that visuoperceptual analysis, explicit memory, and, to a lesser extent, frontal executive functioning predicted perceptual learning in healthy controls. Conversely, perceptual learning was predicted by explicit memory and frontal executive but not by visuoperceptual abilities in individuals with severe AUD (Fama et al., 2004). Individuals with severe AUD could thus display intact implicit memory for visual stimuli thanks to compensatory processes involving higher-order cognitive strategies. Consistently, studies exploring variations in cognitive structure and organization between individuals with severe AUD and healthy controls showed that the visuoperceptual and verbal factors were more related

in individuals with severe AUD than healthy controls, suggesting that individuals with severe AUD probably rely on their overall preserved verbal abilities to make up for their less efficient visuoperceptive skills (Ham & Parsons, 1997). Consistently, earlier studies have highlighted disrupted interactions between low-level bottom-up perceptual processes and higher-level top-down decisional processes in severe AUD. They found that individuals with severe AUD were less able than healthy controls to resolve the conflict induced by perceptual distortions like the binocular depth inversion illusion (Schneider et al., 1996a, b, 1998), or the presentation of negative photographs (reversing the locus of light and dark areas while leaving the edges unchanged, Nelson & Swartz, 1971).

Overall, those studies confirmed that visuoperceptive deficits cannot be explained by confounding factors or higher-level deficits only. They also suggested that individuals with severe AUD may mobilize other cognitive processes to partly compensate visuoperceptive deficits, which highlights the close connections that visuoperception shares with other cognitive systems. This idea, developed by the recent models of vision, will be discussed below. While refining early visuoperceptive findings, these studies still did not investigate specific visuoperceptive processes. More focused investigations have nevertheless punctuated the field, spanning across decades of research. These studies will be reviewed in the next section as they provide more compelling evidence of the precise visuoperceptive mechanisms disrupted in severe AUD.

Explorations of Distinct Visuoperceptive Processes

Mesopic and Color Vision

Studies exploring visual factors contributing to the higher car accident rates of individuals with severe AUD were among the first to assess more precise components of visuoperception in this population. Building on the fact that alcohol intoxication alters vision and that these alterations can have severe consequences when driving (National Highway Traffic Safety Administration, 2000), they explored basic visual characteristics and notably mesopic vision and color vision.

First, Reynolds (1979) could not find any difference between individuals with severe AUD and healthy controls in perception of depth and 3-dimensional structures (i.e., stereopsis), size of the visual field, position of the two visual axes when each eye looks at different objects in the distance (i.e., distance phorias), monocular eye movements (i.e., ductions) and the need to wear glasses. Only slight color vision impairments were identified. Likewise, Campbell et al. (1981) did not observe any group differences in mesopic visual acuity or dazzle recovery times (i.e., recovery of visual acuity after exposure to a bleaching light). As mesopic visual conditions are intermediate illumination conditions situated between the functioning level of rods and cones, no conclusion can be drawn on the integrity of each type of photoreceptor since they may have operated together. Both studies concluded overall that the impairments were insufficient to impair night driving ability.

Regarding color perception, Nelson et al. (1977) demonstrated deterioration of visual recognition of hues in individuals with severe AUD in a short-term memory task, reflecting selective color processing damage. This change in color vision (i.e., dyschromatopsia), possibly caused by macular degeneration, was replicated (Braun & Richer, 1993; Kapitany et al., 1993; Mergler et al., 1988; but see also Smith, 1972). Even though permanent damage to the papillomacular bundle (i.e., the nerve fibers carrying visual information from the macula) has been documented by retinoscopy in advanced forms of severe AUD (Plant & Perry, 1990), two studies more recently reported increased color discrimination thresholds in individuals with severe AUD without any eye anatomic alteration (de Oliveira Castro et al., 2009; Martins et al., 2019). The authors suggested that alcohol could lead to retinal ganglion cell loss progressing from preferential damage to parvocellular and koniocellular ganglion cells involved in color vision to depletion of magnocellular ganglion cells involved in achromatic vision. However, even though any history of degenerative, traumatic, toxic, or infectious diseases affecting the visual system constituted exclusion criteria, these results must be interpreted carefully as they could be due to metabolic diseases or nutritional deficits. Some color vision deficits have been linked to alcohol-related health issues such as cirrhosis or B-group vitamin deficiencies (Cruz-Coke, 1972; Orssaud et al., 2007; Ugarte et al., 1970). It has also been proposed (Varela et al., 1969, but see also Smith & Layden, 1971) that these deficits might be related to a higher prevalence of a genetic polymorphism related to color vision defects of the X-chromosomes in severe AUD. More recent findings also suggested that a specific brain-derived neurotrophic factor polymorphism might be implicated in the emergence of chromatic alterations (Serý et al., 2011).

Motion, Speed and Peripheral Visual Field

Several studies investigated motion perception, especially through speed and eccentricity measures. Wegner et al. (2001) found that individuals with severe AUD were impaired in the detection of motion coherence for slower, but not faster, speeds. Wegner and associates relied on an anatomical model of motion processing delineating two distinct streams, both related to a medial portion of the temporal cortex (MTC): a visual stream for slow motions,

projecting sequentially from the lateral geniculate nucleus (LGN) of the thalamus to the primary visual cortex (V1) and the prestriate cortex including the MTC; and a visual stream for fast motions, projecting in a more parallel and direct way to V1, MTC and the prestriate cortex (Ffytche et al., 1995). Given that these visual streams respectively derive from the parvocellular and magnocellular layers of the LGN, severe AUD could have preferentially damaged parvocellular layer related slow speeds. The same authors also documented deficits in a unidirectional but not bidirectional speed discrimination task, suggesting that impairments may have gone unnoticed in the most difficult speeded tasks. Careful examination of the results showed that healthy controls performed efficiently from the start in the unidirectional task but did show elevated thresholds for the bidirectional task when presented for the first time, which may mask group differences. Perceptive and memory deficits in motion processing were also reported by Chambers and Wilson (1968), who did not discuss the origin of the deficits.

Other researchers examined the flicker fusion phenomenon and recorded lower (i.e., worse) thresholds in severe AUD (Pillunat et al., 1985; Williams, 1984). The flicker fusion threshold measures the frequency at which an intermittent flashing stimulus is seen as entirely immobile, and thus assesses the temporal sensitivity of the visual system. While temporal properties are generally linked to the dorsal visual pathways, and magnocellular cells, in particular, Pillunat et al. (1985) considered that depressed flicker thresholds in severe AUD could be due to general damage at the retinal ganglion cells level, without further specification. Williams (1984) documented specific impairments for lower temporal frequencies, 3 Hz and 10 Hz rather than 30 Hz, and concluded that tonic (parvocellular-related) rather than phasic (magnocellular-related) cells might be impaired. However, these two studies did not consider the influence of the location of the flicker on the retina, another factor of interest when exploring the dorsal and ventral visual pathways, associated with magnocellular and parvocellular retinal ganglion cells, and rod and cone retinal photoreceptors, respectively. Indeed, visual information processed by rods, located in the periphery of the retina, can be transmitted faster than information captured by cones at the center of the retina (Laycock et al., 2007, 2008), so that visual stimuli can be perceived as discontinuous at higher frequencies in peripheral compared to central vision (Tyler, 1985, 1987). With that respect, Bertera and Parsons (1978) measured how the visual scanning abilities of individuals with severe AUD varied according to target eccentricity. They showed that peripheral locations did not influence search times but impacted the number of errors, suggesting an impoverished exploration of the peripheral visual field. While they did not link their results to the different properties of visual streams, these findings overall suggest that individuals with severe AUD could exhibit both parvocellular and magnocellular damage.

Global and Local Processing

Global and local processing corresponds to the tendency to process stimuli either at the general and configural level or based on features and details (Navon, 1977), thus informing on dorsal (mainly magnocellular-based) and ventral (mainly parvocellular-based) visual pathways respectively (Boothe, 2002; Hellige, 1996; Laycock et al., 2007; Merigan & Maunsell, 1993; Thomas et al., 2012). In severe AUD, the interest for global and local processing derives from the observation that individuals with severe AUD use different constructional strategies during visuoperceptual tasks, and is also linked to the early concept of "field-dependence". Initially considered as a personality trait (Goldstein & Chotlos, 1965), field-dependence refers to the "relatively passive submission to the domination of the background and inability to keep an item separate from its context" (Witkin, 1960, p.339), and has been explored from a cognitive perspective via the Rod and Frame Test (Witkin & Asch, 1948). In this test, participants have to adjust a rod displayed in a tilted square frame in order for the rod to be perfectly horizontal or vertical and are considered as field-dependent or independent depending on whether they adjust the rod according to the frame or seem to disregard external cues. Given the inconclusive results obtained, researchers concluded that individuals with severe AUD do not form a homogeneous group and that external factors (e.g., depression) modulate the tendency to be influenced by irrelevant global contextual information (Goldstein et al., 1970; Goldstein & Chotlos, 1965). The same conclusion applied when field-dependence was assessed via the Embedded Figure Test in which participants have to identify a target hidden in a complex background figure: individuals with severe AUD could not systematically be classified as more field-dependent than healthy controls (Jones & Parsons, 1972; Karp & Konstadt, 1965; Steiger et al., 1985).

Subsequent studies rather contradicted the idea that individuals with severe AUD might predominantly rely on global configural visual information, and might thus process featural cues less efficiently. A general opposite finding with that respect is that the lower performance of individuals with severe AUD on the Block Design subtest of the WAIS has generally been linked to a higher tendency to break global configurations (Beatty et al., 1997; Kramer et al., 1989). Indeed, individuals with severe AUD often struggle to solve puzzles configural analysis (Beatty et al., 1997). Kramer et al. (1991) further showed that individuals who made more

configural errors on the WAIS Block Design subtest also made fewer global choices when estimating the similarity between a standard figure and two optional figures sharing more global or local features with the standard, respectively. While the authors did not compare severe AUD and healthy controls groups, preventing any conclusion in terms of actual deficit (but see also Kramer et al., 1989), other findings confirmed the weakened influence of configurations on element processing by showing that individuals with severe AUD perceived simple single shapes in the same way as healthy controls but continued to use a more viewer-centered reference frame when contextual information was added (Robertson et al., 1985). The use of less effective perceptual and organizational strategies has also been highlighted when copying and recalling the Rey-Osterrieth Complex Figure. Daig et al. (2010) documented more piecemeal rather than holistic approaches at recall (but not copy), leading to a less detailed drawing. The unexpected lack of difference in strategy at copy, in contradiction with earlier results (Sullivan et al., 1992), might be due to the small sample. Still, Rosenbloom et al. (2009) obtained the same result with a larger sample. Even though individuals with severe AUD copied the figure less accurately and more slowly than healthy controls, they did not differ on their strategy score. A more holistic strategy at copy was, however, associated with better recall scores in both groups. These results suggested that copy accuracy and copy strategy made independent contributions to recall scores, implying that visuoperceptive constructional and organizational abilities represent distinct processes (Sullivan et al., 1992). Other less conclusive results were however found by Beatty et al. (1996) who reported several deficits in building tasks favoring either featural or configural analysis, including the Block Design subtest, but could not link these difficulties to one specific level of spatial processing, implying that individuals with severe AUD could rely on different strategies depending on the task.

Eventually, another cluster of studies focused on hierarchical tasks requiring a shift between different levels of visuoperceptive processing. Using Navon letters, each big letter composed of a number of smaller letters, Wegner et al. (2001) showed that individuals with severe AUD did not display an overall slower processing of visual cues and did not differ from healthy controls in visual attention allocation when target letters were presented equally often at local and global levels (non-biased condition) or when target letters were more often presented at one specific level (biased condition). They concluded that individuals with severe AUD were able to shift attention between spatial levels correctly and did not display any general deficit at the global level. Besides, in the interfering condition, in which each local target letter was combined alternatively with one closely similar or dissimilar global distractor letter, global information appeared to influence their local target processing excessively compared to healthy controls. Individuals with severe AUD processed local targets faster than healthy controls when the global distractor letter was similar rather than dissimilar than the local target letter, a result implying more influence of global information. More recently, Müller-Oehring et al. (2009) disclosed different findings with a similar hierarchical task: individuals with severe AUD were more sensitive to the interference of local information when having to selectively direct their attention towards global features. Importantly though, individuals with severe AUD did not display less interference from global information when having to process local features, while this pattern of results was expected if they had genuine difficulties in perceiving and discriminating global information. Since distractor effects are thought to be mediated by the asymmetric integration of information from both cerebral hemispheres, they defended the proposal of a defective inter-hemispheric transfer in severe AUD.

While not directly in line with global and local processing, Schulte et al. (2004) also drew attention to potential inter-hemispheric processing deficits in based on the "redundant target effect", i.e., the gain in reaction time observed when confronted with a double (i.e., a target in both visual fields) rather than a single (i.e., a target in one visual field only) visual stimulation (Corballis, 2002). They showed that older individuals with severe AUD did not benefit from the redundant target effect as much as healthy controls or younger individuals with severe AUD did and that the nature of the sensory input influenced this effect. More precisely, the use of equiluminant stimuli, that is, with equal luminance of the background which restricts visual processing to the parvocellular system (Livingstone & Hubel, 1988), was associated with a higher redundant gain in older participants. In contrast, the use of bright luminance contrast stimuli, linked to the magnocellular system, was associated with lower redundancy gains (Livingstone & Hubel, 1988). Following the hypothesis of a less efficient inter-hemispheric transfer, the authors also documented delayed reaction times for crossed targets compared to uncrossed targets in older individuals with severe AUD. Compared to healthy controls, individuals with severe AUD did not show the expected pattern of increase and decrease in speed when responding to targets presented in a visual field on the same side as the response hand (uncrossed) or on the opposite side (crossed). A subsequent study (Schulte et al., 2010) replicated a variation in the redundant target effect. Unlike healthy controls, individuals with severe AUD showed a higher processing advantage for unilateral rather than bilateral visual stimulations, especially when responding with their dominant hand. Given that no crossed-uncrossed difference was found, this processing asymmetry

could not be attributed solely to impaired interhemispheric transfer time. The authors thus suggested the presence of complementary intra-hemispheric changes in connectivity. Altogether, these results support a role for degraded inter- as well as intra-hemispheric connections in some weakened visual performances.

