Pathological brain growth patterns in Autism, and catastrophic interference in establishing long-distance connectivity

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The clinical onset of autism has recently been found to be preceded by a period of abnormally accelerated brain growth, apparently following delayed prenatal development. Findings from comparative neuroanatomy and developmental neuroscience motivate the hypothesis that this growth trajectory will give rise to deviant development of cortico-cortical connectivity. Conduction delay is proportional to brain size, and so developmental growth abnormalites provide a force that should influence development of cortical networks. The growth trajectory seen in children with autism should cause an initial increase in long-distance connectivity, and then a reduction. And the more extreme the under and overgrowth, the greater should be the initial increase in long-distance connectivity, and the subsequent reduction — and so the greater the loss of function during development. To explore this hypothesis, neural networks which modelled interhemispheric interaction were grown at the rate of either typically developing children or severely autistic children. The long-distance connections were lesioned at 'birth', restored, and lesioned again at 'age 4'. The networks that modelled autistic growth were more effected by the lesions at 'birth' — indicating a greater reliance on the long-distance connections — and those that modelled typical development were more effected by the lesions at 'age 4'.