Even though the aetiology of the majority of mental disorders is unknown, bipolar disorder from early on has been thought to have a genetic causation.

The largest twin study in bipolar disorder by McGuffin and colleagues (2003) reported data from 67 bipolar probands (30 monozygotic and 37 dizygotic twins). Monozygotic twins, who share the same genetic code with each other, were found to have significantly higher rates of bipolar disorder, than dizygotic twins, who have different genetic codes.

Monozygotic concordance, where both twins sharing the same genetic code also had bipolar disorder was significantly higher than dizygotic concordance (67% MZ vs. 19% DZ). The heritability estimate was calculated to be 85% (when both twins had bipolar) or even higher to 89% (when one twin had bipolar and the other twin had unipolar).

Of course the risk from first degree relatives, where there is considerable genetic variability, is significantly lower and estimated to be approximately 5-10%, and the risk from unrelated persons even lower at 0.5-1.5% (Craddock and Jones, 1999). McGuffin et al. (2003) also argue that in comparison to twin studies of other disorders, sample sizes of twin studies in bipolar disorder are still too small to completely rule out or exactly determine the role of environmental influences.

Despite the high genetic correlation between mania and depression (0.65), mania appears to be more heritable than depression, with a heritability rate equal to bipolar disorder (85%) in the MZ twins. The hunt for the single gene has now been abandoned as most research suggests that what is inherited is multiple genes of small effect size that interact with each other (Farmer et al., 2007).

Structural brain abnormalities in bipolar patients have also been reported. These briefly include white matter hyper-intensities and differences in the size of temporal lobes, amygdala, basal ganglia, and caudate (Bearden et al., 2001). Functional abnormalities reported include differences in the activation of prefrontal areas, the striatum, and the amygdala (Strakowski et al., 2005). These are all brain areas that suggest subsequent abnormalities in the regulation of mood but it is still unclear whether these are a cause or an effect of the disorder or even a result of the different pharmacological treatments patients have to go through.

key references

Craddock, N. & Jones, I. 1999. Genetics of bipolar disorder. J Med Genet, 36, 585-94.

Farmer, A., Elkin, A. & Mcguffin, P. 2007. The genetics of bipolar affective disorder. Curr Opin Psychiatry, 20, 8-12.

Bearden, C. E., Hoffman, K. M. & Cannon, T. D. 2001. The neuropsychology and neuroanatomy of bipolar affective disorder: a critical review. Bipolar Disord, 3, 106-50; discussion 151-3.

Strakowski, S. M., Delbello, M. P. & Adler, C. M. 2005. The functional neuroanatomy of bipolar disorder: a review of neuroimaging findings. Mol Psychiatry, 10, 105-16.