

Overhaul of our understanding of why autism potentially occurs

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Quinn, an autistic boy, and the line of toys he made before falling asleep. Repeatedly stacking or lining up objects is a behavior commonly associated with autism. Credit: Wikipedia.

An analysis of autism research covering genetics, brain imaging, and cognition led by Laurent Mottron of the University of Montreal has overhauled our understanding of why autism potentially occurs, develops and results in a diversity of symptoms.



The team of senior academics involved in the project calls it the "Trigger-Threshold-Target" model. Brain plasticity refers to the brain's ability to respond and remodel itself, and this model is based on the idea that autism is a genetically induced plastic reaction. The trigger is multiple brain plasticity-enhancing genetic mutations that may or may not combine with a lowered genetic threshold for brain plasticity to produce either intellectual disability alone, autism, or autism without intellectual disability. The model confirms that the autistic brain develops with enhanced processing of certain types of information, which results in the brain searching for materials that possess the qualities it prefers and neglecting materials that don't. "One of the consequences of our new model will be to focus early childhood intervention on developing the particular strengths of the child's brain, rather than exclusively trying to correct missing behaviors, a practice that may be a waste of a once in a lifetime opportunity," Mottron said.

Mottron and his colleagues developed the model by examining the effect of mutations involved in autism together with the brain activity of autistic people as they undertake perceptual tasks. "Geneticists, using animals implanted with the mutations involved in autism, have found that most of them enhance synaptic plasticity – the capacity of brain cells to create connections when new information is encountered. In parallel, our group and others have established that autism represents an altered balance between the processing of social and non-social information, i.e. the interest, performance and brain activity, in favor of non-social information," Mottron explained. "The Trigger-Threshold-Target model builds a bridge between these two series of facts, using the neuro cognitive effects of sensory deprivation to resolve the missing link between them."

The various superiorities that subgroups of autistic people present in perception or in language indicates that an autistic infant's brain adapts to the information it is given in a strikingly similar way to sensory-



deprived people. A blind infant's brain compensate the lack of visual input by developing enhanced auditory processing abilities for example, and a deaf infant readapts to process visual inputs in a more refined fashion. Similarly, cognitive and brain imaging studies of autistic people work reveal enhanced activity, connectivity and structural modifications in the perceptive areas of the brain. Differences in the domain of information "targeted" by these plastic processes are associated with the particular pattern of strengths and weaknesses of each autistic individual. "Speech and social impairment in some autistic toddlers may not be the result of a primary brain dysfunction of the mechanisms related to these abilities, but the result of their early neglect," Mottron said. "Our model suggests that the autistic superior perceptual processing compete with speech learning because neural resources are oriented towards the perceptual dimensions of language, neglecting its linguistic dimensions. Alternatively, for other subgroups of autistic people, known as Asperger, it's speech that's overdeveloped. In both cases, the overdeveloped function outcompetes social cognition for brain resources, resulting in a late development of social skills."

The model provides insight into the presence or absence of intellectual disability, which when causative mutation alter the function of brain cell networking. Rather than simply triggering a normal but enhanced plastic reaction, these mutations cause neurons to connect in a way that does not exist in non-autistic people. When brain cell networking functions normally, only the allocation of brain resources is changed.

As is the case with all children, environment and stimulation have an effect on the development and organization of an <u>autistic child</u>'s <u>brain</u>. "Most early intervention programs adopt a restorative approach by working on aspects like social interest. However this focus may monopolize resources in favor of material that the child process with more difficulties, Mottron said. "We believe that early intervention for <u>autistic children</u> should take inspiration from the experience of



congenitally deaf children, whose early exposure to sign language has a hugely positive effect on their language abilities. Interventions should therefore focus on identifying and harnessing the autistic child's strengths, like written language." By indicating that autistic "restricted interests" result from cerebral plasticity, this model suggest that they have an adaptive value and should therefore be the focus of intervention strategies for <u>autism</u>.

Provided by University of Montreal

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