Other Low-Level Visual Abilities

Other visual properties have also occasionally been assessed to measure additional low-level visual functions. For example, visual acuity appeared preserved in severe AUD (Nicolás et al., 1997; Wegner et al., 2001), even under mesopic visual conditions (Campbell et al., 1981). Visual contrast sensitivity (VCS) measures conversely yielded mixed results. Using sinusoidal gratings of various spatial frequencies, Roquelaure et al. (1995) documented a reduction of VCS in severe AUD. This reduction was more pronounced for low and high spatial frequencies than intermediate ones. Optimal sensitivity was also associated with a lower spatial frequency range compared to healthy controls. Despite noticeable inter-individual differences, the authors concluded that severe AUD could be associated with optic nerve dysfunction, as reflected by changes in low spatial frequencies, and with isolated ametropia (a refractive disorder, such as myopia, hyperopia or astigmatism), as shown by changes in high spatial frequencies. Either way, the fact that individuals who displayed significant VCS changes also displayed higher gamma-glutamyl transpeptidase (i.e., a liver enzyme used as a biomarker for heavy alcohol use) and higher mean corpuscular volume levels (i.e., an increase in the size of red blood cells considered as a possible hematological complication of severe AUD) led to the conclusion that chronic alcohol consumption is involved in these visual changes. Two other studies examined VCS but could not replicate these findings. While the first did not find any difference in VCS in severe AUD (de Oliveira Castro et al., 2009), the second found significant lower VCS for low spatial frequencies (Martins et al., 2019). This second study also revealed that the individuals who displayed VCS alterations exhibited concurrent changes in color vision and that this last measure could better distinguish individuals with severe AUD from healthy controls. Color vision status influenced VCS results, namely, individuals with severe AUD showing color impairments displayed impaired VCS for a broader range of spatial frequencies, and especially higher ones. Unlike Roquelaure et al. (1995), the authors did not, however, speculate on the potential origin of VCS deficits.

One study also explored the low-level spatiotemporal properties of the visual system of individuals with severe AUD via the "shine-through effect" (Chkonia et al., 2012). The shine-through effect refers to the fact that a single misaligned vernier followed by a mask grating of 25 aligned verniers actually "shines" through the 25 elements, appearing as a distinct entity. Conversely, when the misaligned vernier is followed by a mask composed of fewer elements, such as only five aligned verniers, it cannot be as easily processed, leading to lower performances (Kunchulia et al., 2012). In both healthy controls and individuals with severe AUD, discrimination of the vernier offset was similarly affected by backward masking conditions, suggesting comparable spatiotemporal processing of the mask.

Finally, ophthalmologic studies showed that individuals with severe AUD could also display reduced static amplitude of accommodation. This change in the ability of the eye to adapt its optical power to maintain a suitable level of focus on a visual stimulus depending on its distance could be more severe in younger than older individuals with severe AUD compared to age-matched healthy controls (Campbell et al., 2001). Severe AUD could also be associated with slight mydriasis (i.e., larger pupil dilatation). Both results are consistent with the idea that severe AUD could cause a form of peripheral parasympathetic neuropathy (Campbell et al., 2001). More generally, many diseases of the visual system, among which cataract (e.g., Kanthan et al., 2010) or age-related macular degeneration (e.g., Chong et al., 2008) have been associated with alcohol consumption (but see Wang et al., 2008).

How Do Visuoperceptive Deficits Vary Across Individuals with Severe AUD?

Besides age (see the Brief Historical Background section), other factors might modulate visuoperceptive impairments. The influence of gender has been studied. Since women metabolize ethanol more slowly than men and reach, as a result, higher blood alcohol concentration, they might experience more severe short-term consequences and be more vulnerable to the long-term deleterious effects of severe AUD (Acker, 1985). Perceptual processes such as perceptual matching, spatial planning, or orientation have been associated with this gender vulnerability, along with working memory, problem-solving and cognitive flexibility in the visual modality (Acker, 1985; Flannery et al., 2007; Parsons et al., 1990b). A few studies showed that women displayed visuoperceptive deficits similar to men despite shorter abstinence durations and lower lifetime alcohol intake (Acker, 1985; Sullivan et al., 2002), and that this gender difference persisted when consumption was statistically equated (Acker, 1986). Accordingly, the functional reorganizations of brain systems due to severe AUD may occur at different rates across genders (Sullivan et al., 2002). However, contradictory findings revealed an absence of gender differences on visuoperceptive measures ranging from broad multi-determined test (such as the WAIS Digit Symbol subtest or Rey-Osterrieth Complex Figure; Wilson et al., 1987) to more specific measures (such as the Witkin Embedded Figure Test; Sparadseo et al., 1983), or reported more deficits in men (Drake et al., 1990; Fabian et al., 1981, 1984; Silberstein & Parsons, 1979, 1981). In the absence of consensus, it might be more relevant to delineate the exact pattern of visual deficits of women and men rather than

compare their severity, notably as all studies did not include both genders in the same experiment (e.g., Acker, 1985; Fabian et al., 1981; Jones et al., 1980; Silberstein & Parsons, 1979, 1981; Sullivan et al., 2002). Cross-study comparisons often contrasted heterogeneous groups with different comorbidities, various lifetime alcohol consumption rates, and diverse abstinence durations, making it challenging to draw conclusions (e.g., Silberstein & Parsons, 1981; Sullivan et al., 2002). A core issue is that men and women drinking habits often differ, and selecting women with longer drinking histories to match that of men may result in the creation of atypical samples, impairing the generalizability of the results.

Several studies investigated the influence of severe AUD duration and the amount of alcohol consumed to assess whether visuoperceptive deficits are linked to alcohol neurotoxicity, and most of them found non-significant correlations (e.g., Alterman et al., 1984; Bagga et al., 2014a, b; Beatty et al., 1995, 1997; Davies et al., 2005; Durazzo et al., 2013; Fabian et al., 1981; Fein et al., 2006; Forsberg & Goldman, 1985; Franceschi et al., 1984; Goldman et al., 1983; Hochla & Parsons, 1982; Mann et al., 1999; Noonberg et al., 1985; Rosenbloom et al., 2009; Wilson et al., 1987). This lack of correlation may reflect a differential influence of alcohol-related variables on various visuoperceptive abilities. For instance, changes in color vision have been found to correlate with severe AUD duration (Braun & Richer, 1993) and alcohol intake (Mergler et al., 1988). Individuals with severe AUD generally display primary alterations in the perception of the blue-yellow range but can present more complex dyschromatopsia involving the red-green axis in more advanced disease phases (Mergler et al., 1988). However, this gradation of the deficit might be more difficult to observe for other visual components. A few group comparisons contrasting sub-groups of individuals with varying severe AUD durations nevertheless supported this idea of progression in the severity of visuoperceptive impairments, by noting more severe deficits in individuals with longer severe AUD history (Bertera & Parsons, 1978; Ciesielski et al., 1995; Jones, 1971; Jones & Parsons, 1971). However, these studies did not focus on specific visuoperceptive processes and often relied on different arbitrarily chosen cut-offs for severe AUD durations. At present, one of the most interesting result concerning drinking history is probably the observation that individuals with longer severe AUD duration process peripheral cues less efficiently than healthy controls, while individuals with shorter severe AUD duration appear to fall in-between (Bertera & Parsons, 1978). Finally, while some visual properties might be linearly related to lifetime drinking amount, others might be impaired once a certain amount of consumption is reached, and might thus be better appraised as a critical threshold rather than continuous phenomena. Overall, the effects of alcohol may also be cumulative and probably depend on a more complex interaction between the quantity consumed and the severe AUD duration.

Recovery of Visuoperception: Do Deficits Persistent Over Time?

It has often been suggested that visuoperceptive deficits could persist long after alcohol detoxification (Crowe et al., 2019; Oscar-Berman et al., 2014). Several studies have shown residual deficits weeks (Clarke & Haughton, 1975; Forsberg & Goldman, 1985; Jacobson et al., 1970; Müller-Oehring et al., 2015; Obaldia et al., 1981), months (Ioime et al., 2018; Miglioli et al., 1979) or years (Fein et al., 2006; Parsons et al., 1990a; Yohman et al., 1985) after abstinence onset on several visuoperceptive tests. More precise lasting deficits, such as lower complex figure-ground analysis, have also been reported. In a cross-sectional study comparing short-term (1–2 months), long-term (1–3 years) and prolonged (> 5 years) abstinent individuals with severe AUD (Brandt et al., 1983), visual short-term memory and psychomotor skills were recovered in three to five years, whereas scores on the Embedded Figures Test remained impaired after five years. Despite evidence of some recovery following abstinence, these results suggest that even long-term abstinence is sometimes insufficient to achieve full recovery.

However, contrary reports suggesting intact performances on the Trail Making Test or WAIS Block Design subtest in long-term abstinent individuals with severe AUD (8 months−7 years) indicated that total visuoperceptive recovery could happen (Emmerson et al., 1987; Fein & McGillivray, 2007; Harris et al., 2003; Reed et al., 1992; Templer et al., 1975). Recovery of perceptual-motor speed, visuoconstructive ability, nonverbal reasoning, and spatial imagination, after only five weeks of abstinence, have also been documented (Mann et al., 1999). These findings partly match those in a recent study (Mulhauser et al., 2018), which suggest that visuoperception might be more responsive to abstinence than other cognitive skills, as illustrated by a significant improvement on a figure copy test after ten days without alcohol compared to other verbal, attentional and memory tests (see also Ritz et al., 2014). The precise time course of recovery is still debated, and some authors have suggested that most visuoperceptive scores could normalize early with abstinence (Davies et al., 2005; Konrad et al., 2012; Leber et al., 1981). According to Grant et al. (1984), the intermediate phase between withdrawal syndrome (2–3 weeks of abstinence) and the plateau of improvement (one year of abstinence) might be characterized by a "subclinical alcohol-related neuropsychological disorder" that may go unnoticed.

Recovery timings may diverge according to the visuoperceptive sub-skills considered. For instance, visual motion perception and speed discrimination may remain impaired after 3 weeks of abstinence, whereas global and local visual processing may normalize during this period (Wegner et al., 2001). Despite improvements in movement detection and visual search within 2–3 weeks of abstinence, a subtle impairment in locating very rapid visual changes could persist beyond one month (Wilson et al., 1988). Color vision may

also recover quickly during the first month (Kapitany et al., 1993), implying that specific visual-related neurobiological processes might be at work during this early period. More precisely, alcohol-related unspecific color vision deficits may rapidly decrease with abstinence, while specific blue-yellow impairments may persist in line with hepatic damage (Verriest et al., 1980). Such observations stress the need to consider other medical risk factors before attributing every long-term residual and permanent neuropsychological deficit to alcohol neurotoxicity. Besides, just as with severe AUD duration, most studies also failed to report a strong correlation between abstinence duration and visuoperceptive abilities (Davies et al., 2005; Fein et al., 2006; Hochla & Parsons, 1982; Wilson et al., 1987), suggesting that factors other than abstinence duration influence recovery.

Actually, visuoperceptive recovery may also depend on practice opportunities, and thus be experience-dependent in addition to being time-dependent (Ellenberg et al., 1980; Forsberg & Goldman, 1985). Importantly, visuoperceptive practice effects are not limited to increased familiarity with the visuoperceptive stimuli of a particular test. They also induce a significant performance transfer on comparable new visuoperceptive tasks, even during early abstinence (Forsberg & Goldman, 1985, but see Adams et al., 1980). This result implies that experience-dependent processes might also trigger recovery, a result of primary importance for rehabilitation. However, it remains unknown whether normalization of visuoperceptive performances reflects a restoration or a compensatory mechanism. In particular, some individuals with severe AUD might compensate for their low-level perceptual deficits through alternate higher-level executive strategies (Fama et al., 2004; Sassoon et al., 2007).

It thus cannot be concluded that visuoperceptive deficits fully recover with abstinence, nor that they remain stable in the long run. In general, behavioral and cerebral alterations do not follow a linear pattern of recovery in severe AUD: Improvement is usually substantial in the short-term while more modest changes are observed in the mid- and long-term (Bates et al., 2013; Durazzo et al., 2015). Regarding visuoperception, reviews of between-group comparisons with healthy controls showed significant moderate effect sizes within the first year of abstinence, and smaller ones afterward (Crowe et al., 2019; Stavro et al., 2013).

Here again, methodological flaws (e.g., multi-determined visuoperceptive tasks, limited use of size effect estimates, lack of longitudinal explorations) limit the insights offered by these studies. Furthermore, measuring recovery at the group level may also mask substantial inter-individual differences (Bates et al., 2013), as recovery is multi-determined. Alcohol-consumption variables and demographic factors also interact with each other. For instance, recovery could be influenced by the combination of age and severe AUD duration, results suggesting lower recovery in older individuals with severe AUD (Goldman et al., 1983), particularly those with longest severe AUD durations (Ellenberg et al., 1980; Munro et al., 2000; but see also Fein & McGillivray, 2007).

Visuoperceptive Deficits in the Broader Cognitive Profile of Individuals with Severe AUD

While studies on visuoperception in severe AUD rarely clarified the different levels of processing, severe AUD appears associated with deficits all along the classical retino-geniculate-cortical visual pathway (Fig. 3). Damage could arise at the level of the retina, and especially retinal ganglion cells, as indicated by achromatopsia and lower flicker fusion thresholds (de Oliveira Castro et al., 2009; Martins et al., 2019; Pillunat et al., 1985). Severe AUD could also affect the successive visual relays, from optic nerves, as suggested by contrast sensitivity changes (Roquelaure

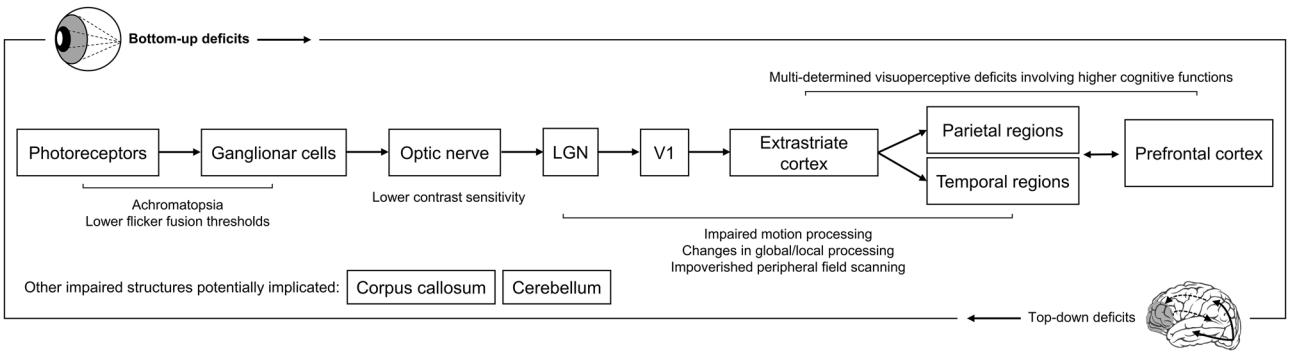

Fig. 3 Deficits all along the retino-geniculo-cortical pathway and beyond. Note. Visuoperceptive deficits in severe AUD are thought to implicate various structures, from the eye up to cortical regions, including the prefrontal cortex. The contour of the figure shows the bidirectionality of visuoperception, which relies on bottom-up and top-down mechanisms and can thus be disrupted by dysfunctional feedforward or feedback mechanisms. Impaired processes related to more precise components of the visual system are mentioned below the corresponding boxes. In line with the idea of widespread deficits, the figure also underlines the potential contribution of the corpus callosum and the cerebellum. LGN, Lateral Geniculate Nucleus; V1, primary visual cortex

et al., 1995), to the LGN up to the primary visual cortex and extrastriate cortex, as illustrated by impaired motion (Wegner et al., 2001) and global–local processing (Schulte et al., 2010). It is more challenging to delineate impairments according to the subdivision of the retino-geniculate-cortical pathway into the functionally distinct ventral (mostly parvocellular) and dorsal (mostly magnocellular) streams. This subdivision originates at the retina but is more evident in the LGN, which contains dedicated layers for each type of cells, as well as at the cortical level, where parvocellular and magnocellular projections pass through primary and secondary visual regions before progressively feeding the temporal and parietal streams, respectively (Merigan & Maunsell, 1993). Results of studies on color vision and specific patterns of deficits, such as impairment for low speeds and low temporal frequencies, have initially linked the impairments to parvocellular damage (Wegner et al., 2001; Williams, 1984). However, the processing of motion, speed, and global visual cues, ordinarily falls under the scope of the magnocellular pathway. Combined with signs of impoverished peripheral field exploration, these results suggest that this pathway could also be impaired in individuals with severe AUD.

