Studies published so far are consistent with the reduction of brain volume observed in Attention Deficit Hyperactivity Disorder (ADHD) patients when compared to healthy controls. Cerebral volume results from the interaction between cortical thickness and cortical surface area.

Researchers from the University of Melbourne (Australia) conducted a cross-sectional study of brain magnetic resonance imaging (MRI) in ADHD children in order to clarify which of these two parameters has a greater influence on the brain volume difference between these patients and healthy controls. Cortical thickness is influenced by the number, size and density of cortical neurons, as well as myelination at the border between white and gray matter. The cortical surface area has its dimensions mediated by the intrinsic properties of neuronal layers in each region. Finally, it should be emphasized that cortical thickness and cortical surface area have independent genetic determinants.

The study was carried out with 35 male children (median age = 13.5 years) and an equal number of controls (median age = 12.9 years). Patients were selected in the University Hospital of Melbourne and met DSM-IV diagnostic criteria. The most common comorbidities were oppositional defiant disorder and dysthymia. Twenty of these patients were drug-naive. Controls were recruited at local schools in Melbourne. Patients and controls necessarily had a quotient of intelligence equal to or greater than 70.

MRI scans were performed on a 3-Tesla Siemens TIM Trio scanner. The FreeSurfer program was used to obtain data on cortical thickness, area and volume (mm3).

The results showed that children with ADHD had lower total cortical volume (7.3%), lower cortical surface area (4.3%) and lower cortical thickness (2.8%), even when controlled for differences in intracranial volume. The greatest differences from control subjects were observed in the volume of the right and left parietal lobes. The right and left frontal cortical regions as well as the left temporal lobe also presented lower cortical thickness and volume in cases when compared to typically developed children. It should be noted that the influence of cortical thickness and cortical surface area on the volume of the cortex showed to be variable according to each region of the brain.

Cortical thickness was a more important determinant of cortical volume in parietal regions, except in the pre-cuneus, where the surface area was more relevant. This region has several connections with other parietal regions and, mainly, with the frontal lobes. Recently published studies highlight the relevant role of pre-cuneus in the neurobiology of ADHD. Finally, cortical surface area proved to be an important determinant of cortical volume also in the left pars opercularis, located in the ventrolateral prefrontal cortex.
The main limitation of this study was the selection of some patients who were using stimulant medication during MRI, what has the potential to influence the results. The authors conclude that both cortical surface area and cortical thickness have an important role on cortical volume differences between ADHD patients and controls. Considering that these two variables have different genetic determinants, it is important to study both in ADHD patients in order to clarify the neurobiology of this clinical condition.

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The work of the Melbourne researchers addresses important issues. On the one hand, there are many published works demonstrating a lower brain volume of children with ADHD when compared to healthy controls1. On the other, until now, there were no studies exploring the components of brain volume, in this case, cortical thickness and cortical surface area. The results show that there is no standard for the importance of each factor. Within the same region, as the parietal, the thickness and the area behave differently.

It is important to emphasize that regions of lower volume depicted in the present study are the same as those found in other researches, mainly frontal and parietal regions2,3. Frontal regions are classically involved in the genesis of ADHD, mainly the dorso lateral prefrontal cortex4. The parietal region has received increasing attention and this work demonstrates that ADHD children present smaller volume in this area, especially the pre-cuneus. These findings are important because they suggest a deficient functioning of the antero-posterior attentional network in the brain, which is probably involved in the genesis of ADHD.

Finally, it would be important to add a limiting factor of this study that was not mentioned by the authors. The main comorbidities cited among the patients in the sample were oppositional-defiant disorder and mood disorders. The latter is usually less prevalent than childhood anxiety disorders5. Thus, the presence of dysthymia may have influenced the findings of the study.

References


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