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Adaptive mechanisms of the visual cortex in congenital versus acquired blindness: An EEG study.

Aisha Bello*

Department of Neurophysiology, University of Lagos, Nigeria.

***Correspondence to:** Aisha Bello**,** Department of Neurophysiology, University of Lagos, Nigeria, E-mail: a.bello@lagos.edu

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Introduction

The visual cortex, traditionally associated with processing visual input, exhibits remarkable neuroplasticity when visual information is absent due to congenital or acquired blindness. Neuroplastic changes in individuals born without sight (congenital blindness) differ significantly from those in individuals who lose vision later in life (acquired blindness). In congenital blindness, the brain develops without any visual input, often resulting in the recruitment of the occipital cortex for non-visual functions such as language, memory, and tactile processing. In contrast, individuals with acquired blindness initially develop with typical visual pathways and only later adapt to the absence of sight. Electroencephalography (EEG) provides a non-invasive means of studying these adaptations by capturing real-time neural activity patterns in the visual cortex under different sensory and cognitive tasks, thereby offering insight into the temporal dynamics of cortical reorganization [1].

EEG studies have revealed distinct differences in cortical rhythms between congenitally and acquired blind individuals, particularly in the alpha and beta frequency bands. In congenitally blind participants, resting-state EEG often shows reduced alpha activity in the occipital regions, reflecting an absence of the visual inhibition mechanisms typically mediated by this rhythm. This contrasts with acquired blind individuals, who may retain some residual visual rhythmicity depending on the duration since vision loss. During task engagement, such as auditory or tactile discrimination, both groups exhibit enhanced occipital activation, but the underlying mechanisms differ. In congenital blindness, cross-modal plasticity appears more robust, enabling the visual cortex to support higher-order cognitive functions, while in acquired blindness, compensatory changes tend to be more constrained by pre-existing visual network architecture. This differential adaptation highlights the timing-dependent nature of cortical reorganization and the importance of early sensory experience in shaping brain function [2].

Task-based EEG studies further illustrate how the visual cortex in blind individuals supports non-visual processing. For example, during Braille reading, congenitally blind subjects show increased event-related potentials (ERPs) in occipital areas, suggesting functional specialization for tactile input.

These responses are often stronger and more focused than those seen in acquired blind individuals, indicating a more complete repurposing of visual areas. Similarly, during auditory localization tasks, EEG source localization techniques have shown that the occipital cortex in congenitally blind individuals becomes selectively responsive to spatial features of sound, contributing to superior auditory spatial resolution. This repurposing is facilitated by long-range connectivity changes, as confirmed by coherence analyses and functional connectivity metrics. In contrast, acquired blind individuals show weaker occipital activation and reduced connectivity strength during the same tasks, implying that the developmental window for large-scale reorganization may close after a certain age or duration of visual experience [3].

The age of onset and duration of blindness also modulate the extent and nature of cortical plasticity. Studies indicate that the earlier blindness occurs, the more likely the occipital cortex will be recruited for non-visual tasks. This is particularly evident in EEG studies examining working memory, language processing, and attention. Congenitally blind individuals often demonstrate enhanced occipital activation during verbal memory tasks, and their EEG shows task-specific synchronization in theta and gamma bands, patterns usually not seen in sighted or acquired blind individuals. These changes suggest that early visual deprivation facilitates the emergence of new functional networks, which are maintained throughout life. In contrast, acquired blind individuals may exhibit transient EEG changes after vision loss, but these changes rarely result in a complete shift in functional role of the visual cortex. Instead, they may rely more on strengthening existing auditory or somatosensory networks without extensive occipital involvement [4].

Despite the neuroplastic potential of the brain, not all changes in the blind visual cortex are functionally beneficial. EEG studies have shown that maladaptive plasticity can also occur, particularly in acquired blindness. Some individuals develop chronic visual hallucinations, a condition known as Charles Bonnet syndrome, which is believed to arise from disinhibition and hyperexcitability in visual cortical areas. This phenomenon is reflected in abnormal EEG signatures such as increased high-frequency activity or erratic oscillatory bursts in occipital regions. Such findings highlight the dual nature of plasticity: while it can enable adaptive changes that support non-visual functioning, it may also lead to dysfunctional neural dynamics in the absence of appropriate sensory inputs. Understanding these processes through EEG not only enhances our knowledge of sensory substitution but also informs the development of rehabilitative strategies, such as neurofeedback training or sensory prosthetics, tailored to the unique neural architecture of congenital versus acquired blindness [5].

Conclusion

EEG studies of individuals with congenital and acquired blindness offer valuable insights into the adaptive mechanisms of the visual cortex under conditions of sensory deprivation. The timing of vision loss plays a crucial role in determining the extent and nature of cortical reorganization, with congenital blindness often resulting in more profound and functionally diverse recruitment of the occipital cortex. EEG signatures, including altered rhythms, enhanced event-related potentials, and reorganized connectivity patterns, provide compelling evidence of cross-modal plasticity and its dependence on early neural development. However, these adaptations are not uniformly beneficial, and maladaptive outcomes such as cortical hyperexcitability may emerge, especially in acquired blindness. Continued research integrating EEG with other neuroimaging and computational modeling approaches will be critical for unraveling the full spectrum of neural changes and for guiding personalized interventions to support sensory compensation in blind individuals.

References

1. Belanoff JK, Jurik J, Schatzberg LD, et al. Slowing the progression of cognitive

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decline in Alzheimer's disease using mifepristone. J Mol Neurosci. 2002;19:201-6.
2. Canevelli M, Adali N, Cantet C, et al. Impact of behavioral subsyndromes on cognitive decline in Alzheimer's disease: data from the ICTUS study. J Neurol. 2013;260:1859-65.
3. Edwards M, Corkill R. Disease-modifying treatments in Alzheimer's disease. J Neurol. 2023;270(4):2342-4.
4. Yang F, Jiang X, Yue F, et al. Exploring dynamic functional connectivity alterations in the preclinical stage of Alzheimer's disease: an exploratory study from SILCODE. J Neural Eng. 2022;19(1):016036.
5. Zhang Y, Wang J, Liu X, et al. Exploring the role of RALYL in Alzheimer's disease reserve by network-based approaches. Alzheimers Res Ther. 2020;12(1):1-4.

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