REDUCED PARAHIPPOCAMPAL MORPHOMETRY IS ASSOCIATED WITH DEFICIENT WORKING MEMORY IN CHILDREN WITH CHROMOSOME 22Q11.2 DELETION SYNDROMEDiana Hobbs, David D. Stephenson Jr., Ashley F.P. Sanders, and *Elliott A. Reaton

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BACKGROUND: Children with 22q11.2 deletion syndrome (22q11.2DS) exhibit a nonverbal learning disability that includes spatial working memory (WM) impairment (Bearden et al., 2001). Hippocampal volume, a region of the brain involved in memory consolidation, is reduced in those with 22q11.2DS (Debbane et al., 2006.)

METHOD: We examined parahippocampal volume in relation to working memory in 26 children with 22q11.2DS and 37 typically developing (TD) peers ages 7 to 16 (M = 11.51 ± 3.14) using FreeSurfer's (v5.3.0) automated subcortical parcellation, the *WISC-IV* Working Memory Index (WMI), and a spatial working memory task built in E-Prime Pro V2.

RESULTS: Results revealed our two measures of working memory were positively correlated, r(47) = 0.67, p < 0.001. Furthermore, a diagnosis of 22q11.2DS predicted lower scores on the WISC-IV WMI ($\beta = -0.65$, t(45) = -5.65, p < 0.001) and performance on a computerized working memory task ($\beta = -0.54$, t(36) = -3.77, p = 0.001) controlling for age and sex. Poorer performance on the computerized working memory task was associated with smaller right parahippocampal volumes controlling for age, gender, and diagnosis (Left parahippocampus: $\beta = 0.39$, t(28) = 1.98, p = 0.06; Right parahippocampus: $\beta = 0.66$, t(28) = 3.25, p < 0.01).

CONCLUSIONS: These results indicate that the parahippocampus is associated with working memory processing, and children with 22q11.2DS have deficiency in both. These results have implications for working memory processing in children with chromosomal 22q11.2 deletion syndrome and potentially for longer-term developmental risks that include psychosis (Vorstman et al., 2015).

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References

Bearden, C. E., Woodin, M. F., Wang, P. P., Moss, E., McDonald-McGinn, D., Zackai, E., . . . Cannon, T. D. (2001). The neurocognitive phenotype of the 22q11. 2 deletion syndrome: selective deficit in visual-spatial memory. *Journal of clinical and experimental neuropsychology*, 23(4), 447-464.

Deleted:

Debbane, M., Schaer, M., Farhoumand, R., Glaser, B., and Eliez, S. (2006). Hippocampal volume reduction in 22q11.2 deletion syndrome. *Neuropsychologia*, 44 (12): 2360 – 2365.

Vorstman JAS, Breetvelt EJ, Duijff SN, et al. A cognitive decline precedes the onset of psychosis in patients with the 22q11.2 deletion syndrome. JAMA psychiatry. 2015: 72 (4):377-385.