Sensory sensitivity after acquired brain injury:

a systematic review

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Conflicts of interests statement

The authors have no conflicts of interest to declare that are relevant to the content of this article.

Availability of data and material

This review was not pre-registered. The data collection forms and the study protocol will be made publicly available via Figshare (https://doi.org/10.6084/m9.figshare.14785293).

Authors contributions

Hella Thielen: conceptualization, data curation, funding acquisition, investigation, methodology, project administration, validation, visualization, writing – original draft, writing – review & editing; Nora Tuts: investigation, writing – review & editing; Lies Welkenhuyzen: investigation, writing – review & editing; Irene Huenges Wajer: conceptualization, supervision, validation, writing – review & editing; Christophe Lafosse: conceptualization, supervision, writing – review & editing; Céline Gillebert: conceptualization, funding acquisition, methodology, supervision, validation, writing – review & editing.

Abstract:

Patients with acquired brain injury frequently report experiencing sensory stimuli as abnormally under- (sensory hyposensitivity) or overwhelming (sensory hypersensitivity). Although, they can negatively impact daily functioning, these symptoms are poorly understood. To provide an overview of the current evidence on atypical sensory sensitivity after acquired brain injury, we conducted a systematic literature review. The primary aim of the review was to investigate the behavioural and neural mechanisms that are associated with self-reported sensory sensitivity. Studies were included when they studied sensory sensitivity in acquired brain injury populations and excluded when they were not written in English, consisted of non-empirical research, did not study human subjects, studied pain, related sensory sensitivity to peripheral injury, or studied patients with a neurodegenerative disorder, meningitis, encephalitis, or a brain tumour. The Web Of Science, PubMed, and Scopus databases were searched for appropriate studies. A qualitative synthesis of the results of the 81 studies that were included suggests that abnormal sensory thresholds and a reduced information processing speed are candidate behavioural mechanisms of atypical subjective sensory sensitivity after acquired brain injury. Furthermore, there was evidence for an association between subjective sensory sensitivity and structural grey or white matter abnormalities, and to functional abnormalities in sensory cortices. However, further research is needed to explore the causation of atypical sensory sensitivity. In addition, there is a need for the development of adequate diagnostic tools. This can significantly advance the quantity and quality of research on the prevalence, aetiology, prognosis, and treatment of these symptoms.

Keywords:

Traumatic brain injury, stroke, sensory hypersensitivity, sensory hyposensitivity, sensory overload, sensory processing

1. Introduction

Acquired brain injuries have become one of the world's leading cause of disability and reduced quality of life (Feigin et al., 2010; Greenwald et al., 2003). These injuries to the central nervous system are non-congenital, not neurodegenerative, nor induced by birth trauma (World Health Organization, 2006). Acquired brain injuries can be traumatic (i.e., traumatic brain injury (TBI)) or non-traumatic (i.e., stroke, anoxia, brain tumours), and can result in long-term impairments in mobility, speech, cognition, and socio-emotional functioning (Chiavaroli et al., 2016; Kohnen et al., 2019; Takizawa et al., 2016). Less well-known consequences of acquired brain injury are post-injury changes in sensory sensitivity causing patients to interpret non-nociceptive sensory stimuli (e.g., light, sound) as overwhelming (i.e., sensory hypersensitivity) or underwhelming (i.e., sensory hyposensitivity) (Alwawi et al., 2020; Chung & Song, 2016; Kumar et al., 2005). These symptoms are subjective by nature and can occur across different sensory modalities (i.e., visual, auditory, gustatory, olfactory, tactile, and vestibular sensitivity), have a significant impact on daily life, and are associated with poor functional recovery (Chorney et al., 2017; Landon et al., 2012; Shepherd et al., 2020).

Self-reported atypical sensory sensitivity is, however, not specific to patients with acquired brain injury. Sensory hypo- and hypersensitivity are also reported in the general population (Greven et al., 2019) and in other clinical populations such as autism spectrum disorder, attention deficit / hyperactivity disorder (ADHD), and schizophrenia (Bijlenga et al., 2017; Landon et al., 2016; Tavassoli et al., 2014). Previous research has identified possible behavioural and neural mechanisms associated with atypical sensory sensitivity in neurotypical adults as well as clinical groups (e.g., autism spectrum disorder, chronic pain patients). For instance, atypical sensory sensitivity has been related to abnormal sensory processing (i.e., atypical sensory thresholds or sensory acuity) (Ashwin et al., 2009; Brinkert & Remington, 2020; Brown & Dunn, 2002), attentional impairments (i.e., reduced selective attention, reduced information processing speed) (Liss et al., 2006; Marco et al., 2011; Panagiotidi et al., 2018, see also Thielen & Gillebert, 2019), and abnormal predictive processing (Ward, 2019). At the neural level, atypical sensory sensitivity has been related to functional abnormalities in the sensory cortices (Green et al., 2015; López-Solá et al., 2014), the insula (López-Solá et al., 2014), the thalamus (Acevedo et al., 2018), and limbic structures (Acevedo et al., 2018; Green et al., 2015). Furthermore, several authors (Green et al., 2016; Greven et al., 2019; Ward, 2019) proposed abnormalities within large-scale brain networks (specifically the salience network and the default mode network) as neural mechanisms of sensory sensitivity.

Similar behavioural (i.e., abnormal identification and discrimination of sensory stimuli, attentional impairments, abnormal prediction of subsequent sensory stimulation) and neural mechanisms (i.e., functional abnormalities in regions associated with sensory processing, atypical brain network

functioning) may relate to atypical sensory sensitivity after acquired brain injury. The primary aim (1) of this systematic review is to provide an overview of the current evidence for these mechanisms in patients with acquired brain injury. In addition, to get a broader view on potential protective or risk factors associated with atypical sensory sensitivity as well as on its prevalence and diagnosis, secondary aims of the systematic review were (2) to investigate the association between atypical sensory sensitivity after acquired brain injury and pre-injury demographic factors, injury characteristics, and comorbid symptomatology, (3) to assess the prevalence of sensory hypo- and hypersensitivity in different types of acquired brain injury as well as across different sensory modalities and (4) to determine the diagnostic tools that are used to assess sensory hypo- and hypersensitivity after acquired brain injury. Furthermore, to explore the evolution of and treatment possibilities for atypical sensory sensitivity we aimed to (5) summarize results concerning the evolution and (6) treatment of sensory hypo- and hypersensitivity after an acquired brain injury as well as (7) its relationship to injury outcomes.

2. Methods

2.1. Search strategy

We followed the recommendations from the Preferred Reporting for Systematic Reviews and Meta-Analyses (PRISMA) statement (Moher et al., 2009). The databases Web of Science, PubMed, and Scopus were searched using a search string that included different types of acquired brain injury as well as a variety of terms relating to sensory sensitivity or sensory intensity. The full search string consisted of the following terms: (("Brain injur*" OR "head injur*" OR stroke OR "subarachnoidal he$morrhage" OR "brain he$morrhage" OR "brain infarction" OR "cerebral infarction" OR "cerebral he$morrhage" OR "intracranial he$morrhage" OR "head trauma" OR "concussion" OR "craniocerebral trauma" OR "cerebrovascular trauma" OR "transient ischemic attack" OR "lacunar infarct" OR "vascular dementia" OR "brain anoxia" OR "brain hypoxia" OR "cerebral anoxia" OR "cerebral hypoxia" OR encephalop*) AND ("sensory *sens*" OR "sensory processing disorder" OR “sensory processing sensitivity” OR "sensory gating" OR "sensory overload" OR “sensory threshold” OR “sensory filtering” OR phonophobia OR photophobia OR osmophobia OR hyperacusis OR *sensitivit* NEAR/2 (light OR visual OR auditory OR sound OR noise OR touch OR tactile OR smell OR olfactory OR gustatory OR temperature OR taste OR vestibular) OR intensity NEAR/2 (light OR visual OR auditory OR sound OR noise OR touch OR tactile OR smell OR olfactory OR gustatory OR temperature OR taste OR vestibular))). The databases were last consulted in October 2021.

2.2. In- and exclusion criteria

Articles were excluded if they were not written in English, if they did not study human subjects (e.g., animal research), or if they did not study self-reported sensory sensitivity in acquired brain injury patients (e.g., research in participants with a neurodegenerative disorder). Articles on vascular dementia were not excluded since stroke can cause vascular dementia (Gorelick et al., 2011). We only included articles that discussed sensory sensitivity after cerebral damage and excluded articles that related atypical sensory sensitivity to peripheral injury (i.e., ocular damage), meningitis, encephalitis (due to the possibility of comorbid peripheral nervous system damage) (Bogovic, 2015), and brain tumours (since we could not specify whether changes in sensory sensitivity are result of the brain injury or of the cancer treatment) (Huang et al., 2019; Raffa et al., 2006). We also excluded articles on toxic encephalopathy due to long term solvent exposure since solvent exposure (in the absence of encephalopathy) can result in abnormal sensitivity to olfactory stimuli (Zibrowski & Robertson, 2006). Articles on pain were excluded when they described photo- or phonophobia solely during migraine episodes since photo- and phonophobia are known symptoms of migraine (Evans et al., 2008). Articles describing abnormal tactile sensitivity or temperature allodynia limited to a hemiplegic or painful body part were also excluded. Articles that studied military veterans were only included if it was explicitly stated that the veterans suffered from a traumatic brain injury (TBI) and not for example solely blast exposure. Only empirical studies were included, meaning that review articles or book chapters were excluded.

2.3. Eligibility assessment

Two reviewers (HT and NT or LW) independently reviewed the abstracts from the various databases for their relevance using the above described in- and exclusion criteria. A third reviewer (CRG) was consulted in case of disagreement (this was the case for four articles, of which three were excluded and one was included (Wehling et al. (2015)).

2.4. Data extraction

From the included articles, we extracted the characteristics of the article (title, authors, year of publication) as well as demographic characteristics of the studied acquired brain injury population (sample size, age, sex, type of acquired brain injury, time since injury) and, if available, the characteristics of the studied control group (sample size, age, and sex). Based on their mean age we classified the studied samples as adult (mean age ≥ 18 years) or non-adult (mean age < 18 years). Articles on TBI based were categorized into two groups based on injury severity: mild traumatic brain injury (mTBI) (including concussions) (Mayer et al., 2017) and moderate to severe TBI. Depending on the mean number of months between brain injury onset and sensory sensitivity assessment we

identified time since injury as (sub)acute (less than six months after injury) or chronic (six months or longer after injury) (based on Bernhardt et al. (2017), Bond (1979), Licastro et al. (2016)). Studies that included both acute and chronic patients were classified as 'acute to chronic'. Data extraction also included the sensory modalities that were studied (i.e., auditory, visual, olfactory, gustatory, tactile, or vestibular sensitivity as well as a sensitivity to light[1]), study design aspects (i.e., what diagnostical tools were used to assess sensory sensitivity), whether the study assessed hypo- and/or hypersensitivity, and a summary of the results.

