# Autism research plagued by major flaw

I've written a lot of stories about autism research, and I'd say one of the biggest scientific developments in the past few years was the creation of 'autistic' mice. Researchers first found many, many genes associated with autism in people, and then created dozens of mouse models that carry one or more of those same genetic glitches.

In the fall of 2011, for example, one team debuted mice with extra copies of a gene called UBE3A. Approximately 1 to 3 percent of children with autism carry extra copies of the same gene. These mutant mice show little interest in social interactions, compared with controls. They also emit fewer vocalizations and repetitively groom themselves. This was heralded as something of an autism trifecta, as the animals mimicked the three 'core' symptoms of people with the disorder: deficits in social behaviors and in communication, as well as repetitive behaviors.

The same goes for mouse models based on environmental, rather than genetic triggers. Mice whose mothers got an infection while pregnant end up with abnormal social interactions and vocalizations, and they repetitively bury marbles. Once again, the animals show all three "core" deficits, and are thus considered to be a valid model of autism.

There's a nice and tidy logic to this approach, understandably appealing to neuroscientists. If a mouse model mimics the three behaviors used to define autism, then studying the cells and circuits of those mice could lead us to a better understanding of the human disorder. But there's a big hole in that logic, according to a provocative commentary published by Eric London in this month's issue of *Trends in Neurosciences*. The problem is that the symptoms of autism — like those of all psychiatric disorders — vary widely from one person to the next. So using the fuzzy diagnostic category of 'autism' to guide research, he writes, "is fraught with so many problems that the validity of research conclusions is suspect."

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