The interpretation of the deficits is further complexified by the lack of well-defined a priori hypotheses stemming from a pre-established theoretical framework of visuoperception in severe AUD. Beyond the right hemisphere and premature aging hypotheses, it has been proposed that visuoperceptive deficits are part of a generalized cerebral dysfunction (Nicolás et al., 1993; Tivis et al., 1995) accounting for the widespread cognitive impairments in severe AUD (Crowe et al., 2019; Stavro et al., 2013). Drawing on the systematic presence of other concomitant deficits, alternative proposals stressed the importance of other specific structures in the occurrence of visuoperceptive-related deficits, such as the prefrontal cortex (Ciesielski et al., 1995; Davies et al., 2005; Sullivan et al., 1992, 2000; Uekermann et al., 2003), the cerebellum (Fitzpatrick & Crowe, 2013; Hillbom et al., 1986; Sullivan et al., 2000) or the corpus callosum (Müller-Oehring et al., 2009; Schulte et al., 2004, 2006). In particular, ascribing a specific role to the prefrontal cortex is consistent with the "frontal lobe hypothesis", which suggests that frontal impairments is a cardinal sign of severe AUD (Moselhy et al., 2001), as cerebral damage appears to be more severe in the frontal lobe (Pfefferbaum et al., 1997). These proposals thus support an anatomically widespread deficit, which appears coherent with current data showing diffuse brain damage in severe AUD, with some areas, however, being more sensitive than others (Bühler & Mann, 2011).

The idea that visuoperception interacts with other cerebral networks is at the heart of the contemporary conceptualizations of vision. These models no longer consider vision as an isolated and purely sensory mechanism (Newen & Vetter, 2017; O'Callaghan et al., 2017) but rather postulate that cognitive and affective factors like our goals, motivations, and current emotional states, influence visuoperceptual processes (Bar, 2004; Barrett & Bar, 2009). Such new theoretical models of vision acknowledge the presence of bottom-up and top-down feedback between low-level sensory-perceptual routes and higher-level cortical regions (Newen & Vetter, 2017), implying that visuoperception communicates flexibly with other cerebral systems. The anatomo-functional properties of the visuoperceptive system and the close connections it maintains with prefrontal regions, and especially the orbitofrontal cortex (Bar et al., 2006; Kringelbach, 2004; Kveraga et al., 2007), further support this proposal. Accordingly, visuoperception impairments could arise from damage to the visual system, higher-level cerebral areas, or communication paths between these structures.

Recontextualizing visuoperceptive deficits in the broader cognitive profile of individuals with severe AUD might provide a way to reconnect the severe AUD literature with the new models of vision. However, the present work reviewed behavioral studies using very different tasks, most of them lacking sensitivity. Any conclusion regarding the cerebral correlates of visuoperception impairments in severe AUD remains, therefore, hypothetical.

Limitations and Research Perspectives

Numerous limitations have been highlighted throughout this review, notably regarding the heterogeneity of tasks and participants' characteristics. The lack of visuoperceptive sensitivity of many tasks is probably the most problematic issue. The use of broad visuoperceptive tasks (e.g., Trail-Making Test, WAIS Block Design and Digit Symbol subtests, Rey-Osterrieth Complex Figure) may be explained by the aim of early studies to explore the right hemisphere or premature aging hypotheses rather than to target specific visuoperceptive processes. However, little effort has been made since then to refine these tasks so that recent studies still rely on these multi-determined tasks to assess visuoperception (Crowe et al., 2019; Oscar-Berman et al., 2014). This concern also applies to recent neuroimaging studies that link anatomical and functional changes to visuoperceptive deficits using similar tests that do not distinguish perceptual from mnemonic or executive processes (Bagga, et al., 2014a, b). Moreover, studies exploring more precise visuoperceptive components have not capitalized on a robust theoretical framework. This lack of up-to-date, careful, and systematic investigation of the visual system in severe AUD results in the total absence of an integrated model of visuoperception. Nevertheless,

this limitation is probably the easiest to overcome. The first step may be to study the dorsal-magnocellular and ventral-parvocellular pathways more thoroughly through their distinct properties, before exploring their interactions, and then the impact of the deficits on other cognitive processes implicated in severe AUD (see Creupelandt et al., 2019 for a detailed research agenda and examples of tasks). In particular, the fact that the new models of vision ascribe different functions to the two visual pathways, from the early processing of basic temporal and spatial visual features to higher-level visual recognition and judgments processes (Bar, 2004; Barrett & Bar, 2009; O'Callaghan et al., 2017), further justifies the need to understand the specific contribution of these two pathways to visuoperceptive deficits in severe AUD. Such investigations have been conducted in other populations (e.g., dyslexia: Ahmadi et al., 2015; Schulte-Körne & Bruder, 2010; autism and schizophrenia: Laycock et al., 2007; Robertson & Baron-Cohen, 2017; Tsermentseli et al., 2008) and could be conducted in relation to severe AUD.

The second significant limitation is the heterogeneity of the severe AUD population. While most recent papers referred to the DSM-IV or DSM-5 criteria, many studies conducted before the '90s did not provide any information regarding severe AUD criteria or only mentioned the quantity and frequency of alcohol consumption. As a result, different definitions of severe AUD may have been adopted. Then, the lack of systematic assessment and control of comorbid psychiatric symptoms or diagnoses among individuals with severe AUD may also have influenced the results (D'Hondt et al., 2018). For instance, depression, which is frequently comorbid with severe AUD (Jeanblanc, 2015), can affect visual functions (Bubl et al., 2010; Norton et al., 2016). Finally, the visual system is highly vulnerable to subtle changes in chemicals so that the side effects of medication, not to mention other addictive chemicals, including nicotine, might have influenced some results (Ceballos et al., 2005, 2006; Durazzo et al., 2013). Greater standardization of patient inclusion criteria and screening of comorbidities are thus needed to facilitate future comparisons across studies.

Those two limitations contribute to the difficulty of isolating the specific contribution of alcohol to visuoperceptive deficits. The problem in assessing the causal role of alcohol applies for most severe AUD studies and cannot be overcome by cross-sectional group comparisons. However, the limited correlations found between alcohol-related variables and visuoperceptive impairments further call into question the specific impact of alcohol on these deficits. The existence of premorbid differences cannot be excluded either, especially since the relatives of individuals with severe AUD may display visuospatial deficits (Schandler et al., 1988; Whipple et al., 1988). Longitudinal studies, however, showed that adolescents with a family history of severe AUD might present pre-existing frontal deficits while displaying reduced temporo-parietal volumes and experiencing visuoperceptive deficits as a post-drinking consequence (Squeglia et al., 2014a, b). Several alternative explanations have been put forward to explain the lack of relationship between alcohol consumption and visuoperceptive deficits: First, some very subtle visuoperceptive deficits might only be observable at the cerebral level, and some visual deficits might only reveal themselves in combination with other impairments, especially frontal ones. This proposal, which builds on the "frontal lobe hypothesis" (Pfefferbaum et al., 1997), is also compatible with the principles of the new models of vision, positing that visuoperception does not act in isolation and mobilizes other cortical circuits, especially in frontal areas. Accordingly, deficits would be more likely to be evidenced by visuospatial tasks engaging executive functions because they simultaneously recruit multiple cognitive functions and cerebral circuitries (Zinn et al., 2004). Second, there might be a threshold beyond which varying degrees of alcohol abuse lead to similar neuropsychological impairments (Beatty et al., 1995). The relationship between severe AUD duration and neuropsychological performance might thus not be linear, and considering other drinking parameters (e.g., repeated withdrawal, physical impairment induced by alcohol, or personality characteristics) might be more relevant (Goldman et al., 1983; Mann et al., 1999). Alternatively, the recovery of some visuoperceptive deficits in the course of abstinence could mask correlations and explain why many studies did not find significant links (Davies et al., 2005). Studies may have targeted specific visuoperceptive skills at the time at which alcohol not longer appeared influential, because this specific skill had already recovered, or because recovery of intertwined deficits (e.g., executive ones) no longer allowed visuoperceptive impairments to be evidenced at the behavioral level. Importantly, all these proposals might not be mutually exclusive, and efforts should thus be made to determine the exact manner in which alcohol impacts visuoperception in the long run.

Finally, the current lack of precise understanding of the pathophysiology underlying visuoperceptive deficits in severe AUD may also strongly contribute to the limited insights regarding the causal role of alcohol. Pathophysiology was rarely mentioned in the papers included. Direct neurotoxic effects of alcohol consumption on visual structures have initially been postulated within the framework of toxic optic neuropathies. However, this proposal is now criticized as optic neuropathies in severe AUD have often been linked to nutritional deficiencies rather than alcohol toxicity (Sharma & Sharma, 2011; Spinazzi, 2019). Vitamin B12 or folic acid shortage could cause accumulations of formic acid, which could inhibit the

electron transport chain and impair mitochondrial functions, leading to disruption of adenosine triphosphate production and, eventually, to defective ATP-dependent axonal transport and demyelination of the optic nerve (Sharma & Sharma, 2011). Impaired oxidative phosphorylation due to malnutrition could also cause axonal loss, especially of parvocellular neurons in the papillomacular bundle, resulting in a thinning of retinal fibers (Ahuja et al., 2016). Still, the studies examined in the present review focused on individuals with severe AUD who did not show signs of nutritional-related dementia onset, which argues against the idea that nutritional factors may solely explain visual-related damage. Animal studies controlling for dietary intake of rats showed that alterations of the retina and optic nerves could occur in the absence of nutritional deficiencies. These studies also implicated oxidative stress mechanisms but interpreted the related biological changes (namely, increase in lipid peroxidation products, decrease in endogenous antioxidants and dysregulation of antiapoptotic proteins) as a direct consequence of ethanol toxicity (Aviñó et al., 2002; Sancho-Tello et al., 2008). Consistently, retinal analyses of monkeys who had been chronically exposed to alcohol revealed a decrease in fatty acids, which are abundant in rod membranes and protect photoreceptors from oxidative stress-induced apoptosis (Pawlosky et al., 2001). Besides, chronic alcohol intake influences the weight and neuronal density of the LGN and superior colliculus in rats, emphasizing that alcohol toxicity also damages visual relays (Fakunle et al., 2012). In this case, alcohol was thought to induce autoimmune inflammatory responses, promoting the production of cytotoxic cytokines in the microglia. Several human studies also documented signs of reduced grey matter integrity (Durazzo et al., 2015, 2017; Fein et al., 2009; Mackey et al., 2019; Rando et al., 2011; Sullivan et al., 2018; Wang et al., 2018; Zhu et al., 2018), and white matter integrity (Bagga et al., 2014b; Schmahmann & Pandya, 2007), in various nodes of the visual system, distributed in the occipital, temporal and parietal cortices, suggesting mechanisms of apoptosis and demyelination. While the exact underlying pathophysiology of these cortical damages also remains to be clarified, a recent study showed that occipital changes in N-acetyl-aspartate (considered as a marker of neuronal viability), glutamate, and choline (considered as a marker of myelination and cell membrane metabolism) ratios could be involved in the visuoperceptive deficits of individuals with severe AUD (Bagga et al., 2014a). Additional work is needed to examine the interactions and influence of these various biological mechanisms.

In sum, we stress the need to adopt a process-centered approach of visuoperception and to develop more precise hypotheses through a careful investigation of the anatomo-functional properties of the human visual pathways. We also emphasize the necessity to consider the inter-individual variability inherent to the severe AUD population, or at least to standardize inclusion procedures and to interrogate the role of visuoperceptive deficits for other cognitive systems. In parallel, the pathophysiology of the deficits should be clarified by bridging behavioral and neuroimaging studies, to develop a new integrated model of visuoperception in severe AUD.

Clinical Implications

The paucity of precise visuoperceptive investigations in severe AUD and the absence of theoretical models also have significant repercussions at the clinical level. We do not precisely know which visuoperceptive processes might be impaired in individuals with severe AUD, to what extent, and in which timeframe. Accordingly, no precise practical guidelines can be provided as it remains unclear as to which components to act upon, at what time point, and for what purpose. Omnibus remediation of visuoperception is a non-operationalizable objective, and cognitive programs should also capitalize on preserved subfunctions and potential compensatory mechanisms, which also remain barely known. Although visuoperceptive skills could improve with practice among individuals with severe AUD (Forsberg & Goldman, 1985), we did not identify any interventional study in this review, suggesting that no rehabilitation program directly exploring visuoperception has been assessed. This reduced clinical interest appears related to a series of interrelated factors.

First, the fact that visuoperceptive deficits have been found to persist after long-term abstinence may have promoted the idea that visuoperception could not improve in individuals with severe AUD. Nevertheless, even though visuoperceptive impairments in severe AUD might be too subtle to affect autonomy and appear to partially improve in the long run (Crowe et al., 2019; Stavro et al., 2013), functional approaches might be justified for individuals with severe AUD who display particularly severe and disabling visuoperceptive deficits. In these specific cases, psychoeducation combined with the implementation of concrete functional adjustments (e.g., brighter lighting, visual magnification) might reduce visuals demands in private and occupational contexts (Prakash et al., 2011; Markowitz et al., 2006).

Second, although we promoted the idea that alcohol consumption led to visuoperceptive deficits and not the other way round, the influence of visuoperceptive deficits on severe AUD maintenance or relapse should be considered. Visuoperception might have been ignored because its influence on relapse is less conceptually evident than that of the executive or emotional deficits (Rupp et al., 2017; Czapla et al., 2016; Wilcox et al., 2014). However, the limitations raised by

the present review regarding the literature on visuoperception in severe AUD suggest that it is premature to conclude to the total absence of a link between visuoperception and relapse (Desfosses et al., 2014). This idea is further reinforced by neuroimaging studies showing that functional changes in visual-related cerebral areas and disrupted connectivity between visual and frontal regions could be linked to relapse (Rando et al., 2011; Wang et al., 2018).

