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## Neurogenomics

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Research from the 30th Annual [Society for Neuroscience](#) Meeting in New Orleans (November 4-9) indicates that functional genomics is now being successfully used to address the issues of nervous system development and pathology. Connie Cepko, of Harvard Medical School, emphasized the particular challenges of working with the nervous system, in particular because of its extreme cellular heterogeneity. One of her favored model systems - the differentiation of photoreceptors in the mouse retina - has, however, proved to be extremely tractable (see the [The Retina Microarray Project at the Cepko lab](#)). Cepko has used microarrays and SAGE (serial analysis of gene expression; see the [SAGE Home Page](#)) to identify downstream targets of the *crx* (cone rod homeobox) gene, a locus to which human forms of blindness map. SAGE has been widely used in tumor cell profiling but rarely exploited in developmental biology or neuroscience. By sequencing a concatenated run of short, 10-14 base-pair tags from the 3' ends of sequences derived from an EST database, the gene expression profile a sample cell or tissue can be obtained. Comparison of SAGE profiles from *crx* knock-out and wild-type mice has revealed a large set of novel, candidate disease genes and potential downstream targets of *crx*.

At a 'New Neurobiology' press conference at the same meeting, Pat Levitt of the University of Pittsburgh revealed how microarray profiling has identified a single gene closely associated with schizophrenia - the first time microarray technology has been used to probe the genetic basis of a complex neurological disorder. The study, just published in [Neuron](#) (Mirnics *et al.*, *Neuron* 2000 **28**:53-67) compared the expression of over 7000 gene transcripts in the prefrontal cortex of schizophrenics and control patients. Expression of one gene, NSF, encoding a protein involved in the presynaptic secretory machinery, is consistently downregulated in the prefrontal cortex of schizophrenics. Some of the genes identified by Levitt and co-workers in this study map closely to the chromosomal regions, such as 22q11-13, already known to be linked to schizophrenia. As Levitt discussed, however, the data don't demonstrate causal relationships, as changes in the expression levels of some genes may simply represent compensation for the underlying disorder. Nevertheless, the use of a high-throughput method has pinpointed candidate genes at a rate far exceeding conventional approaches.

## References

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3. SAGE Home Page, [<http://www.sagenet.org>]
4. *Neuron*, [<http://www.neuron.org/>]