2.5. Quality assessment

The methodological quality of the included articles was assessed by two independent reviewers (HT and NT) using the Mixed Methods Appraisal Tool (Hong et al., 2018).

2.6.Data analysis

We used qualitative synthesis to summarize results on sensory hypo- and hypersensitivity after an acquired brain injury. In alignment with our research aims, we focused on (1) behavioural and neural mechanisms of atypical sensory sensitivity, (2) demographic factors, injury mechanisms, and comorbid symptomatology associated with hypo- or hypersensitivity, (3) the prevalence of self-reported sensory hypo- and hypersensitivity across different modalities, (4) the diagnostic tools used to assess sensory sensitivity, (5) the evolution and (6) treatment of atypical sensory sensitivity after an acquired brain injury, and (7) injury outcomes associated with atypical sensory sensitivity. Conducting a meta-analysis was considered not feasible due to high heterogeneity in the assessment of sensory sensitivity, the study design, and the sample characteristics of the clinical populations in the included studies. Figures were created using Microsoft Excel (2019) and Adobe Illustrator (2020). Details of the included studies (including demographic characteristics of the studied sample, study design aspects) can be found in the supplementary tables as well as in the article extraction file which is available via https://doi.org/10.6084/m9.figshare.14785293.

3. Results

3.1. Search strategy

Figure 1 displays the study flow diagram based on the PRISMA statement (Moher et al., 2009). We identified 998 records through database screening and one additional record through other sources (i.e., library collection). 267 duplicates were removed, leaving 732 articles. Based on the exclusion criteria, we excluded 610 articles. After consulting the full text, an additional 29 articles were excluded

[1] Since a high number of studies focused on light sensitivity specifically, we differentiated between articles on light sensitivity and visual sensitivity (not limited to light sensitivity).

(see Figure 1). For 12 articles the full text was not available, leaving 81 studies to be included in the analysis.

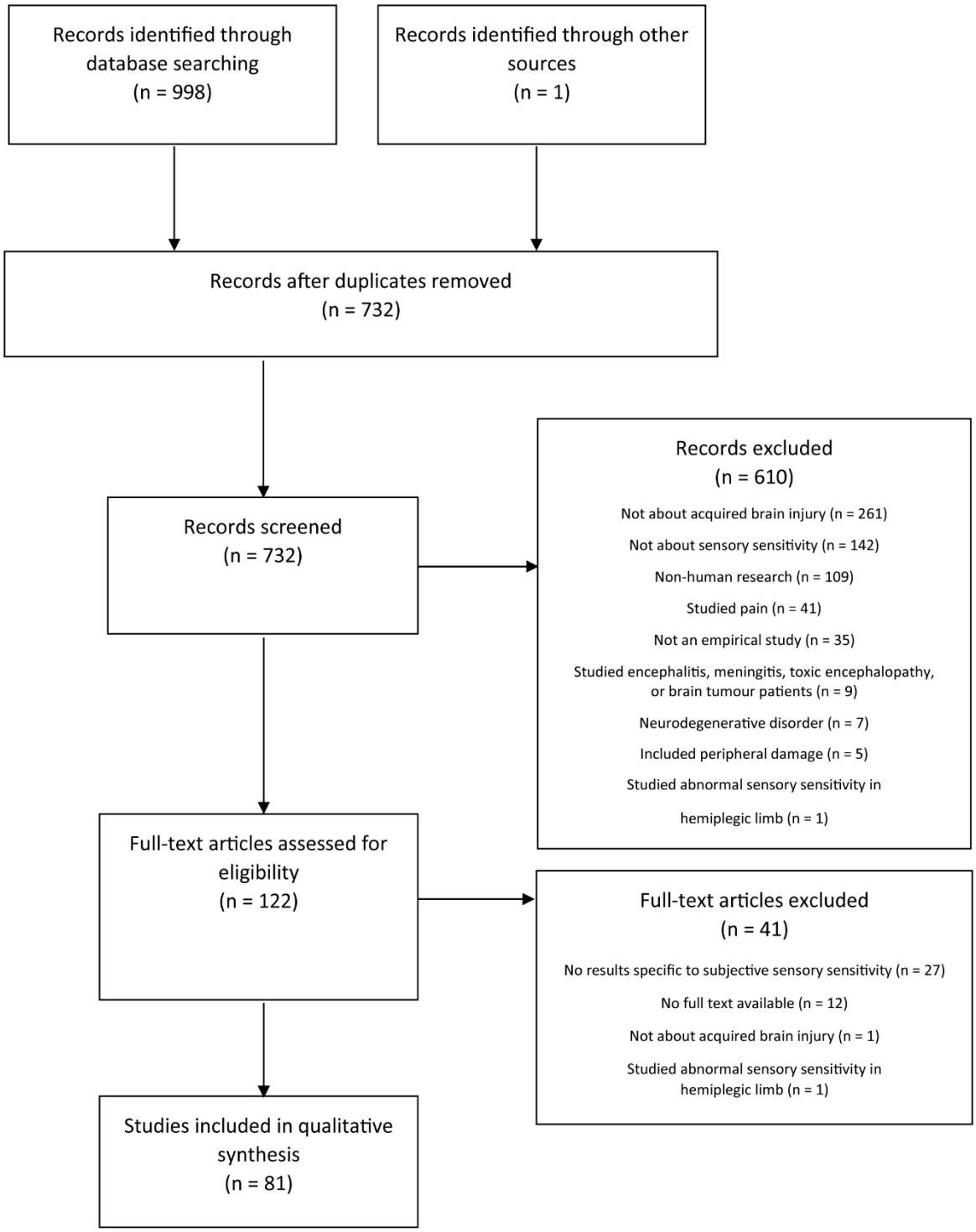

Fig. 1 PRISMA flow diagram for the systematic literature review

3.2. Study characteristics

The majority (74%) of the included studies investigated sensory sensitivity in mild TBI (mTBI) patients. One study studied moderate to severe TBI (Colantonio et al., 2010). Other studies about mild to severe TBI did not clearly describe the severity of TBI (n = 6) or included participants across all TBI severities (n = 6). 95% of the included studies assessed hypersensitivity (see Figures 2 and 3). When considering the different sensory modalities, light sensitivity (73%) and auditory sensitivity (69%) were studied most frequently (see Figure 3). Lastly, more than half of the studies (58%) investigated sensory sensitivity in more than one sensory modality.

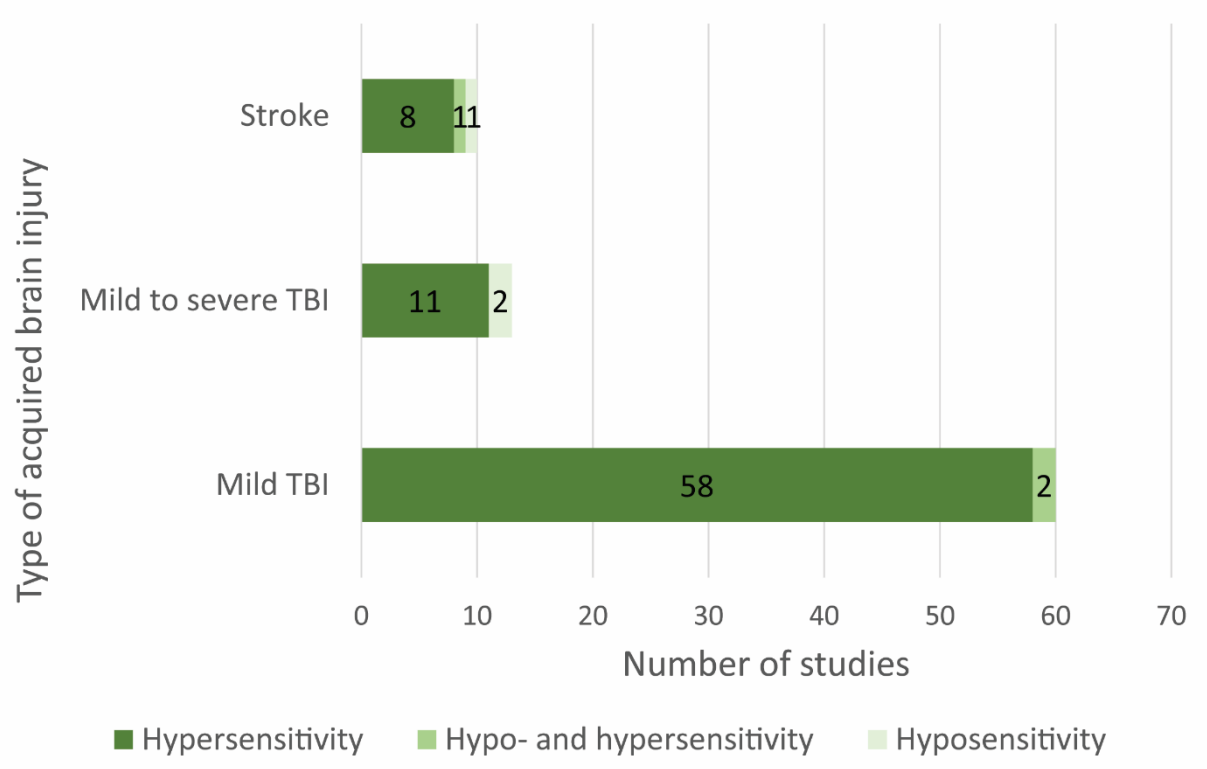

Fig. 2 The number of studies that investigated hypo- and/or hypersensitivity across the different types of acquired brain injury. Note: two studies that studied both TBI and stroke were classified twice.