Third and finally, the current situation may also spring from the tendency to consider visuoperception from a strictly bottom-up perspective. Still, in light of the above-mentioned recent models of vision, visuoperceptive deficits are relevant to consider in the management of severe AUD as (1) individuals with severe AUD may process visual information less efficiently and therefore, base their decisions on degraded visual information (bottom-up deficit), and (2) their visuoperceptive processes may also be disrupted by the presence of concurrent higher-level cognitive and emotional deficits, as these systems are supposed to guide and monitor visuoperception. As a result, the subsequent cognitive stages would not be adequately anticipated and facilitated (top-down deficit). In this updated context, early visuoperceptive deficits may thus affect the whole cognitive continuum during information processing. Future research should reconsider the role of visuoperceptive deficits in some of the classical cognitive and affective impairments associated with severe AUD (e.g., attentional biases, emotional facial expression decoding), which depend on the visual modality and influence relapse (Creupelandt et al., 2019; D'Hondt et al., 2014a, b).

From a clinical perspective, reassessing the importance of visuoperceptive disorders in the overall profile of individuals with severe AUD could help to determine whether introducing a preliminary visuoperceptive rehabilitation program would have a positive effect on the early sensory stages of the cognitive continuum, which may then accelerate the recovery of other visual-related memory or executive processes. Encouraging results in healthy individuals suggest that training basic visual features could transfer from elementary stimuli to complex ones (Peters et al., 2017). These results promise significant benefits and open new perspectives on how to complement already existing cognitive training programs with visuoperceptive exercises.

Conclusions

This review confirmed the presence of visuoperceptive deficits in individuals with severe AUD but also highlighted the lack of deep understanding of these impairments, which have not been conceptualized within a clear theoretical framework. Whereas our understanding of vision has recently evolved, this sensory activity being now considered as a dynamic process that involves rapid interactions between visual and frontal areas, researchers' interest for visuoperception in severe AUD appears to have faded. As a result, the actual implications of visuoperceptive alterations in individuals with severe AUD and the way these impairments interact with other deficits (e.g., executive ones) remain unknown. We thus stress the need to build a more integrated model of visuoperceptive deficits in severe AUD and to reduce the gap with the new theoretical models of vision. This paper, focusing on the behavioral literature, only constitutes the first attempt to bring together a very heterogeneous and scattered literature. Delineating a proper "map" of the deficits and developing concrete clinical applications will only be achieved by combining the present results with data stemming from neuroscience research and studies investigating higher-level visual processing.

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Compliance with Ethical Standards

Conflicts of Interest The authors declare that they have no conflict of interest.

References

Acker, C. (1985). Performance of female alcoholics on neuropsychological testing. *Alcohol and Alcoholism, 20*(4), 379–386. https://doi.org/10.1093/oxfordjournals.alcalc.a044560.

Acker, C. (1986). Neuropsychological deficits in alcoholics: the relative contributions of gender and drinking history. *British Journal of Addiction, 81*(3), 395–403. https://doi.org/10.1111/j.1360-0443.1986.tb00346.x.

Acker, C., Acker, W., & Shaw, G. K. (1984). Assessment of cognitive function in alcoholics by computer: a control study. *Alcohol and Alcoholism, 19*(3), 223–233. https://doi.org/10.1093/oxfordjournals.alcalc.a044439.

Adams, K. M., Grant, I., & Reed, R. (1980). Neuropsychology in alcoholic men in their late thirties: one-year follow-up. *The American Journal of Psychiatry, 137*(8), 928–931. https://doi.org/10.1176/ajp.137.8.928.

Ahmadi, K., Pouretemad, H. R., Esfandiari, J., Yoonessi, A., & Yoonessi, A. (2015). Psychophysical Evidence for Impaired Magno, Parvo, and Konio-cellular Pathways in Dyslexic Children. *Journal of Ophthalmic & Vision Research, 10*(4), 433–440. https://doi.org/10.4103/2008-322X.176911.

Ahuja, S., Kumar, P. S., Kumar, V. P., Kattimani, S., & Akkilagunta, S. (2016). Effect of chronic alcohol and tobacco use on retinal nerve fibre layer thickness: a case–control study. *BMJ Open Ophthalmology*, *1*(1). https://doi.org/10.1136/bmjophth-2016-00000.

Alterman, A. I., Tarter, R. E., Petrarulo, E. W., & Baughman, T. G. (1984). Evidence for impersistence in young male alcoholics. *Alcoholism: Clinical and Experimental Research*, *8*(5), 448–450. https://doi.org/10.1111/j.1530-0277.1984.tb05699.x.

American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.).

American Psychiatric Association. (1987). *Diagnostic and statistical manual of mental disorders* (3rd ed., rev.).

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.).

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text rev.).

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). https://doi.org/10.1176/appi.books.9780890425596.

Andre, J. T., Tyrrell, R. A., Leibowitz, H. W., Nicholson, M. E., & Wang, M. (1994). Measuring and predicting the effects of alcohol consumption on contrast sensitivity for stationary and moving gratings. *Perception & Psychophysics, 56*(3), 261–267. https://doi.org/10.3758/BF03209760.

Aviñó, J., Díaz-Llopis, M., España, E., Johnsen-Soriano, S., Romero, B., Marín, N., Muriach, M., Bosch-Morell, F., & Romero, F. J. (2002). Estrés oxidativo en el nervio óptico de la rata inducido por la administración crónica de etanol. [Chronic ethanol feeding induces oxidative stress in the optic nerve of rats]. *Archivos De La Sociedad Espanola De Oftalmologia*, *77*(5), 263–268.

Bagga, D., Khushu, S., Modi, S., Kaur, P., Bhattacharya, D., Garg, M. L., & Singh, N. (2014a). Impaired visual information processing in alcohol-dependent subjects: a proton magnetic resonance spectroscopy study of the primary visual cortex. *Journal of Studies on Alcohol and Drugs*, *75*(5), 817–826. https://doi.org/10.15288/jsad.2014.75.817.

Bagga, D., Sharma, A., Kumari, A., Kaur, P., Bhattacharya, D., & Garg, M. L. (2014b). Decreased white matter integrity in fronto-occipital fasciculus bundles: relation to visual information processing in alcohol-dependent subjects. *Alcohol, 48*(1), 43–53. https://doi.org/10.1016/j.alcohol.2013.10.009.

Bar, M. (2004). Visual objects in context. *Nature Reviews Neuroscience, 5*(8), 617–629. https://doi.org/10.1038/nrn1476.

Bar, M., Kassam, K. S., Ghuman, A. S., Boshyan, J., Schmid, A. M., & Dale, A. M. (2006). Top-down facilitation of visual recognition. *Proceedings of the National Academy of Sciences of the United States of America, 103*(2), 449–454. https://doi.org/10.1073/pnas.0507062103.

Barrett, L. F., & Bar, M. (2009). See it with feeling: affective predictions during object perception. *Philosophical Transactions of the Royal Society B: Biological Sciences, 364*(1521), 1325–1334. https://doi.org/10.1098/rstb.2008.0312.

Bates, M. E., Buckman, J. F., & Nguyen, T. T. (2013). A Role for Cognitive Rehabilitation in Increasing the Effectiveness of Treatment for Alcohol Use Disorders. *Neuropsychology Review, 23*(1), 27–47. https://doi.org/10.1007/s11065-013-9228-3.

Beatty, W. W., Blanco, C. R., Hames, K. A., & Nixon, S. J. (1997). Spatial cognition in alcoholics: influence of concurrent abuse of other drugs. *Drug and Alcohol Dependence, 44*(2–3), 167–174. https://doi.org/10.1016/S0376-8716(97)01334-3.

Beatty, W. W., Hames, K. A., Blanco, C. R., Nixon, S. J., & Tivis, L. J. (1996). Visuospatial perception, construction and memory in alcoholism. *Journal of Studies on Alcohol*, *57*(2), 136–143. https://doi.org/10.15288/jsa.1996.57.136.

Beatty, W. W., Katzung, V. M., Moreland, V. J., & Nixon, S. J. (1995). Neuropsychological performance of recently abstinent alcoholics and cocaine abusers. *Drug and Alcohol Dependence, 37*(3), 247–253. https://doi.org/10.1016/0376-8716(94)01072-S.

Bertera, J. H., & Parsons, O. A. (1978). Impaired visual search in alcoholics. *Alcoholism: Clinical and Experimental Research*, *2*(1), 9–14. https://doi.org/10.1111/j.1530-0277.1978.tb04685.x.

Blusewicz, M. J., Dustman, R. E., Schenkenberg, T., & Beck, E. C. (1977a). Neuropsychological correlates of chronic alcoholism and aging. *The Journal of Nervous and Mental Disease, 165*(5), 348–355. https://doi.org/10.1097/00005053-197711000-00006.

Blusewicz, M. J., Schenkenberg, T., & Dustman, R. E. (1977b). WAIS performance in young normal, young alcoholic, and elderly normal groups: an evaluation of organicity and mental aging indices. *Journal of Clinical Psychology, 33*(4), 1149–1153. https://doi.org/10.1002/1097-4679(197710)33:4%3c1149::aid-jclp2270330451%3e3.0.co;2-c.

Boothe, R. G. (2002). *Perception of the visual environment*. Springer.

Bowden, S. C. (1988). Learning in young alcoholics. *Journal of Clinical and Experimental Neuropsychology, 10*(2), 157–168. https://doi.org/10.1080/01688638808408232.

Bowden, S. C., Benedikt, R., & Ritter, A. J. (1992). Delayed matching to sample and concurrent learning in nonamnesic humans with alcohol dependence. *Neuropsychologia, 30*(5), 427–435. https://doi.org/10.1016/0028-3932(92)90090-9.

Brandt, J., Butters, N., Ryan, C., & Bayog, R. (1983). Cognitive loss and recovery in long-term alcohol abusers. *Archives of General Psychiatry, 40*(4), 435–442. https://doi.org/10.1001/archpsyc.1983.01790040089012.

Braun, C. M., & Richer, M. (1993). A comparison of functional indexes, derived from screening tests, of chronic alcoholic neurotoxicity in the cerebral cortex, retina and peripheral nervous system. *Journal of Studies on Alcohol*, *54*(1), 11–16. https://doi.org/10.15288/jsa.1993.54.11.

Brion, M., Pitel, A.-L., Beaunieux, H., & Maurage, P. (2014). Revisiting the Continuum Hypothesis: Toward an In-Depth Exploration of Executive Functions in Korsakoff Syndrome. *Frontiers in Human Neuroscience, 8,* 498. https://doi.org/10.3389/fnhum.2014.00498.

Bubl, E., Kern, E., Ebert, D., Bach, M., & Tebartz van Elst, L. (2010). Seeing Gray When Feeling Blue? Depression Can Be Measured in the Eye of the Diseased. *Biological Psychiatry, 68*(2), 205–208. https://doi.org/10.1016/j.biopsych.2010.02.009.

Bühler, M., & Mann, K. (2011). Alcohol and the Human Brain: A Systematic Review of Different Neuroimaging Methods. *Alcoholism: Clinical and Experimental Research*, *35*(10), 1771–1793. https://doi.org/10.1111/j.1530-0277.2011.01540.x.

Burnett, L. J., Marshall, P. H., & Bell, R. W. (1996). Reality monitoring in recovering alcoholics. *Journal of Studies on Alcohol*, *57*(2), 210–216. https://doi.org/10.15288/jsa.1996.57.210.

Butters, N., Cermak, L. S., Montgomery, K., & Adinolfi, A. (1977). Some comparisons of the memory and visuoperceptive deficits of chronic alcoholics and patients with Korsakoff's disease. *Alcoholism: Clinical and Experimental Research*, *1*(1), 73–80. https://doi.org/10.1111/j.1530-0277.1977.tb05771.x.

Butters, N., Lewis, R., Cermak, L. S., & Goodglass, H. (1973). Material-specific memory deficits in alcoholic Korsakoff patients. *Neuropsychologia, 11*(3), 291–299. https://doi.org/10.1016/0028-3932(73)90040-7.

Campbell, H., Doughty, M. J., Heron, G., & Ackerley, R. G. (2001). Influence of chronic alcohol abuse and ensuing forced abstinence on static subjective accommodation function in humans. *Ophthalmic and Physiological Optics, 21*(3), 197–205. https://doi.org/10.1046/j.1475-1313.2001.00567.x.

Campbell, T. D., Sampliner, R. E., Russell, R. M., & Garrett, M. S. (1981). Night driving (mesopic) visual acuity in sober male alcoholics with and without liver disease. *Alcoholism: Clinical and Experimental Research*, *5*(1), 34–37. https://doi.org/10.1111/j.1530-0277.1981.tb04861.x.

Cavalcanti-Galdino, M. K., da Silva, J. A., Mendes, L. C., dos Santos, N. A., & Simas, M. L. B. (2014). Acute effect of alcohol intake on sine-wave Cartesian and polar contrast sensitivity functions. *Brazilian Journal of Medical and Biological Research, 47*(4), 321–327. https://doi.org/10.1590/1414-431X20143209.

Ceballos, N. A., Tivis, R., Lawton-Craddock, A., & Nixon, S. J. (2005). Visual-spatial attention in alcoholics and illicit stimulant abusers: effects of nicotine replacement. *Progress in Neuro-Psychopharmacology & Biological Psychiatry, 29*(1), 97–107. https://doi.org/10.1016/j.pnpbp.2004.10.011.

Ceballos, N. A., Tivis, R., Lawton-Craddock, A., & Nixon, S. J. (2006). Nicotine and cognitive efficiency in alcoholics and illicit stimulant abusers: implications of smoking cessation for substance users in treatment. *Substance Use & Misuse, 41*(3), 265–281. https://doi.org/10.1080/10826080500409076.

Ceccanti, M., Hamilton, D., Coriale, G., Carito, V., Aloe, L., & Chaldakov, G. (2015). Spatial learning in men undergoing alcohol detoxification. *Physiology & Behavior, 149,* 324–330. https://doi.org/10.1016/j.physbeh.2015.06.034.

Chambers, J. L., & Wilson, W. T. (1968). Perception of apparent motion and degree of mental pathology. *Perceptual and Motor Skills, 26*(3), 855–861. https://doi.org/10.2466/pms.1968.26.3.855.

Chkonia, E., Roinishvili, M., Reichard, L., Wurch, W., Puhlmann, H., & Grimsen, C. (2012). Patients with functional psychoses show similar visual backward masking deficits. *Psychiatry Research, 198*(2), 235–240. https://doi.org/10.1016/j.psychres.2012.02.020.

Chong, E.W.-T., Kreis, A. J., Wong, T. Y., Simpson, J. A., & Guymer, R. H. (2008). Alcohol Consumption and the Risk of Age-Related Macular Degeneration: A Systematic Review and Meta-Analysis. *American Journal of Ophthalmology, 145*(4), 707–715. https://doi.org/10.1016/j.ajo.2007.12.005.

Ciesielski, K. T., Waldorf, A. V., & Jung, R. E. (1995). Anterior brain deficits in chronic alcoholism. Cause or effect? *The Journal of Nervous and Mental Disease*, *183*(12), 756–761. https://doi.org/10.1097/00005053-199512000-00005.

Clarke, J., & Haughton, H. (1975). A study of intellectual impairment and recovery rates in heavy drinkers in Ireland. *The British Journal of Psychiatry : The Journal of Mental Science, 126,* 178–184. https://doi.org/10.1192/bjp.126.2.178.

