Brain imagery

to better understand the wide variability across the autism spectrum

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Neuroscience researchers have long attempted to uncover structural brain differences between neurotypical and autistic people. Though research in this seemingly promising field has gradually piled up, no consensus exists so far: different studies report only small differences, which rarely occur in the same brain regions. Over the past few years, the field of brain imaging has moved towards developing large databases (over 1000 brain scans) in order to increase our ability to detect differences. However, these large cohorts' main strength also happens to be their greatest weakness: gathering this many participants means combining age groups, different diagnoses, varied levels of intellectual functioning etc. Too much variety? That is the challenge we are up against! In an article published this summer, we demonstrated that observable brain differences actually depend on the autistic sub-type being studied.

In this study, anatomical brain images of 55 autistic and 37 typically developing adolescents were obtained using magnetic resonance imaging (MRI). A specialized processing software allowed us to reconstruct the surface of each participant's cortex, and to locally measure ***gyrification*** (see box). We can statistically compare these two groups, and model how gyrification evolves with age. With this last analysis, it becomes possible to take into account brain development that occurs during adolescence.

La gyrification, un indice puissant qui lie structure et fonction:

"Lissencephaly" (smooth) Mouse brain

"Gyrencephaly" (folded) Human brain

Humans have very developed brains, which only fit in our relatively small skulls because they present with numerous folds, or ***convolutions***. These bumps and ridges are extremely useful for researchers in locating different regions and follow a similar layout in all humans, though their finer structure varies from one individual to another. Gyrification refers to a measure of "brain folding" at a given point. We think it is strongly related to different cortical functions because folding depends on brain tissue thickness, and also on pressure applied by neuronal fibers, which connect different brain regions. Consequently, gyrification changes throughout brain development, and bears traces of it.

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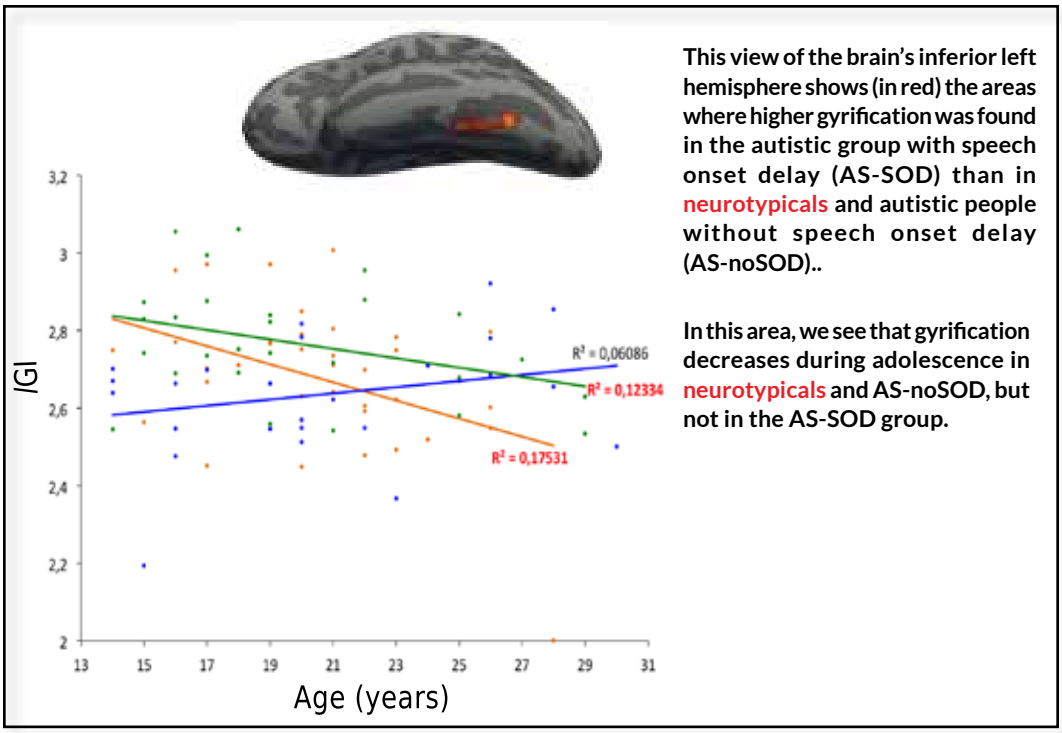
When we directly compared our autistic and typical groups, we did not find any difference in gyrification. In and of itself, this could have been our conclusion, but we chose to go a step further and to separate young autistic participants into two groups: those who had a language acquisition delay in childhood, and those who demonstrated typical speech development. Asides from their language development history, there was no difference between these two groups in symptoms or average level of intelligence: when the study was conducted, they seemed positively similar.

Why did we focus on language delays? This distinction was the basis of Asperger's syndrome diagnosis (no language delay), which is no longer used as an official category. However, some specialists continue to think that this noticeable difference in child development, and therefore probably in brain structure, is significant. Our team has evidenced several times that these two groups demonstrate strengths and interests in markedly different areas: language for ex-Aspergers, and perception (namely auditory and visual) for the others. In our study, this is demonstrated by so-called cognitive peaks: the group with speech onset delay performed significantly better than the other group on the "block" test, which is perception-based, whilst the group without language delays performed better on verbal aptitude tests. These results confirm those found in previous studies (see Sur le Spectre, issue 1).

When the autistic group is separated depending on history of speech acquisition, we not only observe direct differences in gyrification between the two autistic groups and the typical group, but also differences in gyrification changes over time. Brain development therefore follows a unique trajectory in each group. These structural differences are slight: autistic and typically developing brains are largely similar. It is however particularly interesting to notice where these gyrification differences occur: in the group with speech onset delay, it is observed in a region involved in visual perception (the fusiform) and in the group without language delay, it is in an area pertaining to the language network and specifically vocal processing (median temporal).

Example: the left fusiform region



This view of the brain's inferior left hemisphere shows (in red) the areas where higher gyrification was found in the autistic group with speech onset delay (AS-SOD) than in neurotypicals and autistic people without speech onset delay (AS-noSOD)..

In this area, we see that gyrification decreases during adolescence in neurotypicals and AS-noSOD, but not in the AS-SOD group.

We have therefore found brain regions which correspond to specific peaks of ability (language and perception) in two autistic groups, and which evolve in unique ways. It goes without saying that these results do not allow us to directly link autistic abilities to brain structure and development. They may rather spark further interest in investigating heterogeneity in the autism spectrum, and underlying differences in neurological structure. Research should take into account the distinctive features of these sub-groups and individuals instead of lumping them all together, in order to adapt interventions to developmental differences.