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THE DCDC2/INTRON 2 DELETION IMPAIRS SELECTIVELY THE MAGNOCELLULAR-DORSAL STREAM IN NORMAL-READERS

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The *DCDC2*/intron 2 deletion increases the risk to Developmental Dyslexia (DD) and DD-related phenotypes, and it is associated with brain functional and structural measures that are important for fluent reading. Illusory motion perception is specifically processed by the magnocellular-dorsal (M-D) pathway, which is impaired in individuals with DD. We tested the performance in two psychophysical tasks, tapping the M-D and the parvocellular-ventral (P-V) streams, in normal readers grouped according to the presence/absence of the *DCDC2*/intron 2 deletion ('at-risk' and 'not at-risk' groups, respectively). The M-D stream was tested by the Rotating-Tilted-Lines Illusion (RTLI) sensitivity; the P-V pathway, by a grating orientation identification task. Our data showed that the 'at-risk' group needed more contrast to process the illusory rotation in the RTLI task, while they perform similarly to the 'not at-risk' group in the grating orientation identification task. By showing that the DCDC2/intron 2 deletion influenced the inter-individual variation in the RTLI task, our data demonstrated that the function of the M-D, but not of the P-V, pathway is impaired by this genetic variant. Moreover, our data showed a link between the M-D pathway and the dorsal-phonological reading route; importantly, this correlation is not a consequence of reduced exposure to print, as it might be the case if it was found in subjects with DD, being that it has been found in normal-reading adults. Our findings demonstrated, for the first time, that a specific neurocognitive dysfunction tapping the M-D pathway is related with well-defined genetic susceptibility in normal-reading subjects.

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