Corballis, M. C. (2002). Hemispheric interactions in simple reaction time. *Neuropsychologia, 40*(4), 423–434. https://doi.org/10.1016/s0028-3932(01)00097-5.

Creupelandt, C., D'Hondt, F., & Maurage, P. (2019). Towards a Dynamic Exploration of Vision, Cognition and Emotion in Alcohol-Use Disorders. *Current Neuropharmacology, 17*(6), 492–506. https://doi.org/10.2174/1570159X16666180828100441.

Crowe, S. F., Cammisuli, D. M., & Stranks, E. K. (2019). Widespread Cognitive Deficits in Alcoholism Persistent Following Prolonged Abstinence: An Updated Meta-analysis of Studies That Used Standardised Neuropsychological Assessment Tools. *Archives of Clinical Neuropsychology, 35*(1), 31–35. https://doi.org/10.1093/arclin/acy106.

Cruz-Coke, R. (1972). Defective color vision and alcoholism. *Modern Problems in Ophthalmology, 11,* 174–177.

Cutting, J. (1978). Patterns of performance in amnesic subjects. *Journal of Neurology, Neurosurgery, and Psychiatry, 41*(3), 278–282. https://doi.org/10.1136/jnnp.41.3.278.

Cutting, J. (1979). Differential impairment of memory in Korsakoff's syndrome. *Cortex, 15*(3), 501–506. https://doi.org/10.1016/s0010-9452(79)80074-x.

Czapla, M., Simon, J. J., Richter, B., Kluge, M., Friederich, H.-C., Herpertz, S., et al. (2016). The impact of cognitive impairment and impulsivity on relapse of alcohol-dependent patients: Implications for psychotherapeutic treatment. *Addiction Biology, 21*(4), 873–884. https://doi.org/10.1111/adb.12229.

Daig, I., Mahlberg, R., Schroeder, F., Gudlowski, Y., Wrase, J., Wertenauer, F., Bschor, T., Esser, G., Heinz, A., & Kienast, T. (2010). Low effective organizational strategies in visual memory performance of unmedicated alcoholics during early abstinence. *Psycho-Social Medicine*, *7*. https://doi.org/10.3205/psm000069.

Davies, S. J. C., Pandit, S. A., Feeney, A., Stevenson, B. J., Kerwin, R. W., Nutt, D. J., et al. (2005). Is there cognitive impairment in clinically 'healthy' abstinent alcohol dependence? *Alcohol and Alcoholism, 40*(6), 498–503. https://doi.org/10.1093/alcalc/agh203.

Dawson, L. K., & Grant, I. (2000). Alcoholics' initial organizational and problem-solving skills predict learning and memory performance on the Rey-Osterrieth Complex Figure. *Journal of the International Neuropsychological Society, 6*(1), 12–19. https://doi.org/10.1017/s1355617700611025.

de Oliveira Castro, A. J., Rodrigues, A. R., Côrtes, M. I. T., & de Lima Silveira, L. C. (2009). Impairment of color spatial vision in chronic alcoholism measured by psychophysical methods. *Psychology & Neuroscience, 2*(2), 179–187. https://doi.org/10.3922/j.psns.2009.2.009.

Desfosses, M., Meadows, H., Jackson, M., & Crowe, S. F. (2014). The Relationship Between Neuropsychological Functioning and Mental Health Outcomes of Chronic Alcohol Users Involved in Counselling: Prediction of Treatment Outcome. *Australian Psychologist, 49*(5), 287–296. https://doi.org/10.1111/ap.12071.

D'Hondt, F., Kornreich, C., Philippot, P., Campanella, S., & Maurage, P. (2014a). Below and beyond the recognition of emotional facial expressions in alcohol dependence: from basic perception to social cognition. *Neuropsychiatric Disease and Treatment, 10,* 2177–2182. https://doi.org/10.2147/NDT.S74963.

D'Hondt, F., Lepore, F., & Maurage, P. (2014b). Are visual impairments responsible for emotion decoding deficits in alcohol-dependence? *Frontiers in Human Neuroscience, 8,* 128. https://doi.org/10.3389/fnhum.2014.00128.

D'Hondt, F., Lescut, C., Maurage, P., Menard, O., Gibour, B., & Cottencin, O. (2018). Psychiatric comorbidities associated with a positive screening using the Montreal Cognitive Assessment (MoCA) test in subjects with severe alcohol use disorder. *Drug and Alcohol Dependence, 191,* 266–269. https://doi.org/10.1016/j.drugalcdep.2018.07.009.

Di Sclafani, V., Ezekiel, F., Meyerhoff, D. J., MacKay, S., Dillon, W. P., Weiner, M. W., & Fein, G. (1995). Brain Atrophy and Cognitive Function in Older Abstinent Alcoholic Men. *Alcoholism: Clinical and Experimental Research*, *19*(5), 1121–1126. https://doi.org/10.1111/j.1530-0277.1995.tb01589.x.

Drake, A. I., Hannay, H. J., & Gam, J. (1990). Effects of chronic alcoholism on hemispheric functioning: an examination of gender differences for cognitive and dichotic listening tasks. *Journal of Clinical and Experimental Neuropsychology, 12*(5), 781–797. https://doi.org/10.1080/01688639008401019.

Durazzo, T. C., Mon, A., Gazdzinski, S., & Meyerhoff, D. J. (2017). Regional brain volume changes in alcohol-dependent individuals during early abstinence: associations with relapse following treatment: Brain volumes. *Addiction Biology, 22*(5), 1416–1425. https://doi.org/10.1111/adb.12420.

Durazzo, T. C., Mon, A., Gazdzinski, S., Yeh, P.-H., & Meyerhoff, D. J. (2015). Serial longitudinal magnetic resonance imaging data indicate non-linear regional gray matter volume recovery in abstinent alcohol-dependent individuals. *Addiction Biology, 20*(5), 956–967. https://doi.org/10.1111/adb.12180.

Durazzo, T. C., Pennington, D. L., Schmidt, T. P., Mon, A., Abé, C., & Meyerhoff, D. J. (2013). Neurocognition in 1-Month-Abstinent Treatment-Seeking Alcohol-Dependent Individuals: Interactive Effects of Age and Chronic Cigarette Smoking. *Alcoholism: Clinical and Experimental Research*, *37*(10), 1794–1803. https://doi.org/10.1111/acer.12140.

Eckardt, M. J., Ryback, R. S., & Pautler, C. P. (1980). Neuropsychological deficits in alcoholic men in their mid thirties. *The American Journal of Psychiatry, 137*(8), 932–936. https://doi.org/10.1176/ajp.137.8.932.

Ellenberg, L., Rosenbaum, C., Goldman, M. S., & Whitman, R. D. (1980). Recoverability of psychological functioning following alcohol abuse: lateralization effects. *Journal of Consulting and Clinical Psychology, 48*(4), 503–510. https://doi.org/10.1037//0022-006x.48.4.503.

Ellis, R. J., & Oscar-Berman, M. (1989). Alcoholism, Aging, and Functional Cerebral Asymmetries. *Psychological Bulletin, 106*(1), 128–147. https://doi.org/10.1037/0033-2909.106.1.128.

Emmerson, R. Y., Dustman, R. E., Shearer, D. E., & Chamberlin, H. M. (1987). EEG, visually evoked and event related potentials in young abstinent alcoholics. *Alcohol, 4*(4), 241–248. https://doi.org/10.1016/0741-8329(87)90018-8.

Errico, A. L., King, A. C., Lovallo, W. R., & Parsons, O. A. (2002). Cortisol dysregulation and cognitive impairment in abstinent male alcoholics. *Alcoholism: Clinical and Experimental Research*, *26*(8), 1198–1204. https://doi.org/10.1097/01.ALC.0000025885.23192.FF.

Errico, A. L., Parsons, O. A., Kling, O. R., & King, A. C. (1992). Investigation of the role of sex hormones in alcoholics' visuospatial deficits. *Neuropsychologia, 30*(5), 417–426. https://doi.org/10.1016/0028-3932(92)90089-5.

Evert, D. L., & Oscar-Berman, M. (2001). Selective attentional processing and the right hemisphere: effects of aging and alcoholism. *Neuropsychology, 15*(4), 452–461. https://doi.org/10.1037/0894-4105.15.4.452.

Fabian, M. S., Parsons, O. A., & Sheldon, M. D. (1984). Effects of gender and alcoholism on verbal and visual-spatial learning. *The Journal of Nervous and Mental Disease, 172*(1), 16–20. https://doi.org/10.1097/00005053-198401000-00005.

Fabian, M. S., Parsons, O. A., & Silberstein, J. A. (1981). Impaired perceptual-cognitive functioning in women alcoholics. Cross-validated findings. *Journal of Studies on Alcohol*, *42*(3), 217–229. https://doi.org/10.15288/jsa.1981.42.217.

Fakunle, P. B., Ajibade, A. J., Ehigie, L. O., Alamu, O. A., & Ashamu, E. A. (2012). Chronic Ethanol Administration Reduces Neuronal Cells Population in the Lateral Geniculate Body and the Superior Colliculus of Adult Wistar Rats (Rattus norvegicus). *Research Journal of Medical Sciences, 6*(2), 56–60. https://doi.org/10.3923/rjmsci.2012.56.60.

Fama, R., Pfefferbaum, A., & Sullivan, E. V. (2004). Perceptual Learning in Detoxified Alcoholic Men: Contributions From Explicit Memory. *Executive Function, and Age: Alcoholism: Clinical & Experimental Research, 28*(11), 1657–1665. https://doi.org/10.1097/01.ALC.0000145690.48510.DA.

Fama, R., Pfefferbaum, A., & Sullivan, E. V. (2006). Visuoperceptual learning in alcoholic Korsakoff syndrome. *Alcoholism: Clinical and Experimental Research*, *30*(4), 680–687. https://doi.org/10.1111/j.1530-0277.2006.00085.x.

Fein, G., & McGillivray, S. (2007). Cognitive Performance in Long-Term Abstinent Elderly Alcoholics. *Alcoholism: Clinical and Experimental Research*, *31*(11), 1788–1799. https://doi.org/10.1111/j.1530-0277.2007.00481.x.

Fein, G., Shimotsu, R., Chu, R., & Barakos, J. (2009). Parietal Gray Matter Volume Loss Is Related to Spatial Processing Deficits in Long-Term Abstinent Alcoholic Men. *Alcoholism: Clinical and Experimental Research*, *33*(10), 1806–1814. https://doi.org/10.1111/j.1530-0277.2009.01019.x.

Fein, G., Torres, J., Price, L. J., & Di Sclafani, V. (2006). Cognitive performance in long-term abstinent alcoholic individuals. *Alcoholism: Clinical and Experimental Research*, *30*(9), 1538–1544. https://doi.org/10.1111/j.1530-0277.2006.00185.x.

Ffytche, D. H., Guy, C. N., & Zeki, S. (1995). The parallel visual motion inputs into areas V1 and V5 of human cerebral cortex. *Brain, 118*(6), 1375–1394. https://doi.org/10.1093/brain/118.6.1375.

Fitzhugh, L. C., Fitzhugh, K. B., & Reitan, R. M. (1960). Adaptive abilities and intellectual functioning in hospitalized alcoholics. *Quarterly Journal of Studies on Alcohol, 21,* 414–423.

Fitzhugh, L. C., Fitzhugh, K. B., & Reitan, R. M. (1965). Adaptive abilities and intellectual functioning of hospitalized alcoholics: further considerations. *Quarterly Journal of Studies on Alcohol, 26*(3), 402–411.

Fitzpatrick, L. E., & Crowe, S. F. (2013). Cognitive and Emotional Deficits in Chronic Alcoholics: a Role for the Cerebellum? *The Cerebellum, 12*(4), 520–533. https://doi.org/10.1007/s12311-013-0461-3.

Flannery, B., Fishbein, D., Krupitsky, E., Langevin, D., Verbitskaya, E., Bland, C., Bolla, K., Egorova, V., Bushara, N., Tsoy, M., & Zvartau, E. (2007). Gender differences in neurocognitive functioning among alcohol-dependent Russian patients. *Alcoholism: Clinical and Experimental Research*, *31*(5), 745–754. https://doi.org/10.1111/j.1530-0277.2007.00372.x.

Forsberg, L. K., & Goldman, M. S. (1985). Experience-dependent recovery of visuospatial functioning in older alcoholic persons. *Journal of Abnormal Psychology, 94*(4), 519–529. https://doi.org/10.1037/0021-843X.94.4.519.

Franceschi, M., Truci, G., Comi, G., Lozza, L., Marchettini, P., Galardi, G., & Smirne, S. (1984). Cognitive deficits and their relationship to other neurological complications in chronic alcoholic patients. *Journal of Neurology, Neurosurgery & Psychiatry, 47*(10), 1134–1137. https://doi.org/10.1136/jnnp.47.10.1134.

Friedman, T. W., Robinson, S. R., & Yelland, G. W. (2011). Impaired perceptual judgment at low blood alcohol concentrations. *Alcohol, 45*(7), 711–718. https://doi.org/10.1016/j.alcohol.2010.10.007.

Glenn, S. W., & Parsons, O. A. (1991). Impaired efficiency in female alcoholics' neuropsychological performance. *Journal of Clinical and Experimental Neuropsychology, 13*(6), 895–908. https://doi.org/10.1080/01688639108405106.

Glosser, G., Butters, N., & Kaplan, E. (1977). Visuoperceptual processes in brain damaged patients on the digit symbol substitution test. *The International Journal of Neuroscience, 7*(2), 59–66. https://doi.org/10.3109/00207457709147202.

Goldman, M. S., Williams, D. L., & Klisz, D. K. (1983). Recoverability of psychological functioning following alcohol abuse: Prolonged visual–spatial dysfunction in older alcoholics. *Journal of Consulting and Clinical Psychology, 51*(3), 370. https://doi.org/10.1037//0022-006x.51.3.370.

Goldstein, G., & Chotlos, J. W. (1965). Dependency and brain damage in alcoholics. *Perceptual and Motor Skills, 21*(1), 135–150. https://doi.org/10.2466/pms.1965.21.1.135.

Goldstein, G., Neuringer, C., & Klappersack, B. (1970). Cognitive, perceptual, and motor aspects of field dependency in alcoholics. *The Journal of Genetic Psychology, 117*(2), 253–266. https://doi.org/10.1080/00221325.1970.10532583.

Grant, I., Adams, K. M., & Reed, R. (1984). Aging, abstinence, and medical risk factors in the prediction of neuropsychologic deficit among long-term alcoholics. *Archives of General Psychiatry, 41*(7), 710–718. https://doi.org/10.1001/archpsyc.1984.01790180080010.

Grant, I., Adams, K., & Reed, R. (1979). Normal neuropsychological abilities of alcoholic men in their late thirties. *American Journal of Psychiatry, 136*(7), 1263–1269. https://doi.org/10.1176/ajp.136.10.1263.

Gudeman, H. E., Craine, J. F., Golden, C. J., & McLaughlin, D. (1977). Higher cortical dysfunction associated with long term alcoholism. *The International Journal of Neuroscience, 8*(1), 33–40. https://doi.org/10.3109/00207457709150373.

