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Year in School: Senior  
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Funding Source: McNair Scholars Program; Children's Miracle Network

## A preliminary study of brain laterality in autism via magnetic resonance spectroscopy

Autism has been regarded as a left-hemisphere dysfunction syndrome due to its prominent deficits in language and motor skills with preservation of spatial processing. The concepts of cerebral dominance and hemispheric asymmetry have been studied since the 19th century and it is well accepted that the two halves of the human brain are structurally and functionally different. To examine laterality in autism, we selected Magnetic Resonance Spectroscopy (MRS) as a suitable technique because it is non-invasive and measures important neurological metabolites. To optimize sample homogeneity, the subjects were matched for age ( $6.4 \text{ yrs} \pm 5.2$ ), diagnosis of non-dysmorphic, normal brain structure by MRI, normal EEG, normal head circumference ( $0.98 \text{ SD} \pm 1.1$ ), no history of regression, and similar IQS ( $84 \pm 7$ ). Nine autistic children and seven age matched control children scheduled for brain MRI scans were recruited for additional MRS studies under an IRB. A spin-echo PRESS chemical shift imaging sequence with echo time of 30 ms and repetition time of 1500 ms was obtained using a 1.5 Tesla Siemens MRI scanner. Bilaterally symmetrical volumes of interest (VOIs) were selected from both hemispheres in the frontal, temporal, parietal, basal ganglia and cerebellar regions. Focus was on two major resonances, N-acetyl-aspartate (NAA), which is present almost exclusively in neurons and neuronal processes and is considered a marker of axonal integrity, and choline (Cho) which is marker of cell membrane proliferation or disruption. Creatine (Cr) is used as an internal standard. We report a statistically significant increase in Cho/Cr in the autistic brain ( $p = 0.02$ ) compared to that of normal controls, and a borderline right shift in Cho/Cr ratio in the cerebellum ( $p=0.08$ ) in the subjects diagnosed with essential autism ( $n=5$ ). An increase in choline is suggestive of increased cellular proliferation, which could be a result of membrane degradation or an increase in white matter relative to grey matter in autism.