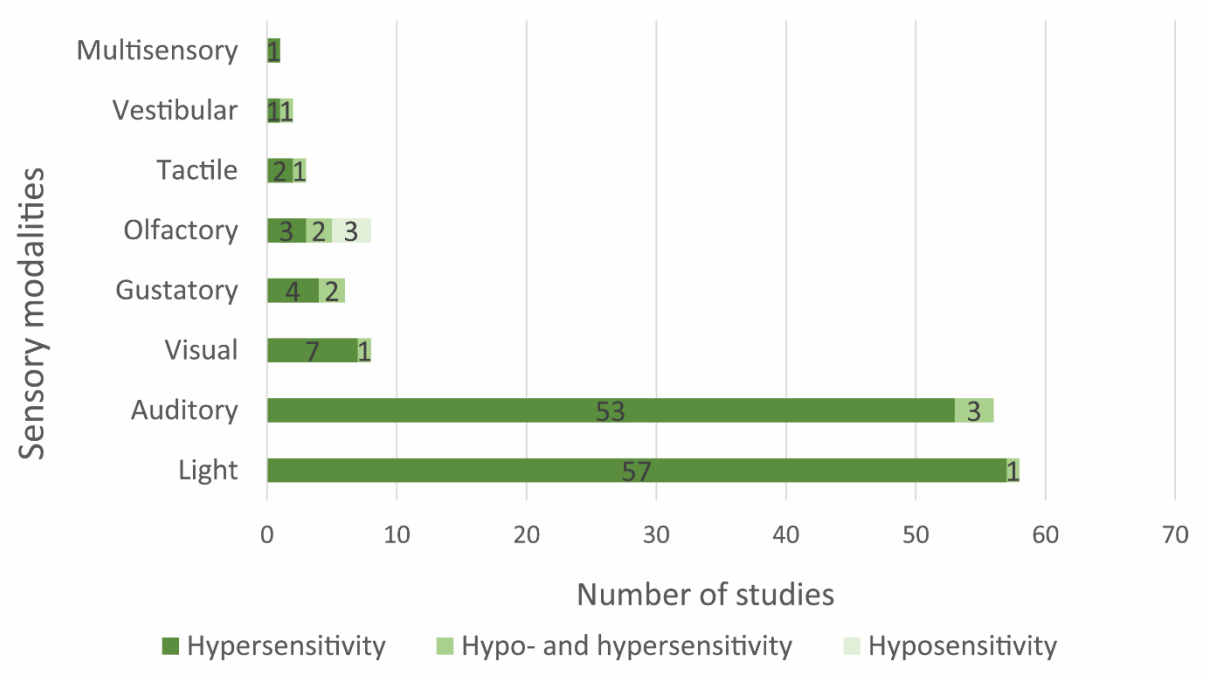

Fig. 3 The number of studies that investigated sensory hypo- and/or hypersensitivity across different sensory modalities. More than half of the studies (58%) investigated sensitivity to multiple sensory modalities and were classified multiple times. Multisensory sensitivity refers to a sensitivity to multiple sensory stimuli that are present simultaneously and belong to different sensory modalities (e.g., experiencing an atypical sensitivity to the combination of visual and auditory stimulation).

3.3. Methodological quality of the included studies

The quality of the included studies is presented in Figure 4 (see also Supplementary Table 1). From the 72 studies that were classified as quantitative descriptive research (see Hong et al., 2018), one fulfilled all quality criteria. Importantly, only half of the studies (50%) assessed sensory hypo- and hypersensitivity using an appropriate method and less than a quarter of the studies (13%) clearly discussed response rate and reasons for non-response (which is needed to assess selection bias). Since there is ongoing debate about the necessity of a correction for multiple comparisons (see for example Frane, 2019), the studies that did not correct for multiple comparisons were marked as 'unclear' regarding the criterium 'appropriate statistical analysis' (if there was no other reason to mark these studies as using an inappropriate statistical analysis).

From the nine studies that were classified as qualitative research, seven fulfilled all quality criteria. Two studies (22%) did not fulfil the quality criteria because the interpretation of the results were not sufficiently supported by the data.

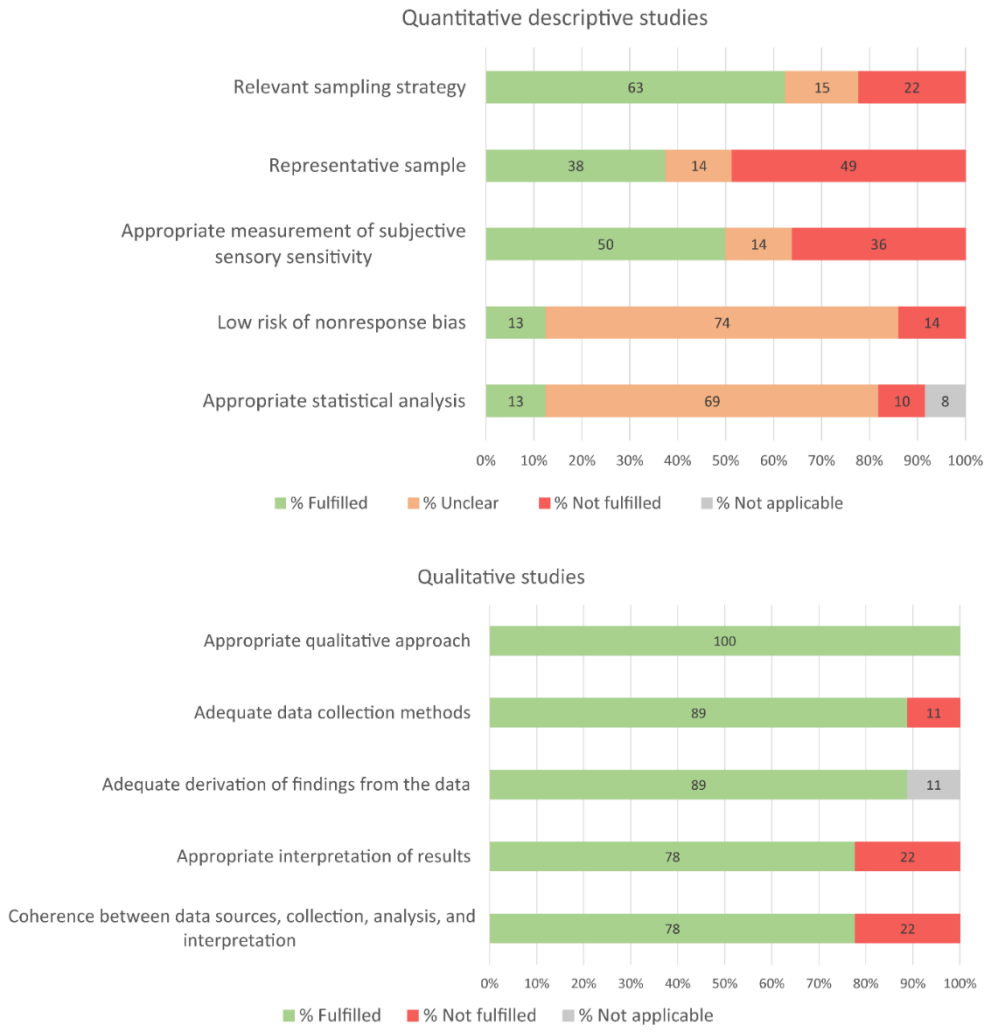

Fig. 4 The % of included quantitative descriptive or qualitative studies for which the methodological criteria of the Mixed Methods Appraisal Tool (Hong et al., 2018) are fulfilled, not fulfilled, unclear, or not applicable.

3.4. The behavioural and neural mechanisms of sensory sensitivity

Table 1 summarizes the results of the studies (n = 18) that investigated behavioural (n = 7) and/or neural mechanisms (n = 10) of sensory sensitivity after acquired brain injury. One study (Pritchard et al., 1999) studied both the behavioural *and* neural mechanisms of atypical sensory sensitivity.

[Insert Table 1]

3.4.1. Behavioural mechanisms of sensory sensitivity

There was no evidence that sensory sensitivity across different sensory modalities (visual, auditory, tactile, gustatory, and olfactory sensitivity) was related to selective or sustained attention performance (Kumar et al., 2005; Shepherd et al., 2019). However, sensory sensitivity did correlate with time taken on neuropsychological assessments of attention and cognitive flexibility (Kumar et al., 2005; Shepherd et al., 2019). Noteworthy, in Shepherd et al. (2019), these correlations only reached significance in

female participants. No evidence was found for a relationship between sensory sensitivity and other measures of psychomotor speed, memory, and executive functioning (Kumar et al., 2005; Nelson et al., 2018; Shepherd et al., 2019).

Chang et al. (2007) and Schrupp et al. (2009) studied the relationship between light and visual motion sensitivity and the critical flicker fusion frequency (i.e., the frequency at which a physically flickering light is no longer perceived to be flickering). Chang et al. (2007) found that the mean critical flicker fusion frequency at the fovea increased according to the severity of light sensitivity in mTBI patients. However, Schrupp et al. (2009) did not find evidence for such a relationship in a similar sample.

Multiple studies reported that patients with olfactory and gustatory hyposensitivity also displayed reduced behavioural sensory awareness (i.e., reduced identification of sensory stimuli or discrimination between stimuli, a higher sensory threshold) (Gudziol et al., 2014; Pritchard et al., 1999). In contrast, Wehling et al. (2015) observed a correspondence between behavioural olfactory hyposensitivity and reduced odour pleasantness, but no relationship with a reduced sense of smell.

3.4.2. Neural mechanisms of sensory sensitivity

Seven studies related atypical sensory sensitivity to structural brain abnormalities. Likova and Tyler (2018) reported pontine degeneration in mTBI patients who expressed being hypersensitive to light and Lewis et al. (2020) concluded that biomarkers indicative of cellular and axonal damage (i.e., blood plasma level of ubiquitin C-terminal hydrolase L1 and glial fibrillary acidic protein) correlated with both light and noise sensitivity. Using diffusion tensor imaging, Astafiev et al. (2016) observed higher fractional anisotropy values near the left optic radiation in mTBI patients with versus without light hypersensitivity. Four case studies (Boucher et al., 2015; Cantone et al., 2019; Mak et al., 2005; Pritchard et al., 1999) related atypical post-stroke sensory sensitivity in different modalities (gustatory and olfactory for Mak et al. (2005), auditory for Boucher et al. (2015), visual for Cantone et al. (2019), and gustatory for Pritchard et al. (1999)) to insular lesions. Even though Boucher et al. (2015) focused on post-stroke hyperacusis, their two cases also reported being hypersensitive to other sensory modalities (i.e., comorbid tactile and olfactory hypersensitivity). The case discussed by Mak et al. (2005) reported a post-stroke change in his sensitivity to temperature in addition to gustatory and olfactory hypersensitivity.

Four studies related atypical sensory sensitivity to functional changes in brain activity. In the study by Astafiev et al. (2016) mTBI patients with light hypersensitivity displayed higher blood-oxygen-level-dependent (BOLD) responses in visual areas. The two stroke cases with auditory hypersensitivity discussed by Boucher et al. (2015) also displayed abnormal auditory event related potentials (i.e., larger P3b amplitude and reduced N1 amplitudes). Furthermore, Yadav and Ciuffreda (2014) and

Ciuffreda et al. (2013) reported that wearing binasal occluders (with or without base-in prisms) had a different effect on the P100 amplitude in chronic mTBI patients who were hypersensitive to visual motion as compared to neurotypical adults.

