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Letter to the Editor

Strong evidence for multiple psychosis susceptibility genes – a rejoinder to Crow

There are many mis-representations of our position in Dr Crow's response (Crow, 2008) to our comment

on his paper, but given recent empirical developments in genetic research (Maher et al. 2008), there is little need, and even less time, to engage in a protracted debate on whether there is a genetic basis to psychosis. In the last 2 months, a synthesis of genome-wide data and large sample sets has convincingly shown that common genetic variants, each of weak effect, are indeed involved in schizophrenia (O'Donovan et al. 2008) and bipolar disorder (Ferreria et al. 2008). Moreover, in schizophrenia, there is now consistent and compelling evidence (The International Schizophrenia Consortium, 2008; Stefansson et al. 2008) from molecular genetics for a contribution to risk from copy number variation (CNV), variants that result in the deletion or duplication of 1000 bases or more of DNA sequence. Read with an open mind, these recent papers should change Dr Crow's views.

While we make no argument that epigenetic changes are *not* involved at all, the molecular data clearly show genetic variation *is* involved. Moreover, the molecular data clearly point to the involvement in psychosis of multiple regions of the genome, not some single sex-linked part of the genome involved in language and speciation as Dr Crow has long proposed. It must surely now be the time for Dr Crow to reject his own hypothesis of a single cause of psychosis, and to use his well-earned reputation in whatever way he can to enhance the ability of geneticists, and epigeneticists (who are often the same people), to get on with the job of tackling the complexities of psychosis for the benefits of our patients.

Declaration of Interest

None.

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