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Spectral differences between genotypes of Prader-Willi syndrome corroborate EEG phenotype of GABAA receptor gene deletion

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Prader-Willi syndrome (PWS) is a neurodevelopmental disorder, originating in genetic abnormalities in the paternal allele of chromosome 15q11-13, either by deletions or non-deletion abnormalities. The most consistent difference between deletion and non-deletion PWS is the absence of the genes ATP10A, GABRB3, GABRA5, GABRG3, OCA2 and HERC2 in deletion PWS, whereas they are present and functional in non-deletion PWS. Two previous studies in Dup15q syndrome and Angelman syndrome have suggested delta, beta, and possibly theta power in the resting EEG as an electrophysiological correlate of the function of these genes. We analyze resting EEG data from 85 PWS patients (54 deletion / 31 non-deletion) to further establish the relationships between the genes and EEG power. Consistent with our hypothesis, we find elevated delta and diminished beta power in deletion PWS compared to non-deletion PWS, and a marginally significant numerical trend towards elevated theta power. This finding corroborates earlier studies that discussed delta and beta power as a potential electrophysiological marker for the function of GABRB3, GABRA5, GABRG3, encoding subunits of GABAA receptors.