Lastly, two studies related visual and auditory reflexes to sensory sensitivity. Troung and Ciuffreda (2016) found that mTBI patients who were hypersensitive to light had abnormal pupillary light reflexes which has been linked to autonomic nervous system dysfunction (Wang et al., 2016). Nölle et al. (2004) found that abnormal performance on central auditory pathway testing in mTBI patients was related to atypical auditory sensitivity.

3.5.Pre-injury factors, injury mechanisms and comorbid symptomatology associated with atypical sensory sensitivity

Details of the studies (n = 28) discussed below can be found in Supplementary Table 2.

3.5.1. Demographic factors

Results on the relationship between sex and sensory sensitivity were inconsistent. Some studies found that females with a mTBI reported light or auditory hypersensitivity more frequently or with a higher severity as compared to males with a mTBI (Brickell et al., 2017; Bunt et al., 2020, 2021; Frommer et al., 2011; Shepherd et al., 2019) However, no evidence for this sex difference was found by other studies (Elliott et al., 2018; Knoll et al., 2020a; Lumba-Brown et al., 2020).

Some studies reported that the prevalence of light hypersensitivity decreased with increasing age (Helmich et al., 2019; Hu et al., 2017; Karr et al., 2020). In contrast, Shepherd et al. (2019, 2021) did not find evidence for a relationship between age and auditory hypersensitivity.

Shepherd et al. (2019) observed an association between sensory sensitivity and place of living with patients from rural areas reporting higher auditory sensitivity after their mTBI than participants from urban areas. However, a more recent study by Shepherd et al. (2021) found no evidence for an association between place of living and auditory sensitivity. No study found a statistically significant association between education level and sensory sensitivity to light or noise (e.g., Elliott et al., 2018; Shepherd et al., 2019).

The severity of light and auditory hypersensitivity was higher in patients with multiple mTBIs as compared to patients with a single mTBI (Chen et al., 2019; Elliott et al., 2018; Shepherd et al., 2019). Elliott et al. (2018) did not find evidence for an association between medical comorbidities (such as diabetes, hypertension, heart, or lung disease) and sensory hypersensitivity. Lastly, Han et al. (2008) found that light hypersensitivity was reported more frequently by TBI patients who took medication (such as antidepressants, antihypertensives, analgesics) than those who did not take medication.

3.5.2. Mechanisms of the brain injury

There was no evidence for a different prevalence or a different severity of light or auditory hypersensitivity according to the cause of a mTBI (i.e., fall, car accident, assault, sport-related mTBI) (Knoll et al., 2020a; Lumba-Brown et al., 2020; Shepherd et al., 2019). However, Goodrich et al. (2013) found that light hypersensitivity was reported more frequently by blast exposed TBI patients as compared to non-blast exposed TBI patients, but this difference was no longer significant when mTBI patients were removed from the analyses. Auditory hypersensitivity displayed a weak negative association with injury severity (Shepherd et al., 2019).

3.5.3.Comorbid symptomatology

Multiple studies reported that the presence of self-reported sensory hypersensitivity was associated with an increase in the severity of other post-concussion symptoms, such as difficulties concentrating, dizziness, irritability, and tinnitus (Astafiev et al., 2016; Chandran et al., 2020; Chorney et al., 2017; Elliott et al., 2018; Forrest et al., 2018; Kumar et al., 2005; Shepherd et al., 2019, 2021). However, a reverse relationship (i.e., auditory sensitivity had a negative association with the presence of comorbid headaches) was reported by Forrest et al. (2018). Furthermore, there is evidence for an association between light and auditory hypersensitivity (i.e., Chandran et al., 2020; Shepherd et al., 2020).

Evidence for a positive relationship between abnormal auditory and light sensitivity and symptoms of depression, anxiety, or post-traumatic stress disorder (PTSD) was found by multiple studies (Al-Ozairi et al., 2015; Assi et al., 2018; Callahan et al., 2018; Callahan & Storzbach, 2019; Elliott et al., 2018; Goodrich et al., 2014; Shepherd et al., 2019, 2021). One study by Nelson et al. (2018) found no evidence for such a relationship.

Furthermore, sensory hypersensitivity was associated with other psychological symptoms such as somatization (positive association) (Callahan et al., 2018; Nelson et al., 2018) and perception of recovery (negative association with auditory hypersensitivity, which was stronger for male participants as compared to female participants) (Shepherd et al., 2019). To date, there is no evidence for a relationship between sensory hypersensitivity and personality traits (e.g., Nelson et al., 2018).

Sensory hypersensitivity was related to reduced subjective sleep quality (Elliott et al., 2018; Howell et al., 2019) but not to abnormal polysomnographic metrics (Elliott et al., 2018).

3.6. The prevalence of atypical sensory sensitivity

Figure 5 displays the prevalence of hypo- and hypersensitivity categorized according to the type of acquired brain injury and sensory modality (based on n = 32 studies, for details see Supplementary Table 3). Most of the studies (91%) investigated the prevalence of light or auditory hypersensitivity after mTBI. Two studies reported prevalences that were specific to moderate to severe TBI patients

(see Figure 5, panel b) and one study considered both mTBI and moderate to severe TBI but did not report prevalences specific to TBI severity (see Figure 5, panel c). No studies mentioned a modality-specific prevalence for atypical sensory sensitivity after non-traumatic acquired brain injury. However, Chung and Song (2016) observed a prevalence of hypo- and hypersensitivity (not specific to a certain sensory modality) in respectively 16% and in 18% of stroke patients. Additionally, during semi-structured interviews stroke patients reported being hypersensitivity to light, noise, textures, and environmental temperatures (Alwawi et al., 2020; Carlsson et al., 2004, 2009).

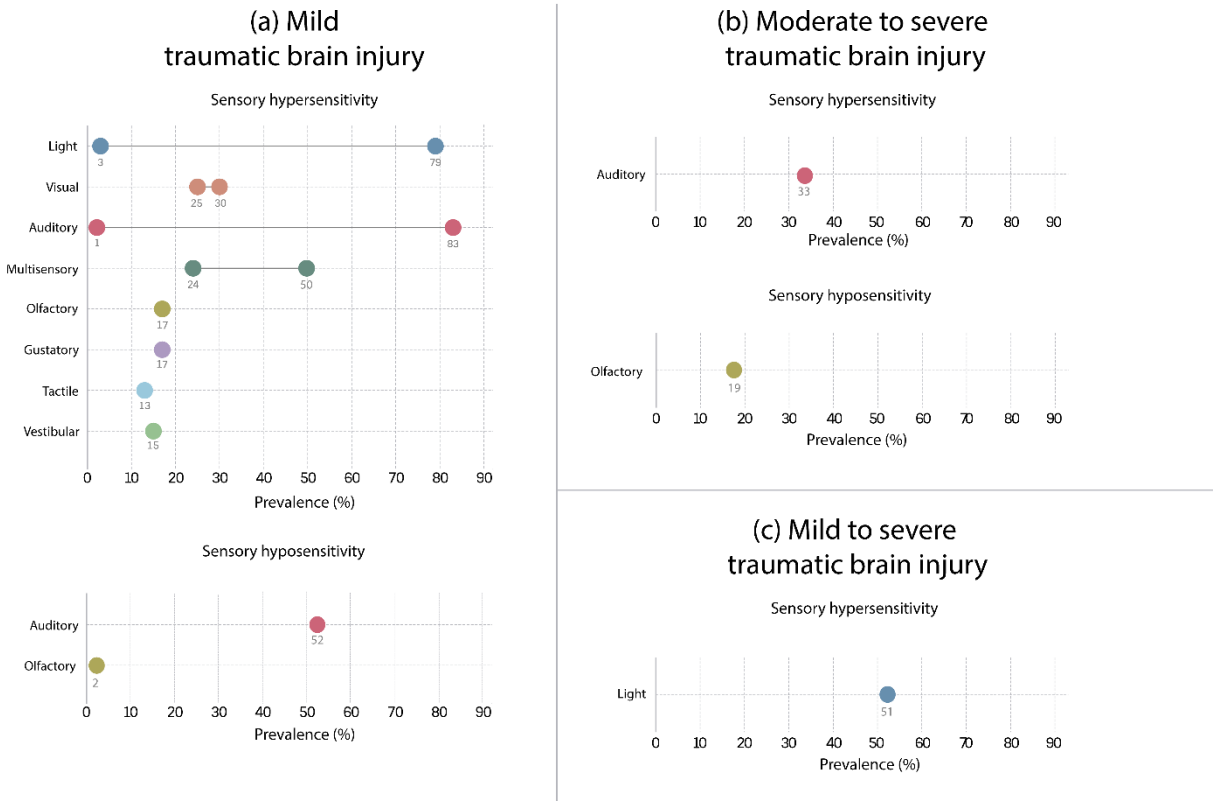

Fig. 5 The prevalence of sensory hyper- or hyposensitivity after a mTBI (panel a) or after moderate to severe TBI (panel b) and mild to severe TBI (panel c) (for details of the studies see Supplementary Table 3). A single dot represents a prevalence estimate from a single study. Two dots connected by a line represent the range of estimated prevalences found in different studies with the dots representing the lowest and highest estimates.

3.7. The diagnostic tools used to assess sensory sensitivity

Table 2 outlines the different diagnostic tools that were used to assess sensory sensitivity. 22% of the included studies did not disclose how they measured sensory sensitivity (e.g., Chandran et al., 2020; Nölle et al., 2004; Truong & Ciuffreda, 2016) and 15% of the studies used a self-developed questionnaire (e.g., Gudziol et al., 2014; Kerr et al., 2018; Zuckerman et al., 2016). Less than half of the studies (36%) used a validated questionnaire such as the Post-concussion Symptom Scale of the Sport Concussion Assessment Tool (e.g., Bunt et al., 2020; Lumba-Brown et al., 2020), the Rivermead Post-concussion Symptom Questionnaire (e.g., King & Kirwilliam, 2013; Lewis et al., 2020), and the

Neurobehavioural Symptom Inventory (Brickell et al., 2017; Callahan & Storzbach, 2019). Most of the used questionnaires (85%) assessed sensory sensitivity using a single item for each modality. Additionally, assessment of sensory sensitivity mainly (in 79% of the studies) focused on light and/or noise sensitivity.

Table 2. The diagnostic tools used to assess sensory sensitivity after an acquired brain injury.

