Researchers find brain network link between development, aging and brain disease

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Credit: Human Brain Project

(Medical Xpress)—A team of bio-researchers with members from across Europe has found evidence that suggests that grey matter development early in life tends to be the first to regress later in life—related findings also suggest a possible link between brain diseases such as Alzheimer's and schizophrenia. In their paper published in

Proceedings of the National Academy of Sciences, the team describes how they came to these conclusions after studying a large number of brain scans.

Many years ago, doctors often referred to schizophrenia as premature or early dementia. This was based on a theory first developed in the late 1800's called retrogenesis, where it was suggested that brain ability deteriorated in reverse order to how it develops. It was also believed to apply in an evolutionary sense, e.g. evolving from apes into humans. In this new effort, the researchers appear to have found evidence to back up this early theory.

The researchers analyzed MRI scans of 484 people who ranged in age from 8 to 85, looking for patterns, most specifically in grey matter, the so-called neural network of the brain—it's believed it serves to coordinate so-called high order processing, such as information related to sights and sounds. They did find a pattern—they noticed that the same parts of the grey matter that developed later in life in young people, were the first to deteriorate later on in life due to natural aging. They also found that the same brain regions were impacted by both Alzheimer's disease and schizophrenia, suggesting a possible link between the two and a link between brain disease and higher order regions of the brain. The findings also suggest that it may soon be possible to offer an early diagnosis for brain diseases, by tracking late development of grey matter.

The researchers note, that it's even conceivable that some day in the future it might be possible to prevent the brain changes from occurring in the first place if abnormally late grey matter development can be prevented. They also note that their findings suggest that environmental factors that slow neural network development could be a contributing factor to life-long ailments, and eventually to certain types of dementia.

More information: A common brain network links development,

aging, and vulnerability to disease, Gwenaëlle Douaud, *PNAS*, DOI: 10.1073/pnas.1410378111

Abstract

Several theories link processes of development and aging in humans. In neuroscience, one model posits for instance that healthy age-related brain degeneration mirrors development, with the areas of the brain thought to develop later also degenerating earlier. However, intrinsic evidence for such a link between healthy aging and development in brain structure remains elusive. Here, we show that a data-driven analysis of brain structural variation across 484 healthy participants (8–85 y) reveals a largely—but not only—transmodal network whose lifespan pattern of age-related change intrinsically supports this model of mirroring development and aging. We further demonstrate that this network of brain regions, which develops relatively late during adolescence and shows accelerated degeneration in old age compared with the rest of the brain, characterizes areas of heightened vulnerability to unhealthy developmental and aging processes, as exemplified by schizophrenia and Alzheimer's disease, respectively. Specifically, this network, while derived solely from healthy subjects, spatially recapitulates the pattern of brain abnormalities observed in both schizophrenia and Alzheimer's disease. This network is further associated in our large-scale healthy population with intellectual ability and episodic memory, whose impairment contributes to key symptoms of schizophrenia and Alzheimer's disease. Taken together, our results suggest that the common spatial pattern of abnormalities observed in these two disorders, which emerge at opposite ends of the life spectrum, might be influenced by the timing of their separate and distinct pathological processes in disrupting healthy cerebral development and aging, respectively.