Ham, H. P., & Parsons, O. A. (1997). Organization of psychological functions in alcoholics and nonalcoholics: a test of the compensatory hypothesis. *Journal of Studies on Alcohol*, *58*(1), 67–74. https://doi.org/10.15288/jsa.1997.58.67.

Hamblin, D. K., Hyer, L. A., Harrison, W. R., & Carson, M. F. (1984). Older alcoholics: profile of decline. *Journal of Clinical Psychology*, *40*(6), 1510–1516. https://doi.org/10.1002/1097-4679(198411)40:61510::aid-jclp22704006423.0.co;2-x.

Harris, C. R., Albaugh, B., Goldman, D., & Enoch, M. A. (2003). Neurocognitive impairment due to chronic alcohol consumption in an American Indian community. *Journal of Studies on Alcohol*, *64*(4), 458–466. https://doi.org/10.15288/jsa.2003.64.458.

Harvey, A. J. (2014). Some effects of alcohol and eye movements on cross-race face learning. *Memory, 22*(8), 1126–1138. https://doi.org/10.1080/09658211.2013.872278.

Harvey, A. J. (2016). When alcohol narrows the field of focal attention. *The Quarterly Journal of Experimental Psychology, 69*(4), 669–677. https://doi.org/10.1080/17470218.2015.1040803.

Haxby, J. V., Lundgren, S. L., & Morley, G. K. (1983). Short-term retention of verbal, visual shape and visuospatial location information in normal and amnesic subjects. *Neuropsychologia, 21*(1), 25–33. https://doi.org/10.1080/17470218.2015.1040803.

Hellige, J. B. (1996). Hemispheric asymmetry for visual information processing. *Acta Neurobiologiae Experimentalis, 56*(1), 485–497.

Hill, S. Y. (1980). Comprehensive assessment of brain dysfunction in alcoholic individuals. *Acta Psychiatrica Scandinavica., 62*(Suppl. 286), 57–75. https://doi.org/10.1111/j.1600-0447.1980.tb08055.x.

Hillbom, M., Muuronen, A., Holm, L., & Hindmarsh, T. (1986). The clinical versus radiological diagnosis of alcoholic cerebellar degeneration. *Journal of the Neurological Sciences, 73*(1), 45–53. https://doi.org/10.1016/0022-510x(86)90062-6.

Hochla, N. A., & Parsons, O. A. (1982). Premature aging in female alcoholics. A neuropsychological study. *The Journal of Nervous and Mental Disease*, *170*(4), 241–245. https://doi.org/10.1097/00005053-198204000-00009.

Hoyer, W. J., Semenec, S. C., & Buchler, N. E. G. (2007). Acute alcohol intoxication impairs controlled search across the visual field. *Journal of Studies on Alcohol and Drugs*, *68*(5), 748–758. https://doi.org/10.15288/jsad.2007.68.748.

Ioime, L., Guglielmo, R., Affini, G. F., Quatrale, M., Martinotti, G., Callea, A., Savi, E., & Janiri, L. (2018). Neuropsychological Performance in Alcohol Dependent Patients: A One-Year Longitudinal Study. *Psychiatry Investigation*, *15*(5), 505–513. https://doi.org/10.30773/pi.2017.09.27.1.

Jacobson, G. R., Pisani, V. D., & Berenbaum, H. L. (1970). Temporal stability of field dependence among hospitalized alcoholics. *Journal of Abnormal Psychology, 76*(1), 10–12. https://doi.org/10.1037/h0029650.

Jeanblanc, J. (2015). Comorbidity Between Psychiatric Diseases and Alcohol Use Disorders: Impact of Adolescent Alcohol Consumption. *Current Addiction Reports, 2*(4), 293–301. https://doi.org/10.1007/s40429-015-0076-5.

Jones, B. M. (1971). Verbal and spatial intelligence in short and long term alcoholics. *The Journal of Nervous and Mental Disease, 153*(4), 292–297. https://doi.org/10.1097/00005053-197110000-00008.

Jones, B. M., Jones, M. K., & Hatcher, E. M. (1980). Cognitive deficits in women alcoholics as a function of gynecological status. *Journal of Studies on Alcohol*, *41*(1), 140–146. https://doi.org/10.15288/jsa.1980.41.140.

Jones, B., & Parsons, O. A. (1971). Impaired abstracting ability in chronic alcoholics. *Archives of General Psychiatry, 24*(1), 71–75. https://doi.org/10.1001/archpsyc.1971.01750070073010.

Jones, B., & Parsons, O. A. (1972). Specific vs generalized deficits of abstracting ability in chronic alcoholics. *Archives of General Psychiatry, 26*(4), 380–384. https://doi.org/10.1001/archpsyc.1972.01750220090017.

Kanthan, G. L., Mitchell, P., Burlutsky, G., & Wang, J. J. (2010). Alcohol Consumption and the Long-Term Incidence of Cataract and Cataract Surgery: The Blue Mountains Eye Study. *American Journal of Ophthalmology, 150*(3), 434–440. https://doi.org/10.1016/j.ajo.2010.04.020.

Kapitany, T., Dietzel, M., Grunberger, J., Frey, R., Koppensteiner, L., Schleifer, G., & Marx, B. (1993). Color vision deficiencies in the course of acute alcohol withdrawal. *Biological Psychiatry, 33*(6), 415–422. https://doi.org/10.1016/0006-3223(93)90169-E.

Kapur, N., & Butters, N. (1977). Visuoperceptive deficits in long-term alcoholics and alcoholics with Korsakoff's psychosis. *Journal of Studies on Alcohol*, *38*(11), 2025–2035. https://doi.org/10.15288/jsa.1977.38.2025.

Karp, S. A., & Konstadt, N. L. (1965). Alcoholism and psychological differentiation: long-range effect of heavy drinking on field dependence. *The Journal of Nervous and Mental Disease, 140*(6), 412–416. https://doi.org/10.1097/00005053-196506000-00003.

Konrad, A., Vucurevic, G., Lorscheider, M., Bernow, N., Thummel, M., & Chai, C. (2012). Broad Disruption of Brain White Matter Microstructure and Relationship with Neuropsychological Performance in Male Patients with Severe Alcohol Dependence. *Alcohol and Alcoholism, 47*(2), 118–126. https://doi.org/10.1093/alcalc/agr157.

Kostandov, E. A., Arsumanov, Y. L., Genkina, O. A., Restchikova, T. N., & Shostakovich, G. S. (1982). The effects of alcohol on hemispheric functional asymmetry. *Journal of Studies on Alcohol*, *43*(5), 411–426. https://doi.org/10.15288/jsa.1982.43.411.

Kramer, J. H., Blusewicz, M. J., Robertson, L. C., & Preston, K. (1989). Effects of chronic alcoholism on perception of hierarchical visual stimuli. *Alcoholism: Clinical and Experimental Research*, *13*(2), 240–245. https://doi.org/10.1111/j.1530-0277.1989.tb00320.x.

Kramer, J. H., Kaplan, E., Blusewicz, M. J., & Preston, K. A. (1991). Visual hierarchical analysis of Block Design configural errors. *Journal of Clinical and Experimental Neuropsychology, 13*(4), 455–465. https://doi.org/10.1080/01688639108401063.

Kreutzer, J., DeLuca, J., & Caplan, B. (Eds.). (2017). Encyclopedia of Clinical Neuropsychology. Springer International Publishing. https://doi.org/10.1007/978-3-319-56782-2.

Kringelbach, M. (2004). The functional neuroanatomy of the human orbitofrontal cortex: evidence from neuroimaging and neuropsychology. *Progress in Neurobiology, 72*(5), 341–372. https://doi.org/10.1016/j.pneurobio.2004.03.006.

Kunchulia, M., Pilz, K. S., & Herzog, M. H. (2012). How alcohol intake affects visual temporal processing. *Vision Research, 66,* 11–16. https://doi.org/10.1016/j.visres.2012.06.010.

Kveraga, K., Boshyan, J., & Bar, M. (2007). Magnocellular Projections as the Trigger of Top-Down Facilitation in Recognition. *Journal of Neuroscience, 27*(48), 13232–13240. https://doi.org/10.1523/jneurosci.3481-07.2007.

Laycock, R., Crewther, D., & Crewther, S. (2008). The advantage in being magnocellular: A few more remarks on attention and the magnocellular system. *Neuroscience & Biobehavioral Reviews, 32*(8), 1409–1415. https://doi.org/10.1016/j.neubiorev.2008.04.008.

Laycock, R., Crewther, S. G., & Crewther, D. P. (2007). A role for the 'magnocellular advantage' in visual impairments in neurodevelopmental and psychiatric disorders. *Neuroscience & Biobehavioral Reviews, 31*(3), 363–376. https://doi.org/10.1016/j.neubiorev.2006.10.003.

Leber, W. R., Jenkins, R. L., & Parsons, O. A. (1981). Recovery of visual-spatial learning and memory in chronic alcoholics. *Journal of Clinical Psychology, 37*(1), 192–197. https://doi.org/10.1002/1097-4679(198101)37:1%3c192::AID-JCLP2270370140%3e3.0.CO;2-M.

Liberati, A., Altman, D. G., Tetzlaff, J., Mulrow, C., Gøtzsche, P. C., Ioannidis, J. P. A., et al. (2009). The PRISMA statement for reporting systematic reviews and meta-analyses of studies that evaluate healthcare interventions: Explanation and elaboration. *The BMJ, 339,*. https://doi.org/10.1136/bmj.b2700.

Livingstone, M., & Hubel, D. (1988). Segregation of form, color, movement, and depth: anatomy, physiology, and perception. *Science, 240*(4853), 740–749. https://doi.org/10.1126/science.3283936.

Mackey, S., Allgaier, N., Chaarani, B., Spechler, P., Orr, C., Bunn, J., Allen, N. B., Alia-Klein, N., Batalla, A., Blaine, S., Brooks, S., Caparelli, E., Chye, Y. Y., Cousijn, J., Dagher, A., Desrivieres, S., Feldstein-Ewing, S., Foxe, J. J., Goldstein, R. Z., ENIGMA Addiction Working Group. (2019). Mega-Analysis of Gray Matter Volume in Substance Dependence: General and Substance-Specific Regional Effects. *The American Journal of Psychiatry*, *176*(2), 119–128. https://doi.org/10.1176/appi.ajp.2018.17040415.

Mann, K., Günther, A., Stetter, F., & Ackermann, K. (1999). Rapid recovery from cognitive deficits in abstinent alcoholics: a controlled test-retest study. *Alcohol and Alcoholism, 34*(4), 567–574. https://doi.org/10.1093/alcalc/34.4.567.

Markowitz, S. N. (2006). Principles of modern low vision rehabilitation. *Canadian Journal of Ophthalmology, 41*(3), 289–312. https://doi.org/10.1139/I06-027.

Martins, I. C. V. D. S., Souza, G. D. S., Brasil, A., Herculano, A. M., Lacerda, E. M. D. C. B., Rodrigues, A. R., Rosa, A. A. M., Ventura, D. F., Castro, A. J. D. O., & Silveira, L. C. D. L. (2019). Psychophysical Evaluation of Visual Functions of Ex-Alcoholic Subjects After Prolonged Abstinence. *Frontiers in Neuroscience*, *13*, 179. https://doi.org/10.3389/fnins.2019.00179.

Mergler, D., Blain, L., Lemaire, J., & Lalande, F. (1988). Colour vision impairment and alcohol consumption. *Neurotoxicology and Teratology, 10*(3), 255–260. https://doi.org/10.1016/0892-0362(88)90025-6.

Merigan, W. H., & Maunsell, J. H. (1993). How parallel are the primate visual pathways? *Annual Review of Neuroscience, 16*(1), 369–402. https://doi.org/10.1146/annurev.ne.16.030193.002101.

Miglioli, M., Buchtel, H. A., Campanini, T., & De Risio, C. (1979). Cerebral hemispheric lateralization of cognitive deficits due to alcoholism. *The Journal of Nervous and Mental Disease, 167*(4), 212–217. https://doi.org/10.1097/00005053-197904000-00003.

Molina, J. A., Bermejo, F., del Ser, T., Jimenez-Jimenez, F. J., Herranz, A., Fernandez-Calle, P., & Sainz, M. J. (1994). Alcoholic cognitive deterioration and nutritional deficiencies. *Acta Neurologica Scandinavica, 89*(5), 384–390. https://doi.org/10.1111/j.1600-0404.1994.tb02651.x.

Moriyama, Y., Mimura, M., Kato, M., Yoshino, A., Hara, T., Kashima, H., Watanabe, A. (2002). Executive dysfunction and clinical outcome in chronic alcoholics. *Alcoholism: Clinical and Experimental Research*, *26*(8), 1239–1244. https://doi.org/10.1097/01.ALC.0000026103.08053.86.

Moselhy, H. F., Georgiou, G., & Kahn, A. (2001). Frontal lobe changes in alcoholism: a review of the literature. *Alcohol and Alcoholism, 36*(5), 357–368. https://doi.org/10.1093/alcalc/36.5.357.

Mulhauser, K., Weinstock, J., Ruppert, P., & Benwarec, J. (2018). Changes in Neuropsychological Status during the Initial Phase of Abstinence in Alcohol Use Disorder: Neurocognitive Impairment and Implications for Clinical Care. *Substance Use & Misuse, 53*(6), 881–890. https://doi.org/10.1080/10826084.2017.1408328.

Müller-Oehring, E. M., Jung, Y.-C., Pfefferbaum, A., Sullivan, E. V., & Schulte, T. (2015). The Resting Brain of Alcoholics. *Cerebral Cortex, 25*(11), 4155–4168. https://doi.org/10.1093/cercor/bhu134.

Müller-Oehring, E. M., Schulte, T., Fama, R., Pfefferbaum, A., & Sullivan, E. V. (2009). Global-local interference is related to callosal compromise in alcoholism: a behavior-DTI association study. *Alcoholism: Clinical and Experimental Research*, *33*(3), 477–489.

Munro, C. A., Saxton, J., & Butters, M. A. (2000). The neuropsychological consequences of abstinence among older alcoholics: a cross-sectional study. *Alcoholism: Clinical and Experimental Research*, *24*(10), 1510–1516. https://doi.org/10.1111/j.1530-0277.2000.tb04569.x.

Navon, D. (1977). Forest before trees: The precedence of global features in visual perception. *Cognitive Psychology, 9*(3), 353–383. https://doi.org/10.1016/0010-0285(77)90012-3.

Nawrot, M., Nordenstrom, B., & Olson, A. (2004). Disruption of eye movements by ethanol intoxication affects perception of depth from motion parallax. *Psychological Science, 15*(12), 858–865. https://doi.org/10.1111/j.0956-7976.2004.00767.x.

Nelson, T. M., Sinha, B. K., & Olson, W. M. (1977). Short-term memory for hue in chronic alcoholics. *The British Journal of Addiction to Alcohol and Other Drugs, 72*(4), 301–307. https://doi.org/10.1111/j.1360-0443.1977.tb00695.x.

Nelson, T. M., & Swartz, P. (1971). Perceptual conflict and alcoholics. *Perceptual and Motor Skills, 33*(3), 1023–1028. https://doi.org/10.2466/pms.1971.33.3f.1023.