Tool used to assess sensory sensitivity	% of studies (n = 81)
Unclear	22%
Self-developed	15%
Rivermead Post-concussion Symptom Questionnaire	15%
Post-concussion Symptom Scale (from the Sport Concussion Assessment Tool)	10%
Medical file record	9%
Neurobehavioural Symptom Inventory	5%
Post-concussion Symptom Scale (from the Immediate Post-Concussion Assessment & Cognitive Testing)	5%
Self-reported discomfort	5%
Subjective description (Case)	4%
Self-reported intensity	3%
Concussion Symptom Checklist	1%
Head Injury Symptom Checklist	1%
Structured Interview for Assessing Perceptual Anomalies	1%
Postconcussion Symptom Inventory	1%
Interview	1%
Problem Checklist from the Head Injury Family Interview	1%
Adult/Adolescent Sensory Profile	1%

3.8. Evolution of sensory hypo- or hypersensitivity after an acquired brain injury

Research on the evolution of sensory sensitivity focused solely on hypersensitivity and was limited to six studies in mTBI patients and one study in stroke patients (see Supplementary Table 4). There is, to date, no research on the evolution of sensory hyposensitivity.

Barker-Collo et al. (2018) and Shepherd et al. (2021) provided longitudinal measures of sensory hypersensitivity at baseline, 1-, 6-, and 12-months post-injury in mTBI patients (aged 16 years or older). Barker-Collo et al. (2018) found a decreasing trend of the prevalence of light and auditory hypersensitivity from baseline to 12-months post injury (see Figure 6, panel a). Similarly, Shepherd et

al. (2021) reported that the prevalence of auditory hypersensitivity at baseline (44%) was higher than at 12-months post-injury (27%). Additionally, Shepherd et al. (2021) implied that the severity of auditory sensitivity decreased after baseline (see Figure 6, panel b). However, it must be noted that it is unclear if these reductions in mean auditory sensitivity severity remained significant after correction for multiple comparisons.

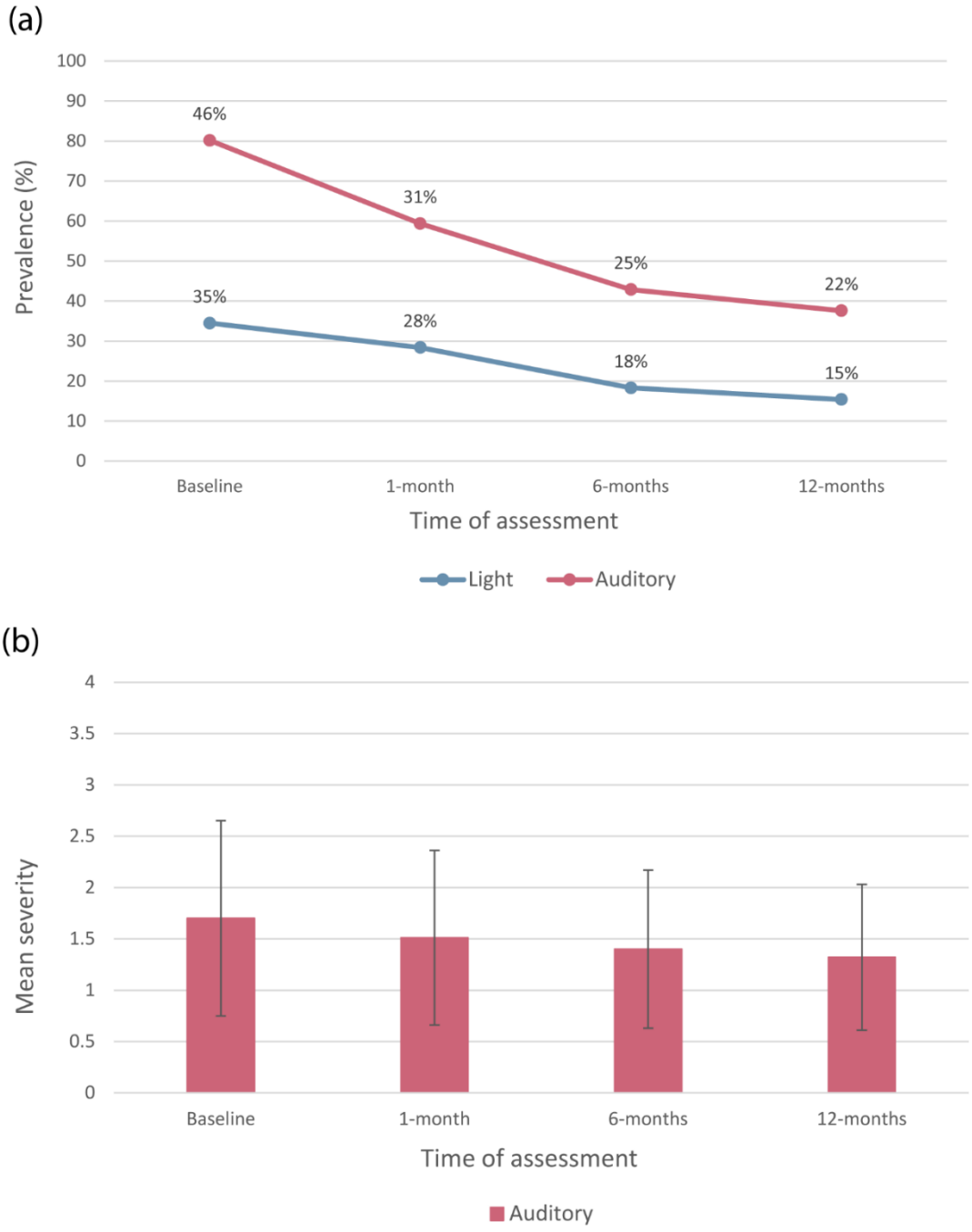

Fig. 6 Panel a: The prevalence of light or auditory hypersensitivity after mTBI as reported by Barker-Collo et al. (2018). **Panel b**: The severity of auditory hypersensitivity after mTBI as reported by Shepherd et al. (2021). The severity scale ranged from 0 indicating no hypersensitivity to 4 indicating severe hypersensitivity. Baseline = maximally 2 weeks post-injury.

Even though the prevalence and severity of sensory hypersensitivity seem to decrease at a group level (Barker-Collo et al., 2018; Shepherd et al., 2021), the evolution of sensory hypersensitivity also varies inter-individually with some patients reporting earlier or greater alleviations of symptoms as compared to others (Alwawi et al., 2020; Truong et al., 2014). Truong et al. (2014), for instance, reported that a reduction of light hypersensitivity was only present in 50% of their sample of 62 mTBI patients and that alleviation of light hypersensitivity was lower in patients who reported other comorbid post-concussion symptoms (such as auditory hypersensitivity). Furthermore, other studies highlight that the severity of the sensory sensitivity symptoms can wax and wane intra-individually (for instance, the severity can vary according to circadian patterns) (Rabinowitz & Fisher, 2020; Truong et al., 2014).

3.9. Treatment of sensory hypo- or hyper sensitivity after acquired brain injury

Eight studies investigated a possible treatment for hypersensitivity after a TBI (see Supplementary Table 5). Reductions in visual hypersensitivity were reported when wearing binasal occluders (Ciuffreda et al., 2013; Yadav & Ciuffreda, 2014), coloured glasses (Clark et al., 2017), or contact lenses (Truong et al., 2014). Similarly, self-reported discomfort when exposed to a computer screen decreased when using a non-liquid crystal display (non-LCD) screen (Mansur et al., 2018) that refreshed at a lower rate than a standard LCD screen. Lastly, Gunter et al. (2018) and Teare-Ketter et al. (2021) described cases with light hypersensitivity after a mTBI. The cases were both symptom free after several weeks of physical therapy (no specific treatment for the hypersensitivity symptoms was mentioned).

Considering auditory hypersensitivity, Hallberg et al. (2005) described a treatment program in which chronic TBI patients with auditory hypersensitivity gradually exposed themselves to an increasing intensity of environmental sounds while participating in daily life. To control this gradual exposure, patients wore individually designed attenuators which were inserted in the external auditory canal to exclude environmental sounds. Throughout the treatment, holes with an increasing diameter (1 mm to 3 mm) were drilled in the attenuators to increasingly expose participants to more external sounds. In addition, the treatment consisted of assisting patients in identifying and challenging maladaptive coping styles (i.e., inflexible avoidance) related to their sensory hypersensitivity. By means of semi-structured interviews participants evaluated the treatment program as positive: patients reported participating in a higher number of social situations as compared to before their treatment as well as being less distracted by environmental sounds.

3.10. Injury outcomes related to atypical sensory sensitivity

Fifteen studies examined the association between functional recovery and sensory sensitivity (see Supplementary Table 6). Sensory hypersensitivity was associated with an increased recovery time (Falk

et al., 2021; Forrest et al., 2018; O'Kane et al., 2014), increased persistence of other post-concussion symptoms (e.g., Kerr et al., 2018; Zemek et al., 2016; Zuckerman et al., 2016), hospital reattendance (Mistry & Rainer, 2018), and decreased chances of gaining clearance to resume driving (MacDonald et al., 2018). In contrast, Mortera et al. (2018) reported that veterans with a mTBI who returned to productivity were twice as likely to report light hypersensitivity as compared to veterans with a mTBI who did not return to productivity. Lau et al. (2011) did not find evidence for a statistically significant association between light or auditory hypersensitivity and length of recovery.

Nine studies (see Supplementary Table 6) investigated the relationship between quality of life and hypersensitivity. Multiple studies reported that sensory hypersensitivity was associated with a self-reported reduction in quality of life in adult samples (e.g., reduced participation in social activities or economic difficulties) (Alwawi et al., 2020; Carlsson et al., 2004, 2009; Shepherd et al., 2020; Trulsson et al., 2003). However, Vassilyadi et al. (2014) found no evidence for a relationship between hypersensitivity to light or noise and quality of life in a non-adult sample.

Shepherd et al. (2020) found that the association between hypersensitivity and quality of life remained significant even after controlling for sex, age at injury, education level, and injury severity. Furthermore, this association differed according to sensory modality: light hypersensitivity was strongly associated with experiencing bodily pain while noise hypersensitivity was strongly associated with limitations related to emotional problems.

Colantonio et al. (2010) found an effect of sex on the relationship between auditory hypersensitivity and quality of life: men with a TBI reported a greater reduction in their quality of life due to their hypersensitivity than women with a TBI. There was no evidence for significant sex difference with regard to the reported impact of light hypersensitivity on quality of life.

Table 1. An overview of studies (n = 18) discussing the behavioural and neural mechanisms of sensory sensitivity after an acquired brain injury.

