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Congenital nystagmus mitigates against motion sickness

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Motion sickness is provoked when ambiguity or ‘conflict’ within vestibular signals or within visual-vestibular interactions challenge the constancy of ‘uprightness’ and the interpretation of self-versus environmental motion. A familiar ‘conflict’ experience which frequently provokes nausea, is attempting to read in a car. The car motion causes passive head movement evoking the slow phase vestibular-ocular reflex which stabilises the eyes on the external environment. This reflex must be cancelled by contra-directional slow phase pursuit, to keep the eyes fixed ‘on page’. The implication of slow phase eye movements in motion sickness raises the question of susceptibility in people with abnormal eye movements.

An ideal population to study these implications are people with ‘CN’ type congenital nystagmus ¹. CN-congenital nystagmus evolves typically two to three months after birth and persists through life. It is characterized by an ‘off-target’ drift of the eyes with exponentially increasing velocity during fixation of stationary or moving objects. Resetting saccades return the eyes to target creating nystagmus.

Hence, in the underlying study we addressed the question whether people with CN-congenital nystagmus due to their eye movement abnormalities are less susceptible to develop motion sickness in comparison to standard population norms.

From November 2021 until May 2025, we recruited participants with CN-congenital nystagmus from the clinical services as well as searching the archives of the University of Basel Hospital and addressing support groups in the UK and in Germany. Severe peripheral vestibular impairment, bilateral vestibular loss, severe neurological disorders and insufficient knowledge of the project languages (German and English) were exclusion criteria. Participants consented to being filmed and completing a motion sickness susceptibility questionnaire ‘MSSQ’², probing susceptibility in land, sea and air transport and fairgrounds, and a visually induced motion sickness susceptibility questionnaire, ‘VIMSSQ’³, probing responses to dynamic visual displays. Based on a statistical power calculation we aimed at recruiting n=35 participants with CN-congenital nystagmus in total. The study was approved by the local Research Ethics Committee, Ethikkommission Nordwest- und Zentralschweiz (EKNZ) (2020-02744).

34 eligible participants with CN-congenital nystagmus (mean age 44.4 SD 17.8 years, 20 males, 14 females) participated. Their MSSQ scores were lower for both childhood (until the age of 12) (means 4.20 vs. 7.75, 95% CI of mean 1.97 – 6.44 vs. 7.07 – 8.48; corrected $p < 0.001$) and adult (during the latest 10 years of adult life) motion sickness *versus* standard population norms² (means 2.94 vs. 5.11, 95% CI of mean 1.43 – 4.46 vs. 4.51 – 5.70, corrected $p = 0.007$) [Figure 1]. CN-congenital nystagmus VIMSSQ scores were also lower than population norms³ for ‘nausea’ (means 0.56 vs. 1.06, 95% CI of mean 0.21 – 0.91 vs. 0.99 – 1.12; corrected $p = 0.018$) marginally lower for ‘headache’ (means 1.03 vs. 1.41, 95% CI of mean 0.70 – 1.36 vs. 1.35 – 1.47; $p = 0.065$) factors, but similar to normal for factors ‘dizziness’ (means 0.71 vs. 0.98, 95% CI of mean 0.39 – 1.02 vs. 0.92 – 1.05; $p = 0.184$), ‘fatigue’ (means 1.18 vs. 1.15, 95% CI of mean 0.83 – 1.53 vs. 1.08 – 1.21, corrected $p = 0.49$), ‘eye strain’ (means 1.76 vs. 1.57, 95% CI of mean 0.42 – 2.11 vs. 1.50 – 1.63, corrected $p = 0.60$) and ‘avoidance’ (means 1.00 vs. 1.16, 95% CI of mean 0.67 – 1.33 vs. 1.10 – 1.23, corrected $p = 0.43$). 44% of participants with CN-congenital nystagmus also had clinical diagnoses of migraine which usually increases motion sickness susceptibility⁴, hence underlining the protective effect of CN-congenital nystagmus, despite the high prevalence of migraine.

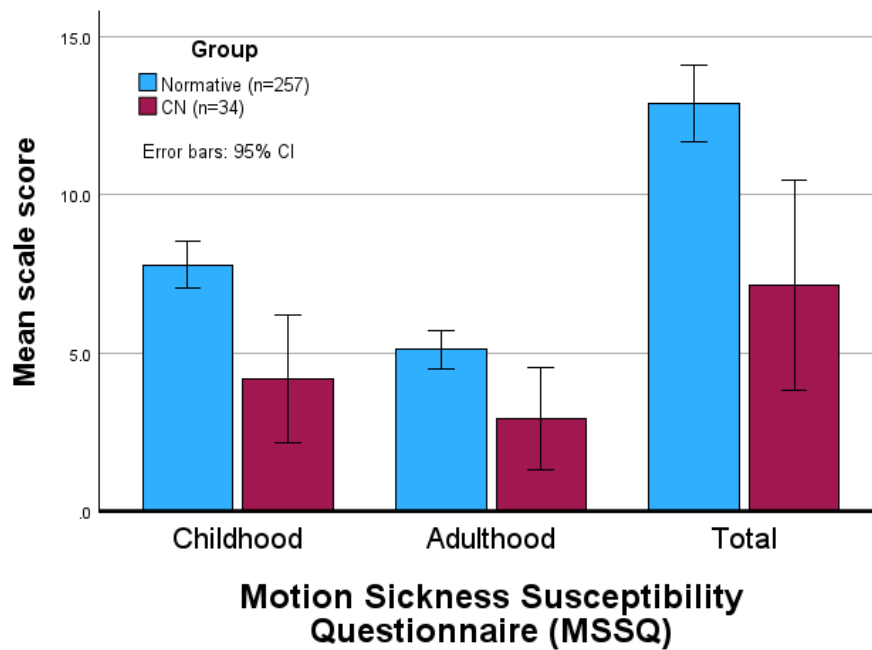


Figure 1: Motion Sickness Susceptibility Questionnaire mean scores are shown with 95% confidence intervals for 34 subjects, with ‘CN’ type congenital nystagmus in comparison with normative data of 257 individuals².

We conclude that the classical ‘nausea’ of motion sickness and the frequent secondary symptom of ‘headache’ induced by motion are absent or low in the CN-congenital nystagmus population. Evident explanations are twofold. Firstly, people with CN-congenital nystagmus could be highly adapted to motion and altered visual-vestibular interaction from early life because their visual world is unstable from birth. Secondly, slow phase eye movements are an important component of visual-vestibular control of balance and visual orientation and are organised in a forebrain-brainstem-cerebellar neural network. The disorder of CN-congenital nystagmus is hypothesized to be the result of inappropriate positive feedback gain within the network causing the eyes to ‘run off target’⁵. In CN-congenital nystagmus, network dysfunction may preclude brainstem signal conflicts that typically evoke motion sickness with nausea and vomiting.

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Conflict of Interest Statements: The authors declare no conflict of interest.

Ethics approval: This study involves human participants and the study was approved by the local Research Ethics Committee, Ethikkommission Nordwest- und Zentralschweiz (EKNZ) (2020-02744). Participants gave informed consent to participate in the study before taking part.