Newen, A., & Vetter, P. (2017). Why cognitive penetration of our perceptual experience is still the most plausible account. *Consciousness and Cognition, 47,* 26–37. https://doi.org/10.1016/j.concog.2016.09.005.

National Highway Traffic Safety Administration. (2000). *A Review of the Literature on the Effects of Low Doses of Alcohol on Driving-Related* Skills. U.S. Department of Transportation. https://one.nhtsa.gov/people/injury/research/pub/Hs809028/index_.htm.

National Heart, Lung, and Blood Institute. (2014). *Quality Assessment Tool for Observational Cohort and Cross-Sectional Studies*. U.S. National Institutes of Health. http://www.nhlbi.nih.gov/health-pro/guidelines/in-develop/cardiovascular-risk-reduction/tools/cohort.

Nicolás, J. M., Catafau, A. M., Estruch, R., Lomena, F. J., Salamero, M., & Herranz, R. (1993). Regional cerebral blood flow-SPECT in chronic alcoholism: relation to neuropsychological testing. *Journal of Nuclear Medicine, 34*(9), 1452–1459.

Nicolás, J. M., Estruch, R., Salamero, M., Orteu, N., Fernandez-Solà, J., Sacanella, E., & Urbano-Márquez, A. (1997). Brain impairment in well-nourished chronic alcoholics is related to ethanol intake. *Annals of Neurology, 41*(5), 590–598. https://doi.org/10.1002/ana.410410507.

Nixon, S. J., Kujawski, A., Parsons, O. A., & Yohman, J. R. (1987). Semantic (verbal) and figural memory impairment in alcoholics. *Journal of Clinical and Experimental Neuropsychology, 9*(4), 311–322. https://doi.org/10.1080/01688638708405053.

Noonberg, A., Goldstein, G., & Page, H. A. (1985). Premature aging in male alcoholics: "Accelerated aging" or "increased vulnerability"? *Alcoholism: Clinical and Experimental Research*, *9*(4), 334–338. https://doi.org/10.1111/j.1530-0277.1985.tb05555.x.

Norton, D. J., McBain, R. K., Pizzagalli, D. A., Cronin-Golomb, A., & Chen, Y. (2016). Dysregulation of visual motion inhibition in major depression. *Psychiatry Research, 240,* 214–221. https://doi.org/10.1016/j.psychres.2016.04.028.

Obaldia, R. D., Leber, W. R., & Parsons, O. A. (1981). Assessment of neuropsychological functions in chronic alcoholics using a standardized version of Luria's Neuropsychological Technique. *International Journal of Neuroscience, 14*(1–2), 85–93. https://doi.org/10.3109/00207458108985819.

O'Callaghan, C., Kveraga, K., Shine, J. M., Adams, R. B., & Bar, M. (2017). Predictions penetrate perception: Converging insights from brain, behaviour and disorder. *Consciousness and Cognition, 47,* 63–74. https://doi.org/10.1016/j.concog.2016.05.003.

Orssaud, C., Roche, O., & Dufier, J. L. (2007). Nutritional optic neuropathies. *Journal of the Neurological Sciences, 262*(1–2), 158–164.

Oscar-Berman, M., & Bonner, R. T. (1985). Matching- and delayed matching-to-sample performance as measures of visual processing, selective attention, and memory in aging and alcoholic individuals. *Neuropsychologia, 23*(5), 639–651. https://doi.org/10.1016/0028-3932(85)90065-x.

Oscar-Berman, M., Goodglass, H., & Cherlow, D. G. (1973). Perceptual laterality and iconic recognition of visual materials by Karsakoff patients and normal adults. *Journal of Comparative and Physiological Psychology, 82*(2), 316–321. https://doi.org/10.1037/h0034094.

Oscar-Berman, M., & Schendan, H. E. (2000). Asymmetries of Brain Function in Alcoholism: Relationship to Aging. In L. T. Connor & L. K. Obler (Eds.), *Neurobehavior of Langage and Cognition: Studies of Normal Aging and Brain Damage* (pp. 213–240). Kluwer Academic Pub. https://pearl.plymouth.ac.uk/handle/10026.1/1203.

Oscar-Berman, M., Valmas, M. M., Sawyer, K. S., Ruiz, S. M., Luhar, R. B., & Gravitz, Z. R. (2014). Profiles of impaired, spared, and recovered neuropsychologic processes in alcoholism. In E. V. Sullivan & A. Pfefferbaum (Eds.), *Handbook of Clinical Neurology* (Vol. 125, pp. 183–210). Elsevier B. V. https://doi.org/10.1016/B978-0-444-62619-6.00012-4.

Oscar-Berman, M., & Weinstein, A. (1985). Visual Processing, Memory, and Lateralization in Alcoholism and Aging. *Developmental Neuropsychology, 1*(2), 99–112. https://doi.org/10.1080/87565648509540303.

Parsons, O. A., Schaeffer, K. W., & Glenn, S. W. (1990a). Does neuropsychological test performance predict resumption of drinking in posttreatment alcoholics? *Addictive Behaviors, 15*(3), 297–307. https://doi.org/10.1016/0306-4603(90)90073-7.

Parsons, O. A., Sinha, R., & Williams, H. L. (1990b). Relationships between neuropsychological test performance and event-related potentials in alcoholic and nonalcoholic samples. *Alcoholism: Clinical and Experimental Research*, *14*(5), 746–755. https://doi.org/10.1111/j.1530-0277.1990.tb01239.x.

Pawlosky, R. J., Bacher, J., & Salem, N. J. (2001). Ethanol consumption alters electroretinograms and depletes neural tissues of docosahexaenoic acid in rhesus monkeys: nutritional consequences of a low n-3 fatty acid diet. *Alcoholism, Clinical and Experimental Research, 25*(12), 1758–1765. https://doi.org/10.1111/j.1530-0277.2001.tb02187.x.

Pearson, P., & Timney, B. (1998). Effects of moderate blood alcohol concentrations on spatial and temporal contrast sensitivity. *Journal of Studies on Alcohol*, *59*(2), 163–173. https://doi.org/10.15288/jsa.1998.59.163.

Peters, J. C., van den Boomen, C., & Kemner, C. (2017). Spatial Frequency Training Modulates Neural Face Processing: Learning Transfers from Low- to High-Level Visual Features. *Frontiers in Human Neuroscience, 11,* 1. https://doi.org/10.3389/fnhum.2017.00001.

Pfefferbaum, A., Sullivan, E. V., Mathalon, D. H., & Lim, K. O. (1997). Frontal lobe volume loss observed with magnetic resonance imaging in older chronic alcoholics. *Alcoholism: Clinical and Experimental Research*, *21*(3), 521–529. https://doi.org/10.1111/j.1530-0277.1997.tb03798.x.

Pillunat, L. E., Christ, T., Luderer, H. J., & Stodtmeister, R. (1985). Flicker fusion frequency and organic syndrome in alcoholics. *Perceptual and Motor Skills, 60*(2), 487–494. https://doi.org/10.2466/pms.1985.60.2.487.

Pitel, A.-L., Lannuzel, C., Viader, F., Vabret, F., Eustache, F., & Beaunieux, H. (2013). Alcoolo-dépendance et syndrome de Korsakoff: un continuum? *Revue de neuropsychologie, 5*(3), 179–186. https://doi.org/10.1684/nrp.2013.0270.

Plant, G. T., & Perry, V. H. (1990). The anatomical basis of the caecocentral scotoma. *Brain, 113*(5), 1441–1457. https://doi.org/10.1093/brain/113.5.1441.

Prakash, J., Ryali, V., Srivastava, K., Bhat, P. S., Shashikumar, R., & Singal, A. (2011). Tobacco-alcohol amblyopia: A rare complication of prolonged alcohol abuse. *Industrial Psychiatry Journal, 20*(1), 66–68. https://doi.org/10.4103/0972-6748.98424.

Quaglino, V., De Wever, E., & Maurage, P. (2015). Relations Between Cognitive Abilities, Drinking Characteristics, and Emotional Recognition in Alcohol Dependence: A Preliminary Exploration. *Alcoholism: Clinical and Experimental Research*, *39*(10), 2032–2038. https://doi.org/10.1111/acer.12841.

Rando, K., Hong, K.-I., Bhagwagar, Z., Li, C.-S.R., Bergquist, K., Guarnaccia, J., & Sinha, R. (2011). Association of Frontal and Posterior Cortical Gray Matter Volume With Time to Alcohol Relapse: A Prospective Study. *The American Journal of Psychiatry, 168*(2), 183–192. https://doi.org/10.1176/appi.ajp.2010.10020233.

Reed, R. J., Grant, I., & Rourke, S. B. (1992). Long-term abstinent alcoholics have normal memory. *Alcoholism: Clinical and Experimental Research*, *16*(4), 677–683. https://doi.org/10.1111/j.1530-0277.1992.tb00660.x.

Reitan, R. M. (1967). Psychologic changes associated with aging and with cerebral damage. *Mayo Clinic Proceedings, 42*(10), 653–673.

Reshchikova, T. N. (1985). Interhemispheric relations in patients with chronic alcoholism. *Neuroscience and Behavioral Physiology, 15*(2), 161–164. https://doi.org/10.1007/BF0118698.

Reynolds, D. C. (1979). A visual profile of the alcoholic driver. *American Journal of Optometry and Physiological Optics, 56*(4), 241–251. https://doi.org/10.1097/00006324-197904000-00005.

Riege, W. (1977). Inconstant nonverbal recognition memory in Korsakoff patients and controls. *Neuropsychologia, 15*(2), 269–276. https://doi.org/10.1016/0028-3932(77)90035-5.

Ritz, L., Segobin, S., Le Berre, A. P., Lannuzel, C., Boudehent, C., & Vabret, F. (2014). Brain structural substrates of cognitive procedural learning in alcoholic patients early in abstinence. *Alcoholism, Clinical and Experimental Research, 38*(8), 2208–2216. https://doi.org/10.1111/acer.12486.

Roberts, L. A., & Bauer, L. O. (1993). Reaction time during cocaine versus alcohol withdrawal: longitudinal measures of visual and auditory suppression. *Psychiatry Research, 46*(3), 229–237. https://doi.org/10.1016/0165-1781(93)90091-T.

Robertson, C. E., & Baron-Cohen, S. (2017). Sensory perception in autism. *Nature Reviews. Neuroscience, 18*(11), 671–684. https://doi.org/10.1038/nrn.2017.112.

Robertson, L. C., Stillman, R., & Delis, D. C. (1985). The effect of alcohol abuse on perceptual reference frames. *Neuropsychologia, 23*(1), 69–76. https://doi.org/10.1016/0028-3932(85)90045-4.

Roquelaure, Y., Le Gargasson, J. F., Kupper, S., Girre, C., Hispard, E., & Dally, S. (1995). Alcohol consumption and visual contrast sensitivity. *Alcohol and Alcoholism, 30*(5), 681–685. https://doi.org/10.1093/oxfordjournals.alcalc.a045781.

Rosenbloom, M. J., Sassoon, S. A., Pfefferbaum, A., & Sullivan, E. V. (2009). Contribution of Regional White Matter Integrity to Visuospatial Construction Accuracy, Organizational Strategy, and Memory for a Complex Figure in Abstinent Alcoholics. *Brain Imaging and Behavior, 3*(4), 379–390. https://doi.org/10.1007/s11682-009-9080-5.

Rupp, C. I., Derntl, B., Osthaus, F., Kemmler, G., & Fleischhacker, W. W. (2017). Impact of Social Cognition on Alcohol Dependence Treatment Outcome: Poorer Facial Emotion Recognition Predicts Relapse/Dropout. *Alcoholism: Clinical and Experimental Research, 41*(12), 2197–2206. https://doi.org/10.1111/acer.13522.

Ryan, C., & Butters, N. (1980). Learning and memory impairments in young and old alcoholics: Evidence for the premature-aging hypothesis. *Alcoholism: Clinical and Experimental Research*, *4*(3), 288–293. https://doi.org/10.1111/j.1530-0277.1980.tb04816.x.

Ryan, C., & Butters, N. (1984). Alcohol consumption and premature aging. A critical review. In M. Galanter (Ed.), *Recent Developments in Alcoholism* (Vol. 2, pp. 223–250). Springer.

Samuelson, K. W., Neylan, T. C., Metzler, T. J., Lenoci, M., Rothlind, J., & Henn-Haase, C. (2006). Neuropsychological

functioning in posttraumatic stress disorder and alcohol abuse. *Neuropsychology, 20*(6), 716–726. https://doi.org/10.1037/0894-4105.20.6.716.

Sancho-Tello, M., Muriach, M., Barcia, J., Bosch-Morell, F., Genovés, J. M., & Johnsen-Soriano, S. (2008). Chronic alcohol feeding induces biochemical, histological, and functional alterations in rat retina. *Alcohol and Alcoholism, 43*(3), 254–260. https://doi.org/10.1093/alcalc/agn006.

Sassoon, S. A., Fama, R., Rosenbloom, M. J., O'Reilly, A., Pfefferbaum, A., & Sullivan, E. V. (2007). Component cognitive and motor processes of the digit symbol test: differential deficits in alcoholism, HIV infection, and their comorbidity. *Alcoholism: Clinical and Experimental Research*, *31*(8), 1315–1324. https://doi.org/10.1111/j.1530-0277.2007.00426.x.

Schandler, S. L., Brannock, J. C., Cohen, M. J., Antick, J., & Caine, K. (1988). Visuospatial learning in elementary school children with and without a family history of alcoholism. *Journal of Studies on Alcohol*, *49*(6), 538–545. https://doi.org/10.15288/jsa.1988.49.538.

Schmahmann, J. D., & Pandya, D. N. (2007). The Complex History of the Fronto-Occipital Fasciculus. *Journal of the History of the Neurosciences, 16*(4), 362–377. https://doi.org/10.1080/09647040600620468.

Schneider, U., Dietrich, D. E., Sternemann, U., Seeland, I., Gielsdorf, D., & Huber, T. J. (1998). Reduced binocular depth inversion in patients with alcoholism. *Alcohol and Alcoholism, 33*(2), 168–172. https://doi.org/10.1093/oxfordjournals.alcalc.a008374.

Schneider, U., Leweke, F. M., Niemcyzk, W., Sternemann, U., Bevilacqua, M., & Emrich, H. M. (1996a). Impaired binocular depth inversion in patients with alcohol withdrawal. *Journal of Psychiatric Research, 30*(6), 469–474. https://doi.org/10.1016/s0022-3956(96)00031-3.

Schneider, U., Leweke, F. M., Sternemann, U., Weber, M. M., & Emrich, H. M. (1996b). Visual 3D illusion: a systems-theoretical approach to psychosis. *European Archives of Psychiatry and Clinical Neuroscience, 246*(5), 256–260. https://doi.org/10.1007/bf02190277.