Study	Type of Acquired brain injury Sample size Age of sample [Age range]	Time since injury	Sensory modality	Hypo- or hypersensitivity	Measurement of subjective sensory sensitivity	Behavioural mechanisms	Neural mechanisms	Summary of results
Behavioural mechanisms of sensory sensitivity								
Shepherd et al. (2019)	mTBI N = 151 Adult	Acute	Auditory	Hyper	Rivermead Post-concussion Symptoms Questionnaire	Complex attention (via a composite score that was based on the performance on the Continuous Performance Test, the Stroop Test, and the Shifting Attention Test) Cognitive flexibility (via a composite score that was based on the performance on the Stroop Test and the Shifting Attention Test) Information processing speed (via the reaction time on the Stroop Test) Psychomotor speed (via a composite score that was based on the performance on the		There was no evidence for a correlation between auditory sensitivity and the composite scores measuring complex attention, psychomotor speed, visual, or verbal memory. Auditory sensitivity did correlate with reaction time on the Stroop test which is thought to measure information processing speed and the composite score of cognitive flexibility (which was based on similar tests as the complex attention score). These correlations were only significant in female participants.

						Finger Tapping Task and the Symbol Digit Coding Test) Visual and verbal memory (via adaptations of the Rey Auditory Verbal Learning Test and the Rey Visual Design Learning Test)	
Nelson et al. (2018)	mTBI N = 219 Adult	Acute	Light Auditory	Hyper	Post-concussion Symptom Scale of the Sport Concussion Assessment Tool 3	Immediate Postconcussion and Cognitive Testing	There was no evidence that factor scores containing both light and auditory sensitivity correlated with cognitive performance on the Immediate Postconcussion and Cognitive Testing.
Kumar et al. (2005)	mTBI N = 30 Adult [16-52]	Acute	Visual Auditory Tactile Gustatory Olfactory	Hyper	Structured Interview for Assessing Perceptual Anomalies	Selective and Sustained attention (via the Stroop test and the Digit Vigilance Test) Psychomotor speed (via the Digit Symbol Substitution Test) Executive functioning (via Animal fluency test, Wisconsin Card Sorting Test, Stroop test, and the Tower of London) Visual and verbal memory (via the Auditory Verbal	Sensory sensitivity across several sensory modalities correlated with time taken on a Digit Vigilance Test (test for selective and sustained attention) but there was no evidence for a correlation with performance on the Stroop test (test for selective attention and inhibition) or with performance on other tests measuring psychomotor speed, executive functioning, or visual and verbal memory.

						Learning Test, Complex Figure Test)	
Gudziol et al. (2014)	TBI (both mild, moderate, and severe) N = 110 Adult [18-69]	Acute to chronic	Olfactory	Hypo	Subjective evaluation of sense of smell (1 item)	The 'Sniffin' Sticks' test for odour threshold, discrimination, and identification Odour threshold, discrimination, and identification performance were combined in a composite score	Five patients (one with mTBI and four with moderate to severe TBI) who reported a reduced olfactory functioning due to their TBI completed the 'Sniffin' Sticks' test. They all displayed deficient performance on the olfactory testing (hyposmic: n = 3, anosmic: n = 2).
Wehling et al. (2015)	Stroke N = 78 Adult	Chronic	Olfactory	Hypo	Self-developed questionnaire (1 item)	Olfactory identification via the Scandinavian Odour Identification Test	Self-reported sense of smell (judged as quite poor to excellent) did not differ significantly between normosmic (n = 44) and hypo- / anosmic stroke patients (n = 30) (as identified using the Scandinavian Odour Identification Test). However, the hypo- /anosmic stroke patients reported a significantly lower odour pleasantness than normosmic stroke patients.
Schrupp et al. (2009)	mTBI N = 14 Adult [18-59]	Chronic	Light Visual motion sensitivity	Hyper	Self-developed questionnaire (> 1 item per sensory modality)	Critical flicker fusion frequency at the fovea as well as in the right and left hemifield (10° horizontal retinal eccentricity)	There was no evidence for a correlation between light or visual motion sensitivity and the mean critical flicker fusion frequency at the fovea, in the right, or in the left hemifield.

Chang et al. (2007)	mTBI N = 18 Adult [19-72]	Chronic	Light Visual motion sensitivity	Hyper	Self-developed questionnaire (> 1 item per sensory modality)	Critical flicker fusion frequency at the fovea		The mean critical flicker fusion frequency threshold at the fovea was significantly higher in mTBI patients who were hypersensitive to light or visual motion (n is not mentioned) as compared to mTBI patients with no light or visual motion hypersensitivity. The mean critical flicker frequency threshold increased according to light sensitivity severity.
Neural mechanisms of sensory sensitivity								
Truong and Ciuffreda (2016)	mTBI N = 32 Adult [21-60]	Not reported	Light	Hyper	Not reported		Pupillary light reflexes	mTBI patients who were photosensitive (n = 21) had a larger baseline pupil diameter, a larger minimum pupil diameter, faster peak dilation velocity (i.e., time between stimulus onset and peak dilation), faster redilation recovery, and larger pupil diameter (6 seconds after stimulus onset) as compared to mTBI patients who were not photosensitive (n =11).

Lewis et al. (2020)	mTBI N = 27 Adult	Acute	Light Auditory	Hyper	Rivermead Post-concussion Symptoms Questionnaire	Levels of glial fibrillary acidic protein (GFAP), Tau, ubiquitin C-terminal hydrolase L1 (UCH-L1), and cell-free DNA (cfDNA) in blood plasma	Plasma levels of UCL-L1 and GFAP were highly correlated (r > .9) with sensitivity for light and noise. There was no evidence for a correlation between Tau and cfDNA with sensitivity for light and noise.
Cantone et al. (2019)	Stroke N = 1 Adult [62]	Acute	Visual	Hyper	Subjective description	Lesion location	Subjective description of hypersensitivity to light (facial expression of fear and disgust with a neurovegetative reaction and horripilation in response to visual stimuli) after right temporal-insular lesion.
Likova and Tyler (2018)	mTBI N = 16 Adult [42-81]	Acute to chronic	Light	Hyper	Self-reported light induced discomfort when exposed to a white field stimulus flickering	Tensor-Based Morphometry	mTBI patients with light hypersensitivity (n = 11) showed mid-pontine shrinkage, consistent with degeneration of nuclei of the trigeminal complex. mTBI patients without light hypersensitivity (n = 5) showed bilateral expansion at the pontine / medulla junction.

Astafiev et al. (2016)	mTBI N = 20 Adult [20-57]	Chronic	Light	Hyper	Head Injury Symptom Checklist	Task-related and resting-state functional MRI Diffusion Tensor Imaging	mTBI patients with light hypersensitivity (n = 6) had higher BOLD magnitudes in the middle temporal and lateral occipital visual areas during a visual tracking task than mTBI patients without light sensitivity (n = 11). Similarly, task-evoked BOLD activity in the middle temporal and lateral occipital visual areas correlated with light sensitivity. mTBI patients with light hypersensitivity also had higher fractional anisotropy values near the left optic radiation.
Boucher et al. (2015)	Stroke N = 2 Adult [29-40]	Chronic	Auditory (Tactile) (Olfactory)	Hyper	Hearing Sensitivity Questionnaire, loudness discomfort task	Auditory event related potential (ERP) paradigms (mismatch negativity and auditory oddball task) and lesion location	Two chronic stroke cases (both female) reported hyperacusis after insular lesion. Compared to a matched control group (n = 10), these cases showed a significantly larger P3b amplitude at the mid-parietal electrode (Pz) during an auditory oddball task. Case #1 had a reduced N1 amplitude in both the auditory oddball as well as the mismatch negativity paradigms. Case #1 mentioned a comorbid tactile hypersensitivity while Case #2 mentioned a comorbid olfactory hypersensitivity.

Yadav and Ciuffreda (2014)	mTBI N = 15 Adult [25-65]	Chronic	Visual motion sensitivity	Hyper	Based on medical file records		Visual evoked potentials during a conventional visual evoked potential (P100) testing while wearing binasal occluders and/or base-in prisms	Wearing binasal occluders with or without a base-in prism when looking at a full-field checkerboard stimulus (vs. not wearing binasal occluders or base-in prisms) decreased the P100 amplitude in control subjects (n = 20). In mTBI patients with visual motion hypersensitivity (n = 15) this amplitude increased as compared to the condition where participants did not wear binasal occluders or base-in prisms, but only when wearing binasal occluders without base-in prisms. Wearing the binasal occluders resulted in a self-reported reduction of symptoms in mTBI patients with visual motion hypersensitivity.
Ciuffreda et al. (2013)	mTBI N = 10 Adult	Chronic	Visual motion sensitivity	Hyper	Not reported		Visual evoked potential (P100) during a conventional full-field visual evoked potential testing while wearing binasal occluders	Wearing binasal occluders while looking at a full-field checkerboard stimulus (vs. not wearing binasal occluders) decreased the P100 amplitude in neurotypical adults (n = 10) while in mTBI patients with visual motion sensitivity (n = 10) this amplitude increased. mTBI patients also reported less symptoms of visual hypersensitivity when wearing binasal occluders while they caused discomfort for the neurotypical adults.

Mak et al. (2005)	Stroke N = 1 Adult [70]	Chronic	Gustatory Olfactory (Temperature)	Hyper	Rating of the intensity of gustatory and olfactory stimuli		Lesion location	A stroke case with an insular lesion reported increased intensity ratings of gustatory and olfactory stimuli especially when stimuli were presented to the contralesional nostril or the contralesional side of the tongue. The stroke patient also mentioned post-stroke alterations in sensitivity to temperature.
Nölle et al. (2004)	mTBI N = 31 Adult [24-56]	Chronic	Auditory	Hyper (n = 2) and hypo (transient hearing loss) (n = 16)	Not reported		Central auditory pathway testing (recording of otoacoustic emissions, strapedial reflexes, and auditory brainstem responses)	TBI patients with hypo- or hyperacusis displayed reduced otoacoustic emissions amplitudes as compared to neurotypical adults (n = 12). Sixteen of the TBI patients also displayed abnormal strapedial reflexes. Auditory brainstem responses were normal in all TBI patients.
Studies discussing both behavioural and neural mechanisms of sensory sensitivity								

Pritchard et al. (1999)	Stroke N = 4 Adult [52-65] TBI N = 3 Adult [19-72]	Stroke: Acute TBI: Not reported	Gustatory	Not reported	Self-reported intensities of gustatory stimuli	Gustatory identification test	Lesion location	Three stroke cases with insular lesions reported lower taste intensities on the ipsilesional side as compared to the contralesional side of the tongue. These cases also showed reduced gustatory identification when stimuli were presented to the ipsilesional side of the tongue as compared to neurotypical adults. Another stroke case (who also had an insular lesion) reported no taste intensity differences between the ipsi- and contralesional sides of the tongue. She showed no taste identification impairments. Three TBI patients (with lesions in the frontal and temporal lobes without insular damage) did not report a difference in intensity for taste stimuli applied to the right or left sides of the tongue. They also showed no taste identification impairments.

