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Congenital Blindness and Psychosis: A Literature Review

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ABSTRACT

Low vision has been declared by the World Health Organization (WHO) to be one of the major ophthalmologic problems requiring global attention. Low-vision patients experience a reduced vision-related quality of life due to impaired visual function. However, this condition that interfere their daily life could also make a significant effect on their mental health. Therefore, the final goal of low-vision rehabilitation is to improve the daily quality of life. However, the absence of vision at birth appears to protect against psychosis, whereas later-life visual loss appears to predispose to the development of psychotic symptoms. Thus, our purpose in this literature review is to learn how the onset of visual loss may affect patient's mental health.

1. Introduction

Low vision has been declared by the World Health Organization (WHO) to be one of the major ophthalmologic problems requiring global attention. Low-vision patients experience a reduced vision-related quality of life (VR. QoL) due to impaired visual function. However, this condition that interfere their daily life could also make a significant effect on their mental health.Therefore, the final goal of low-vision rehabilitation is to improve the daily quality of life. Accordingly, objective assessment of VR-QoL in low-vision patients is an important part of the rehabilitation process. Likewise, low vision has been associated with lower psychosocial wellbeing, manifested as a loss of interest in and enjoyment of physical activities. The age of onset of visual loss may be crucial. Strikingly, the absence of vision at birth appears to protect against psychosis, whereas later-life visual loss appears to predispose to the development of psychotic symptoms. Thus, our purpose in this literature review is to learn how the onset of visual loss may affect patient's mental health.

The bayesian brain

Predictive coding accounts of psychosis take as their starting point the assumption that the brain is a hierarchical Bayesian inference machine. In Bayesian inference, prior predictions about the world are represented as probability distributions of the causes of inputs lower in the hierarchy. These priors are then combined with data to form a posterior probability distribution. How much weight the prior has relative to the data is determined by the inverse variance of the prior probability distribution, or precision.[1]

Predictive coding approaches to the symptoms of schizophrenia

Recent accounts of psychotic symptoms in schizophrenia[2] have adopted a broadly Bayesian approach, although details of the precise deficit tend to differ between accounts. All theories focus on a neuromodulatory deficit such that the

variables in the hierarchy are inappropriately optimized. Aberrant precision of prediction errors causes a state where previously irrelevant stimuli become abnormally salient in terms of their ability to update beliefs higher in the hierarchy. In this setting, prior precision may increase to compensate for the over-weighting of sensory evidence, culminating in delusions and hallucinations. Alternatively, a primary abnormality of prior precision may underwrite false inference about the world or self.[3]

Of all sensory modalities, vision affords the perceiver the most amount of information about the world; for example, vision can convey more information about someone approaching from a distance than touch, hearing, or smell.[3] A visual prior will usually affect the perception of an object in another modality. For a fully sighted individual, it is rare for what someone hears to trump what they see. Vision confers a consistency[4] and a context to integrate data from other modalities.[5] Notably, there are cases of psychotic illness including schizophrenia following congenital cortical deafness,[6] consistent with the primacy of vision, as opposed to hearing, in organizing our multi- and supramodal world models.

Sensory impairment as a window onto psychosis

It is possible then to account for why adult visual loss can result in visual hallucinations, following visual deprivation[7] or in Charles-Bonnet syndrome. In neurodegenerative disease such as Parkinson's disease and Alzheimer's, visual impairment is also a risk factor for the development of visual hallucinations1 and indeed visual hallucinations in such disorders occur more frequently in conditions of poor ambient light.[2] Interestingly, the occurrence of complex (as opposed to simple) visual hallucinations in neurodegenerative disorders is relatively greater than in hallucinosis secondary to visual loss.[4]

When healthy adults are blindfolded, a majority of subjects report visual hallucinations, after a day.[7] Full insight—awareness of the nonveridical nature of the experience—into these experiences is maintained. In Parkinson's disease, worsening insight regarding visual hallucinations accompanies worsening neuromodulatory dysfunction[3] and disease progression into cortical areas and circuits that have been implicated in the specification of higher-level priors.[3]

When individuals deprived of tactile or visual stimulation are played meaningful auditory stimuli (such as jokes), there is a resultant decrease in psychopathology compared with those played "white noise," Psychedelic drugs like lysergic acid diethylamide (LSD), which act via serotonergic 5HT2A receptors, are potent inducers of visual hallucinations.[37] Blind subjects given LSD all experienced hallucinations in multiple modalities, although congenitally blind subjects did not report visual hallucinations. Of 13 late-blind subjects who experienced visual hallucinations, only two experienced complex hallucinations.[4]

The challenge faced by the developmentally visually deprived brain

For a congenitally blind person, there is no rich visual signal with which to shape one's priors about the world. Each of the other sensory modalities samples a much smaller part of the sensorium, in a noisier fashion, and priors must be built up, piecemeal, from the information contained therein. It is essential then that these hard-won priors (both supramodal and within individual modalities[43]) remain stable, so as to enable effective interaction within the world. Congenitally blind individuals show reduced integration between nonvisual modalities. Congenitally blind individuals evince a significantly attenuated illusion.[4] One study shows that visual deprivation altered the response properties of single neurons in the cat anterior ectosylvian sulcus, a cortical area implicated in higher-order multisensory processing: dark rearing caused a shift in the neuronal population away from neurons whose responses could be effectively driven by stimuli in a number of different sensory modalities towards neurons whose responses were primarily driven only by unisensory stimuli and which could now only be modulated by a

simultaneously presented stimulus in a second modality.[5]