Schulte, T., Muller-Oehring, E. M., Rohlfing, T., Pfefferbaum, A., & Sullivan, E. V. (2010). White Matter Fiber Degradation Attenuates Hemispheric Asymmetry When Integrating Visuomotor Information. *Journal of Neuroscience, 30*(36), 12168–12178. https://doi.org/10.1523/jneurosci.2160-10.2010.

Schulte, T., Müller-Oehring, E. M., Salo, R., Pfefferbaum, A., & Sullivan, E. V. (2006). Callosal involvement in a lateralized stroop task in alcoholic and healthy subjects. *Neuropsychology, 20*(6), 727–736. https://doi.org/10.1037/0894-4105.20.6.727.

Schulte, T., Pfefferbaum, A., & Sullivan, E. V. (2004). Parallel interhemispheric processing in aging and alcoholism: relation to corpus callosum size. *Neuropsychologia, 42*(2), 257–271. https://doi.org/10.1016/S0028-3932(03)00155-6.

Schulte-Körne, G., & Bruder, J. (2010). Clinical neurophysiology of visual and auditory processing in dyslexia: A review. *Clinical Neurophysiology, 121*(11), 1794–1809. https://doi.org/10.1016/j.clinph.2010.04.028.

Schweizer, T. A., Vogel-Sprott, M., Danckert, J., Roy, E. A., Skakum, A., & Broderick, C. E. (2006). Neuropsychological profile of acute alcohol intoxication during ascending and descending blood alcohol concentrations. *Neuropsychopharmacology, 31*(6), 1301–1309. https://doi.org/10.1038/sj.npp.1300941.

Selby, M. J., & Azrin, R. L. (1998). Neuropsychological functioning in drug abusers. *Drug and Alcohol Dependence, 50*(1), 39–45. https://doi.org/10.1016/S0376-8716(98)00002-7.

Serý, O., Sťastný, F., Zvolský, P., Hlinomazová, Z., & Balcar, V. J. (2011). Association between Val66Met polymorphism of Brain-Derived Neurotrophic Factor (BDNF) gene and a deficiency of colour vision in alcohol-dependent male patients. *Neuroscience Letters, 499*(3), 154–157. https://doi.org/10.1016/j.neulet.2011.05.038.

Sharma, P., & Sharma, R. (2011). Toxic optic neuropathy. *Indian Journal of Ophthalmology, 59*(2), 137–141. https://doi.org/10.4103/0301-4738.77035.

Shelton, M. D., Parsons, O. A., & Leber, W. R. (1984). Verbal and visuospatial performance in male alcoholics: a test of the premature-aging hypothesis. *Journal of Consulting and Clinical Psychology, 52*(2), 200–206. https://doi.org/10.1037/0022-006X.52.2.200.

Silberstein, J. A., & Parsons, O. A. (1979). Neuropsychological impairment in female alcoholics. *Currents in Alcoholism, 7,* 481–495.

Silberstein, J. A., & Parsons, O. A. (1981). Neuropsychological impairment in female alcoholics: Replication and extension. *Journal of Abnormal Psychology, 90*(2), 179–182. https://doi.org/10.1037/0021-843X.90.2.179.

Smith, J. W. (1972). Color vision in alcoholics. *Annals of the New York Academy of Sciences, 197,* 143–147.

Smith, J. W., & Layden, T. A. (1971). Color vision defects in alcoholism. II. *The British Journal of Addiction to Alcohol and Other Drugs*, *66*(1), 31–37. https://doi.org/10.1111/j.1360-0443.1971.tb02363.x.

Snortum, J. R. (1965). Performance of different diagnostic groups on the tachistoscopic and copy phases of the Bender-Gestalt. *Journal of Consulting Psychology, 29*(4), 345–351. https://doi.org/10.1037/h0022406.

Sparadseo, F. R., Zwick, W., & Butters, N. (1983). Cognitive functioning of alcoholic females: an exploratory study. *Drug and Alcohol Dependence, 12*(2), 143–150. https://doi.org/10.1016/0376-8716(83)90038-8.

Spinazzi, M. (2019). Optic Neuropathies Caused by Micronutrients Deficiencies. In V. R. Preedy & R. R. Watson (Eds.), *Handbook of Nutrition, Diet, and the Eye (Second Edition)* (pp. 441–460). Academic Press. https://doi.org/10.1016/B978-0-12-815245-4.00027-2.

Squeglia, L. M., Jacobus, J., & Tapert, S. F. (2014a). The effect of alcohol use on human adolescent brain structures and systems. In *Handbook of Clinical Neurology* (Vol. 125, pp. 501–510). Elsevier. http://linkinghub.elsevier.com/retrieve/pii/B9780444626196000288.

Squeglia, L. M., Rinker, D. A., Bartsch, H., Castro, N., Chung, Y., & Dale, A. M. (2014b). Brain Volume Reductions in Adolescent Heavy Drinkers. *Developmental Cognitive Neuroscience, 9,* 117–125. https://doi.org/10.1016/j.dcn.2014.02.005.

Stavro, K., Pelletier, J., & Potvin, S. (2013). Widespread and sustained cognitive deficits in alcoholism: a meta-analysis. *Addiction Biology, 18*(2), 203–213. https://doi.org/10.1111/j.1369-1600.2011.00418.x.

Steiger, H., Negrete, J. C., & Marcil, G. (1985). Field dependence in alcoholics: relation to years of drinking, severity of alcohol dependence and emotional distress. *Journal of Studies on Alcohol*, *46*(6), 486–489. https://doi.org/10.15288/jsa.1985.46.486.

Sullivan, E. V., Fama, R., Rosenbloom, M. J., & Pfefferbaum, A. (2002). A profile of neuropsychological deficits in alcoholic women. *Neuropsychology, 16*(1), 74–83. https://doi.org/10.1037//0894-4105.16.1.74.

Sullivan, E. V., Harris, R. A., & Pfefferbaum, A. (2010). Alcohol's effects on brain and behavior. *Alcohol Research & Health, 33*(1–2), 127–143.

Sullivan, E. V., Mathalon, D. H., Ha, C. N., Zipursky, R. B., & Pfefferbaum, A. (1992). The contribution of constructional accuracy and organizational strategy to nonverbal recall in schizophrenia and chronic alcoholism. *Biological Psychiatry, 32*(4), 312–333. https://doi.org/10.1016/0006-3223(92)90036-y.

Sullivan, E. V., Rosenbloom, M. J., & Pfefferbaum, A. (2000). Pattern of Motor and Cognitive Deficits in Detoxified Alcoholic Men. *Alcoholism: Clinical and Experimental Research*, *24*(5), 611–621. https://doi.org/10.1111/j.1530-0277.2000.tb02032.x.

Sullivan, E. V., Zahr, N. M., Sassoon, S. A., Thompson, W. K., Kwon, D., Pohl, K. M., & Pfefferbaum, A. (2018). The Role of Aging, Drug Dependence, and Hepatitis C Comorbidity in Alcoholism Cortical Compromise. *JAMA Psychiatry, 75*(5), 474–483. https://doi.org/10.1001/jamapsychiatry.2018.0021.

Templer, D. I., Ruff, C. F., & Simpson, K. (1975). Trail Making Test Performance of Alcoholics Abstinent at Least a Year. *International Journal of the Addictions, 10*(4), 609–612. https://doi.org/10.3109/10826087509026738.

Thomas, C., Kveraga, K., Huberle, E., Karnath, H.-O., & Bar, M. (2012). Enabling global processing in simultanagnosia by psychophysical biasing of visual pathways. *Brain, 135*(5), 1578–1585. https://doi.org/10.1093/brain/aws066.

Tivis, R., Beatty, W. W., Nixon, S. J., & Parsons, O. A. (1995). Patterns of cognitive impairment among alcoholics: are there subtypes? *Alcoholism: Clinical and Experimental Research*, *19*(2), 496–500. https://doi.org/10.1111/j.1530-0277.1995.tb01537.x.

Tsagareli, M. G. (1995). The interhemispheric functional organization on human visuo-spatial perception. *NeuroReport, 6*(6), 925–928. https://doi.org/10.1097/00001756-199504190-00025.

Tsermentseli, S., O'Brien, J. M., & Spencer, J. V. (2008). Comparison of Form and Motion Coherence Processing in Autistic Spectrum Disorders and Dyslexia. *Journal of Autism and Developmental Disorders, 38*(7), 1201–1210. https://doi.org/10.1007/s10803-007-0500-3.

Tyler, C. W. (1985). Analysis of visual modulation sensitivity. II. Peripheral retina and the role of photoreceptor dimensions. *Journal of the Optical Society of America A*, *2*(3), 393–398. https://doi.org/10.1364/josaa.2.000393.

Tyler, C. W. (1987). Analysis of visual modulation sensitivity. III. Meridional variations in peripheral flicker sensitivity. *Journal of the Optical Society of America A*, *4*(8), 1612–1619. https://doi.org/10.1364/JOSAA.4.001612.

Uekermann, J., Daum, I., Schlebusch, P., Wiebel, B., & Trenckmann, U. (2003). Depression and cognitive functioning in alcoholism. *Addiction, 98*(11), 1521–1529. https://doi.org/10.1046/j.1360-0443.2003.00526.x.

Ugarte, G., Cruz-Coke, R., Rivera, L., Altschiller, H., & Mardones, J. (1970). Relationship of color blindness to alcoholic liver damage. *Pharmacology, 4*(5), 297–308. https://doi.org/10.1159/000136150.

Varela, A., Rivera, L., Mardones, J., & Cruz-Coke, R. (1969). Color vision defects in non-alcoholic relatives of alcoholic patients. *The British Journal of Addiction to Alcohol and Other Drugs, 64*(1), 67–73. https://doi.org/10.1111/j.1360-0443.1969.tb01111.x.

Verriest, G., Francq, P., & Pierart, P. (1980). Results of colour vision tests in alcoholic and in mentally disordered subjects. *Ophthalmologica, 180*(5), 247–256. https://doi.org/10.1159/000308982.

Vivian, T. N., Goldstein, G., & Shelly, C. (1973). Reaction time and motor speed in chronic alcoholics. *Perceptual and Motor Skills, 36*(1), 136–138. https://doi.org/10.2466/pms.1973.36.1.136.

Wang, J., Fan, Y., Dong, Y., Ma, M., Dong, Y., & Niu, Y. (2018). Combining gray matter volume in the cuneus and the cuneus-prefrontal connectivity may predict early relapse in abstinent alcohol-dependent patients. *PLoS ONE, 13*(5), e0196860. https://doi.org/10.1371/journal.pone.0196860.

Wang, S., Wang, J. J., & Wong, T. Y. (2008). Alcohol and Eye Diseases. *Survey of Ophthalmology, 53*(5), 512–525. https://doi.org/10.1016/j.survophthal.2008.06.003.

Wegner, A. J., Gunthner, A., & Fahle, M. (2001). Visual performance and recovery in recently detoxified alcoholics. *Alcohol and Alcoholism, 36*(2), 171–179. https://doi.org/10.1093/alcalc/36.2.171.

Whipple, S. C., Parker, E. S., & Noble, E. P. (1988). An atypical neurocognitive profile in alcoholic fathers and their sons. *Journal of Studies on Alcohol*, *49*(3), 240–244. https://doi.org/10.15288/jsa.1988.49.240.

Wilcox, C. E., Dekonenko, C. J., Mayer, A. R., Bogenschutz, M. P., & Turner, J. A. (2014). Cognitive control in alcohol use disorder: Deficits and clinical relevance. *Reviews in the Neurosciences, 25*(1), 1–24. https://doi.org/10.1515/revneuro-2013-0054.

Williams, D. E. (1984). Visual electrophysiology and psychophysics in chronic alcoholics and in patients on tuberculostatic chemotherapy. *American Journal of Optometry and Physiological Optics, 61*(9), 576–585. https://doi.org/10.1097/00006324-198409000-00007.

Wilson, B., Kolb, B., Odland, L., & Wishaw, I. Q. (1987). Alcohol, sex, age, and the hippocampus. *Psychobiology, 15*(4), 300–307. https://doi.org/10.3758/BF03327286.

Wilson, J. T. L., Wiedmann, K. D., Phillips, W. A., & Brooks, D. N. (1988). Visual Event Perception in Alcoholics. *Journal of Clinical and Experimental Neuropsychology, 10*(2), 222–234. https://doi.org/10.1080/01688638808408237.

Witkin, H. A. (1960). The problem of individuality in development. In B. Kaplan & S. Wapner (Eds.), *Perspectives in psychological theory* (pp. 335–361). International Universities Press.

Witkin, H. A., & Asch, S. E. (1948). Studies in space orientation. IV. Further experiments on perception of the upright with displaced visual fields. *Journal of Experimental Psychology*, *38*(6), 762–782. https://doi.org/10.1037/h0053671.

Witkin, H. A., Oltman, P. K., Raskin, E., & Karp, S. A. (1971). *A manual for the Embedded Figures Test*. Consulting Psychologist Press.

Wittchen, H. U., Jacobi, F., Rehm, J., Gustavsson, A., Svensson, M., & Jönsson, B. (2011). The size and burden of mental disorders and other disorders of the brain in Europe 2010. *European Neuropsychopharmacology, 21*(9), 655–679. https://doi.org/10.1016/j.euroneuro.2011.07.018.

World Health Organization. (2004). *International statistical classification of diseases and related health problems* (10th ed.). http://apps.who.int/classifications/apps/icd/icd10online2004/fr-icd.htm.

World Health Organization. (2019). *International statistical classification of diseases and related health problems* (11th ed.). https://icd.who.int/.

Yohman, J. R., Parsons, O. A., & Leber, W. R. (1985). Lack of recovery in male alcoholics' neuropsychological performance one year after treatment. *Alcoholism: Clinical and Experimental Research*, *9*(2), 114–117. https://doi.org/10.1111/j.1530-0277.1985.tb05530.x.

Zahr, N. M. (2014). Chapter 17 - Structural and microstructral imaging of the brain in alcohol use disorders. In E. V. S. and A. Pfefferbaum (Ed.), *Handbook of Clinical Neurology* (Vol. 125, pp. 275–290). Elsevier. https://doi.org/10.1016/B978-0-444-62619-6.00017-3.

Zhang, X. L., Begleiter, H., & Projesz, B. (1997). Is working memory intact in alcoholics? An ERP study. *Psychiatry Research*, *75*(2), 75–89. https://doi.org/10.1016/S0925-4927(97)00043-7

Zhu, J., Wang, Y., Wang, H., Cheng, W., Li, Z., & Qian, Y. (2018). Abnormal gray matter asymmetry in alcohol dependence. *NeuroReport, 29*(9), 753–759. https://doi.org/10.1097/WNR.0000000000001027.

Zhuang, X., King, A., McNamara, P., Pokorny, J., & Cao, D. (2012). Differential effects of alcohol on contrast processing mediated by the magnocellular and parvocellular pathways. *Journal of Vision, 12*(11), 16. https://doi.org/10.1167/12.11.16.

Zinn, S., Stein, R., & Swartzwelder, H. S. (2004). Executive Functioning Early in Abstinence From Alcohol. *Alcoholism: Clinical & Experimental Research, 28*(9), 1338–1346. https://doi.org/10.1097/01.ALC.0000139814.81811.62.