Studies were ordered first on time since injury (not reported, acute, acute to chronic, chronic) and then chronologically

4. Discussion

Even though atypical sensory sensitivity after acquired brain injury is a clinically relevant symptom that can have a profound effect on quality of life or functional recovery, it is often overlooked by clinicians and researchers. This systematic review provides an overview of the existing literature on the mechanisms, prevalence, diagnosis, evolution, and treatment of sensory hypo- and hypersensitivity after acquired brain injury. Such an overview is beneficial for both clinicians and researchers as it can inform evidence-based practice, decision-making, theory building, and research initiatives. A limitation of this review is that a grey literature search was not conducted. Therefore, the results of the review may be influenced by publication bias since only published manuscripts were included. For future research it could be interesting to include the names of the diagnostic tools mentioned in Table 2 in a search string to investigate if studies that focused on concussion symptoms in general also provided relevant information on sensitivity to light or noise. However, we chose not to include such terms since an overview of diagnostic tools was not yet available prior to the execution of this systematic review, inclusion of the terms was not a priority considering the primary aims of the systematic review, and their inclusion could furthermore bias results towards research on light and noise sensitivity in mTBI as well as limit feasibility. This study has the advantage of reviewing evidence regarding *hypo*- and *hyper*sensitivity across all sensory modalities and across several types of acquired brain injury. Furthermore, we did not exclude studies based on sample characteristics such as age of the participants or time since injury. This review focuses on subjective symptoms of sensory sensitivity which are often viewed as less reliable, less valid, and more biased than objective, easily quantifiable measures. However, as is mentioned in the context of pain, sensory sensitivity is by definition subjective as it cannot be directly observed. Therefore, in our opinion, focussing on patient-reported sensory sensitivity is, to date, the best available proxy for studying symptoms of sensory sensitivity (similar to what has been described for the assessment of pain by Wideman et al., 2019). By providing an overview of the available evidence on factors related to subjective sensory sensitivity this review can inspire research on multimodal approaches to sensory sensitivity (including assessment of the behavioural and neural mechanisms of subjective sensory sensitivity).

4.1 The behavioural mechanisms of atypical sensory sensitivity after acquired brain injury

In neurotypical adults and other clinical groups, abnormal identification and discrimination of sensory stimuli, attentional impairments, and abnormal prediction of subsequent sensory stimulation are proposed behavioural correlates of atypical sensory sensitivity. However, after acquired brain injury, the literature has only provided empirical evidence for an association between atypical sensory sensitivity on the one hand, and reduced information processing and atypical sensory thresholds on

the other hand (Gudziol et al., 2014; Kumar et al., 2005; Shepherd et al., 2019). There is, to date, no evidence for an association between sensory sensitivity and reduced selective or sustained attention after acquired brain injury (Kumar et al., 2005; Shepherd et al., 2019). However, sensory sensitivity did correlate with information processing speed (i.e., time taken on attention-based neuropsychological tests) (Gualtieri & Johnson, 2006) and cognitive flexibility (in female participants) (Shepherd et al., 2019). It must be noted that Shepherd et al. (2019) used identical neuropsychological tests to measure both cognitive flexibility and attention, but the performance on these tests was operationalized in a slightly different manner (Gualtieri & Johnson, 2006). This indicates that the operationalization of performance on an attention-based task (e.g., number of errors, time taken on test) is important when considering its relationship to sensory sensitivity. Lastly, previous studies (Kumar et al., 2005; Shepherd et al., 2019) that investigated the relationship between sensory sensitivity across different modalities (visual, auditory, tactile, gustatory, and olfactory sensitivity) only used assessments of visual attention. To advance our understanding of the relationship between attention and sensory sensitivity after brain injury, studies should investigate this relationship within and across other sensory modalities. It must further be noted that the possibility remains that the underlying mechanisms that contribute to sensory hypo- and hypersensitivity after brain injury differ from those seen in other clinical groups and neurotypical adults. Further research using similar sensory sensitivity paradigms across different clinical groups as well as in neurotypical children and adults is needed to investigate whether the experienced symptoms of sensory sensitivity as well as its underlying mechanisms are similar, identical, or dissimilar across the different populations.

Studies that investigated the association between a subjective sensory sensitivity and objective identification and discrimination of sensory stimuli are sparse. Research on this relationship mainly focused on gustatory and olfactory sensitivity where subjective *hypo*sensitivity was related to reduced identification or discrimination of taste and smell stimuli (Gudziol et al., 2014). To date, it remains unclear if sensory *hyper*sensitivity is associated with a heightened identification or discrimination of sensory stimuli. Chang et al. (2007) reported that light hypersensitivity was related to a heightened critical flicker fusion frequency (but see Schrupp et al. (2009)). This means that visual stimuli that are normally perceived as constant (such as lights or computer screens), may cause discomfort because they are perceived as flickering (at a higher frequency) by hypersensitive patients. Correspondingly, using a non-LCD screen that does not flicker (but only refreshes when new content is shown) alleviated light hypersensitivity in mTBI patients (Mansur et al., 2018). Further research is needed to examine whether subjective hypersensitivity across several modalities is related to heightened sensory processing (e.g., increased identification or discrimination of sensory stimuli, reduced sensory thresholds).

4.2 Neural mechanisms of atypical sensory sensitivity after acquired brain injury

Research on the neural mechanisms of sensory *hyper*sensitivity yielded variable results. For instance, hypersensitivity has been related to structural grey or white matter abnormalities in different brain regions (e.g., the insula or the pons) (e.g., Astafiev et al., 2016; Boucher et al., 2015, Cantone et al., 2019; Likova, & Tyler, 2018) and to functional abnormalities in sensory cortices (Astafiev et al., 2016). In addition, atypical sensory sensitivity has been related to atypical event related potentials (e.g., Boucher et al., 2015; Ciuffreda et al., 2013; Yadav & Ciuffreda, 2014), central pathology (as measured using auditory reflexes) (Nölle et al., 2004), or autonomic nervous dysfunction (as measured using the pupillary light reflex) (Truong & Ciuffreda, 2016). Given the small sample size of the studies discussed above (see Table 1), replication of these results is warranted. It remains unclear how the different results can be unified into a comprehensive framework on the direct and indirect contribution of neural damage to atypical sensory sensitivity. In this regard, further research on the neuroanatomy of abnormal sensory (hypo- and hyper) sensitivity at a high spatial resolution is warranted. To distinguish whether injury to a certain region is truly associated with the symptomatology or whether it simply reflects high vulnerability to injury, it is advised that future studies consider the lesions of patients *with* as well as *without* atypical sensory sensitivity. In addition, further functional magnetic imaging research could reveal how network abnormalities or abnormal cortical activation might be related to atypical sensory sensitivity.

4.3 Potential protective and risk factors associated with atypical sensory sensitivity after acquired brain injury

To gain information about potential protective and risk factors, a second aim of the systematic review was to provide an overview of demographic variables, injury mechanisms, and comorbid symptomatology associated with atypical sensory sensitivity after an acquired brain injury. The results discussed below are based upon research about sensory *hyper*sensitivity. Firstly, we observed inconsistent results regarding the relationship between sensory sensitivity and age or sex (Brickell et al., 2017; Bunt et al., 2020, 2021; Frommer et al., 2011; Helmich et al., 2019; Hu et al., 2017; Lumba-Brown et al., 2020; Shepherd et al., 2019). These inconsistencies between studies could be due to differences in sample characteristics (i.e., time since injury), study design (i.e., diagnostic tools used to assess sensory sensitivity, sensory modalities of interest), or other factors. Furthermore, it remains unclear how we should interpret these associations: do they reflect age- and sex-related differences in underlying neural or cognitive mechanisms, in factors related to the maintenance of symptoms (e.g., illness beliefs), or in health behaviour in general? There are, for instance, indications of sex-related

differences in the relationship between sensory sensitivity and cognitive flexibility (Shepherd et al., 2019), perception of recovery (Shepherd et al., 2019), and quality of life (Colantonio et al., 2010).

To date, there is no evidence for a relationship between sensory sensitivity and education level (Elliott et al., 2018; Shepherd et al., 2019). However, there was inconsistent evidence regarding an association between place of living and auditory hypersensitivity (Shepherd et al., 2019, 2021). These results may reflect an association between sensory sensitivity and socio-economic status (which is broader than solely education level and additionally includes occupation and income (e.g., Cirino et al., 2002)), a link between sensory sensitivity and pre-injury exposure (and habituation) to sensory stimuli, or other psychosocial factors (e.g., availability of social support, pre-injury depression, and anxiety levels).

When considering medical background, there is evidence for a relationship between atypical sensory sensitivity and the number of mTBIs or medication use(e.g., Chen et al., 2019; Han et al., 2008). This indicates a possible relationship between the severity of atypical sensory sensitivity and medical (i.e., vascular or neural) or cognitive reserve. However, auditory hypersensitivity was negatively associated with injury severity (Shepherd et al., 2019) and the severity or prevalence of hypersensitivity did not differ according to the cause of the TBI (e.g., incidental causes such as falls and car accidents or causes that increase the incidence of acquiring multiple TBIs such as sport-related TBI) (e.g., Knoll et al., 2020a; Lumba-Brown et al., 2020).

Noteworthy, multiple studies found an association between atypical sensory sensitivity and symptoms of anxiety, depression, post-traumatic stress, and lower sleep quality (e.g., Al-Ozairi et al., 2015; Assi et al., 2018; Callahan et al., 2018; Callahan & Storzbach, 2019; Elliott et al., 2018; Goodrich et al., 2014; Shepherd et al., 2019). Furthermore, there is evidence for a relationship between illness beliefs such as somatization or perception of recovery and sensory hypersensitivity (Callahan et al., 2018; Nelson et al., 2018; Shepherd et al., 2019). This indicates that coping can influence the incidence or the persistence of atypical sensory sensitivity after acquired brain injury. These results seem to support the ‘anxiety hypothesis’ as well as the ‘negative affect hypothesis’ of sensory hypersensitivity (Shepherd et al., 2019). The anxiety hypothesis postulates that sympathetic overarousal (often linked to stress or anxiety) leads to a hypervigilance for environmental stimuli, whereas the negative affect hypothesis postulates that self-reported sensory sensitivity is linked to tendency to negatively appraise situations or the self. However, the causal relationship between sensory sensitivity and maladaptive coping, depression, anxiety, or stress after acquired brain injury remains unclear.