Vision most clearly provides the spatial scene within which sensory data from other modalities can be most efficiently contextualized and integrated; it enables the construction of multimodal or supramodal representations. Congenitally blind individuals are less susceptible to the somatic version of the rubber hand illusion wherein, in normally sighted but blindfolded individuals, proprioceptive and tactile information are integrated to create a false sense of bodily ownership.[6]

A computational solution to the challenge of visual deprivation: evidence for increased topdown/neuromodulatory drive in congenitally blind individuals

In considering the neuronal instantiation of predictive inference in the brain, it is useful to distinguish between driving and modulatory signals. Driving signals convey information about the presence or magnitude of prediction error and are typically thought to be mediated by strong intrinsic forward connections, relying on fast AMPA receptor-mediated glutamatergic currents; driving connections elicit a spiking response in their targets.[4]

An increase in top-down modulatory signaling would be one way to ensure the stability of higher-level priors in the visually impaired brain. Carriere et al demonstrated that on a single-neuron level, visual deprivation shifts the responses of higher-level, multisensory neurons towards a profile indicative of an increased modulatory influence and a decreased sensory driving response.[4] This modulatory change may be NMDAR-dependent. A Transcranial Magnetic Stimulation (TMS) study has demonstrated that visual deprivation does indeed cause increases in NMDAR-dependent cortical excitability in humans.[5]

Furthermore, it is known that across species, early visual deprivation leads to plastic changes in cerebral cortex whereby visual cortex comes to instantiate sensory processing for other modalities.[2,3] If the relative changes in NMDAR subunit composition (along with other relevant changes in receptor function) are retained in brain areas which go on to serve nonvisual processing, then it is likely that the resulting computational changes (ie, greater influence of top-down modulation) would also affect processing in other modalities, even supramodally. This is consistent with the idea that, fundamentally, the cortex is "metamodal": rather than being specialized for a particular kind of sensory input it is specialized for a particular kind of computation.[6]

How might computational changes occurring in the visually deprived brain protect against schizophrenia?

Predictive coding theories of schizophrenia state that abnormal precision of prediction errors or priors gives rise to false inferences about one's own thoughts, movements, and even emotions manifest as hallucinations and delusions. Patients with schizophrenia have highly variable estimates of the visual consequences of their actions. This variability correlates with the strength of delusions of control. Furthermore, they rely more on external visual information than controls in making their judgments. They may have imprecise internal predictions about the sensory consequences of action, which prompts greater reliance on external cues.[71] We suggest that in visually impaired individuals this situation is reversed: relatively greater precision of internal predictions, as a consequence of the impossibility of visual calibration. This is fundamentally opposed to the low precision internal predictions posited in schizophrenia.[4]

Altered conscious vision, reality, and psychosis

So how big is the role of precision as encoding uncertainty in hierarchical predictive coding— and how this is affected by early visual experience. The explanatory scope of this formulation is appealing because it links neurodevelopment with Bayesian belief updating and the opportunity for false inference—of the sort associated with positive psychotic symptoms. A key aspect of this formulation is that it rests upon the top-down

control of precision at various levels of the cortical hierarchy. In turn, this means the brain must be equipped with predictions or beliefs about precision, namely, beliefs about beliefs. This is important because it speaks to a form of metacognition, namely, beliefs about the precision of beliefs lower in the hierarchy.[5]

The protection-against-schizophrenia model

"To see or not to see" may be the fundamental question by which we can elucidate the still unknown causes of one of the most devastating human experiences – schizophrenia. For the last 100 years, both "absent" and "perfect" vision have been associated with a lower risk for the disorder. Therefore, we argue that vision itself and aberrations in visual functioning may be fundamental to the development of the disorder and, thus, may provide important information about its causes. In this article, we present the "Protection-Against-Schizophrenia" (PaSZ) model by reviewing human visual functioning from two perspectives: (1) "Absent" vision or how congenital blindness contributes to PaSZ and (2) "perfect" vision or how aberrations in visual functioning are associated with an increased risk for psychotic symptomatology. Grading the risk for developing schizophrenia as a function of an individual's visual capacity, we argue that (early) diagnostic and capacity. On the onehand, individuals who suffer from visual deterioration (who previously had "normal" visual skills) may reduce their risk for developing schizophrenia through an improvement in visual capacity. On the other hand, individuals who suffer from visual impairment (who have never developed "normal" vision) may, in fact, reducetheir risk forschizophreniathrougha decline in visual capacity. Hence, rather than categorizing human behavior into, e.g., "normal" and "mentally disordered," as has traditionally been the practice of diagnostic manuals, our approach may be the first to use a continuous scale to represent psychiatrically relevant human behavior. This not only provides a scientific basis for more fine-grained diagnostic assessments, earlier detection, and more appropriate therapeutic assignments, but this model also outlines a trajectory for unraveling the causes of the schizophrenia disorder.[6]

Psychosis and Schizophrenia

Negative symptoms and thought disorder are also central features schizophrenia. Whilst they have received less consideration from predictive coding theorists, they can be brought into the explanatory fold.[7] In brief, thought disorder would arise when the contextual predictions that constrain cognition are imprecise (subtending aberrant prediction errors that derail one's train of thought) and negative symptoms may arise from maladaptive predictions about the consequences of one's actions: if we experience our agency unpredictably, why act at all.[7] There is a dearth of research pertaining to the precision of the relevant predictions in congenitally blind individuals, but we would predict that they would show similarly increased stability in the relevant domains.

2. Conclusion

Congenitally blind individuals will show lower psychosis-proneness than the sighted population. If congenitally blind individuals exhibit markedly stable prediction error signaling, they should show reduced schizotypic. Our biological knowledge of the computational changes outlined above will change as our basic understanding of both blindness and schizophrenia evolve.

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