4.4 A biopsychosocial model of atypical sensory sensitivity

The results discussed above suggest that the aetiology of atypical sensory sensitivity is multifactorial and may include both biological (such as injury severity), social (such as place of living), and

psychological factors (such as anxiety, stress, coping). Therefore, we propose that a model of sensory sensitivity after an acquired brain injury should not only consider the behavioural and neural mechanisms of sensory sensitivity but also the influence of these biopsychosocial factors. It remains unclear if the relationship between these biopsychosocial factors and sensory sensitivity differs for sensory hypo- and hypersensitivity. Since previous research mainly focused on sensory hypersensitivity, more research on the mechanisms of sensory hyposensitivity is needed. Furthermore, instead of considering an identical stable pathological process that underlies atypical sensory sensitivity in each patient (a latent disease model) it is possible that the underlying mechanisms of these symptoms vary inter- and intra-individually (Rabinowitz & Fisher, 2020). For instance, in the acute phase after injury atypical sensitivity might be linked to neurogenic injury-related factors, while in the chronic phase after injury the maintenance of these symptoms might be linked to psychosocial factors (e.g., perceived social support, coping, and anxiety). Future research is needed to grasp how inter- and intra-individual differences might covary with the biopsychosocial correlates of atypical sensory sensitivity. Lastly, it must be noted that the aim of this systematic review was to investigate the underlying mechanisms of abnormal sensory sensitivity in acquired brain injury populations. The results described above provides evidence for certain behavioural, neural, and psychosocial *correlates* of sensory sensitivity. Whether these relationships are causal remains unclear and necessitates further research in larger samples (for example using lesion studies or randomized experimental designs).

4.5 The prevalence and diagnosis of atypical sensory sensitivity after acquired brain injury

As illustrated in Figure 5, there was a large variability in the reported prevalence of sensory hypersensitivity across the different sensory modalities. This variation as well as the focus on mTBI might be due to a lack of appropriate and validated diagnostic tools for sensory sensitivity. Since light and auditory hypersensitivity are known symptoms of a concussion (e.g., Tator et al., 2016), questionnaires on post-concussive symptoms (such as the Rivermead Post-concussion Questionnaire) (e.g., Potter et al., 2006) often assess light and/or noise hypersensitivity. However, as illustrated in Figure 5, atypical sensory sensitivity is not limited to light or noise sensitivity but can extend across different modalities. Furthermore, the limited number of results regarding sensory sensitivity after stroke (Alwawi et al., 2020; Carlsson et al., 2004; Carlsson et al., 2009; Chung & Song, 2016; Wehling et al., 2015) or moderate to severe TBI (Goodrich et al., 2014; Knoll et al., 2020b) indicate that atypical sensory sensitivity is also prevalent after more severe brain injury. To date, there is no validated measure that is adapted to acquired brain injury, that can be used in patients with severe cognitive disabilities, and can assess sensory sensitivity across all modalities (visual, auditory, tactile, gustatory, olfactory, vestibular). Therefore, the prevalence of atypical sensory sensitivity in other modalities, after

moderate to severe brain injury, as well as hyposensitivity in general might be underestimated due to a lack of diagnostic tools. The development of such diagnostic tools would further facilitate the assessment of sensory hypo- and hypersensitivity across different types of acquired brain injury. For instance, since the current literature is limited to TBI and stroke, it is uncertain how prevalent atypical sensory sensitivity is after hypoxia or anoxia. Furthermore, it is unclear how prevalent hypo- or hypersensitivity are across different types of strokes (e.g., stroke due to infarction vs. haemorrhage, lacunar infarction vs. severe stroke), indicating the need for further research. Lastly, research on the prevalence of abnormal sensory sensitivity in children with a brain injury was limited to four studies of which the majority investigated sport-related TBI. Further research in children and adolescents with other types of brain injury is advised, especially since these symptoms might have a large impact on the social and academic development of children.

4.6 The evolution and treatment of atypical sensory sensitivity after acquired brain injury

In contrast to its relatively high prevalence, knowledge on the evolution and treatment of hypo- and hypersensitivity after acquired brain injury is limited. There is evidence that the prevalence and severity of sensory hypersensitivity decreases within the first year after a mTBI (Barker-Collo et al., 2018; Shepherd et al., 2021) (see Figure 6), nevertheless the symptomatology remained substantial in the chronic stage after brain injury (e.g., Alwawi et al., 2020; Truong et al., 2014). The recovery of atypical sensory sensitivity after brain injury shows inter- and intra-individual variation (Alwawi et al., 2020; Rabinowitz & Fisher, 2020; Truong et al., 2014), which could be due to an influence of other covariates (such as medical background, coping, or comorbid symptomatology). Furthermore, it remains unclear whether hypo- and hypersensitivity symptoms are more prevalent in the acute phase and then recover spontaneously or whether these symptoms become more prevalent when patients leave a hospital context (which is a controlled sensory environment) and return to their sensory rich daily lives. Patients with mild acquired brain injury (such as a mTBI) often return to the sensory rich daily lives quicker than patients with severe acquired brain injury (such as a severe TBI or a stroke) (Prince & Bruhns, 2017). Therefore, mTBI patients might be confronted earlier and to a greater extent with sensory sensitivity abnormalities than patients with severe injury. The latter patients can have severe motor, cognitive, or speech impairments which are often the focus of rehabilitation. We hypothesize that this may explain the negative relationship between auditory hypersensitivity and injury severity (Shepherd et al., 2019). Future research is needed to understand if and how individual characteristics and/or underlying mechanisms might influence prognosis. Moreover, more knowledge regarding symptom evolution can guide clinical decisions on whether to offer treatment as well as when to start treatment.

An overview of the research on the treatment of sensory sensitivity consisted of a small number of studies that focused on hypersensitivity. Some studies reported that patients with visual hypersensitivity benefited from tools such as coloured glasses, contact lenses, or non-LCD screens (e.g., Clark et al., 2017; Mansur et al., 2018; Truong et al., 2014). However, the ecological validity of some of these studies (e.g., Clark et al., 2017; Mansur et al., 2018) is limited since patients did not use these tools in their daily lives but in a controlled, experimental setting in the presence of others, thus increasing the risk of observer bias. Furthermore, although these tools may provide immediate relief, their long-term effects are unclear. These treatments may indeed be detrimental in the long term. Firstly, these tools may result in increased avoidance of sensory stimuli which could impair sensory adaptation as well as might lead to using maladaptive, inflexible coping strategies. Secondly, relying on an external tool to provide symptom relief might decrease patient empowerment. In contrast, Hallberg et al. (2005) found that a treatment program consisting of psychological interventions combined with gradual desensitization to sounds in the daily lives of participants, resulted in less self-reported disabilities in TBI patients. However, since there was no control group it is not certain to what extent these effects can be explained by spontaneous recovery. Furthermore, Hallberg et al. (2005) did not include a quantitative evaluation of their recovery and did not include a follow-up assessment. Similar treatment strategies can be found in graded exposure or desensitization treatments used for chronic pain (e.g., López-De-Uralde-Villanueva et al., 2016), post-traumatic stress, or anxiety disorders (e.g., Forbes et al., 2007; McLay et al., 2011). For these clinical groups evidence-based protocols for graded exposure exist which can act as inspiration for the development of future evidence-based rehabilitation protocols for brain injury patients (e.g., Foa et al., 2009; Simons et al., 2019). Noteworthy, the described treatments do not seem to target behavioural or neural factors that may initiate the symptoms but rather focus on psychological factors related to maintenance of symptoms or providing external tools that provide relief of symptoms.

4.7 Conclusion

A better understanding of the underlying behavioural and neural correlates of sensory sensitivity as well as the biopsychosocial factors that play a role in the incidence and persistence of abnormal sensory sensitivity are essential to efficiently treat abnormal sensory sensitivity as well as predict symptom evolution. To achieve this, certain inconsistencies in the existing literature must be resolved. Ideally, similar paradigms are used across different sensory modalities, different types of brain injury, and different phases after injury (e.g., the (sub)acute and chronic phases). To date, most of the research used an unvalidated diagnostic tool to assess sensory sensitivity and assessment was often limited to light and auditory hypersensitivity after a mTBI. This again emphasizes the large need for validated diagnostic tools that are adapted to acquired brain injury patients (i.e., can be used after

mild and severe brain injury) and assess hypo- and hypersensitivity across multiple modalities. It must be noted that a hyposensitivity to vestibular, visual, or tactile stimuli might be hard to diagnose in patients with motor disabilities (e.g., hemiparesis) (e.g., Lawrence et al., 2001; Wallen et al., 2001) as well as patients with sensory dysfunctions (such as hemianopia or hemispatial neglect) (e.g., Goodwin, 2014) which are highly prevalent after an acquired brain injury. Correspondingly, the studies that assessed hyposensitivity did not indicate whether their included participants had peripheral injuries that could explain their symptoms (e.g., Nölle et al., 2004). Lastly, the terminology that is used to describe atypical sensory sensitivity showed large variation across different studies. For instance, nomenclature used to describe auditory sensitivity included hyperacusis, phonophobia, and noise sensitivity, but the definition of these concepts as well as the distinction between these concepts remain unclear (see also Hallberg et al., 2005). This highlights the need for the development of a golden standard regarding assessment that takes the aforementioned challenges into consideration, as well as a consensus regarding the definition of atypical sensory sensitivity after acquired brain injury.

Further research on effective diagnosis and treatment of atypical sensory sensitivity is of high importance. Firstly, sensory hypersensitivity is negatively related to functional recovery time and quality of life (e.g., Alwawi et al., 2020; Carlsson et al., 2004, 2009; Shepherd et al., 2020; Trulsson et al., 2003). Secondly, experiencing atypical sensory sensitivity was related to increased self-reported severity of other neurological (e.g., tinnitus) or cognitive symptoms (e.g., difficulty concentrating) (e.g., Chandran et al., 2020; Chorney et al., 2017; Elliott et al., 2018; Kumar et al., 2005; Shepherd et al., 2019). Thirdly, acquired brain injury patients report that their sensory sensitivity symptoms are often not addressed by health care providers, increasing patients' feelings of anxiety and stress (Alwawi et al., 2020; Landon et al., 2012). Since an evidence-based treatment protocol is not yet available, early interventions including adequate diagnosis and evidence-based psychoeducation are needed to facilitate recovery and adaptive coping. The development of valid diagnostic tools can advance our understanding of the aetiology of atypical sensory sensitivity as well as its prevalence, evolution, and treatment and simultaneously increase the methodological quality of future research. These advances in scientific knowledge can lead to better patient care as well as a reduction in the disabilities related to atypical sensory sensitivity after acquired brain injury.

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