BJPsych | The British Journal of Psychiatry (2015) 206, 343–344

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Edited by Kiriakos Xenitidis and Colin Campbell

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Intergenerational transmission of psychopathy

The conclusions drawn by Auty *et al*[1] – that the development and persistence of psychopathic personality characteristics is most likely down to both genetic and environmental factors – are well-judged, modest, and consistent with their results. Their report highlights a large and potentially important dataset, and the topic of intergenerational transmission of personality characteristics is underresearched.

Unfortunately, although it was not mentioned in the limitations, I am concerned that the results of the analysis remain open to confounding by genetic factors. For example, the mediation model involving disrupted employment as an intergenerational mediator of psychopathic trait from one generation to the next (male and female) was statistically significant. But, in finding that unemployment was a cause of psychopathic traits in the offspring, given that some genetic influence to psychopathy exists, and that correlation between genetic risk for psychopathy and unemployment is likely, the conclusions seem vulnerable to the alternative explanation that it is shared genetic material, not psychosocial risk factors, being measured in the models. This could be an important threat to inference. For example, in the study of intergenerational transmission of conduct problems by D'Onofrio *et al*,[2] the investigators identified evidence for complete confounding of environmental risk factors by shared genetic liability. Readers should be cautious, therefore; bringing about changes in personality structure in the offspring of psychopathic individuals by intervening in the parental psychosocial environment may be injudicious.

1 Auty KM, Farrington DP, Coid JW. Intergenerational transmission of psychopathy and mediation via psychosocial risk factors. *Br J Psychiatry* 2015; **206**: 26–31.

2 D'Onofrio BM, Slutske WS, Turkheimer E, Emery RE, Harden KP, Heath AC, et al. Intergenerational transmission of childhood conduct problems: a Children of Twins Study. *Arch Gen Psychiatry* 2007; **64**: 820–9.

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doi: 10.1192/bjp.206.4.343

Authors' reply: We agree that the intergenerational transmission of psychopathy could be driven by genetic factors as well as by environmental factors. The degree of influence that environmental factors have on intergenerational transmission of personality features and the possible confounding by genetic features are of central importance in understanding the aetiology of psychopathology.

The Cambridge Study in Delinquent Development (CSDD) has not collected any genetic material. However, it has collected detailed information on environmental measures and can provide some insight into this issue. Current work involves comparing the intergenerational transmission of psychopathy for offspring with resident fathers (up to age 16) with that for offspring with non-resident fathers. We would expect that, to the extent that the intergenerational relationship is driven by genetics, it would be just as strong for those with non-resident fathers as for those with resident fathers. To the extent that environment matters, the relationship should be stronger for those with resident fathers.

We have found that, for male offspring with a resident father, transmission of Hare Psychopathy Checklist: Screening Version (PCL:SV) Factor 1 and Factor 2 scores is strong and statistically significant ($P<0.001$). However, for males with a non-resident father, the transmission is weaker. It was significant only for the more behavioural Factor 2 scores ($P=0.021$) and not for the Factor 1 scores, which measure psychopathic personality features.

These results suggest that environmental factors might be important in the transmission of psychopathic personality features to male offspring. For female offspring with a resident father, the transmission of Factor 1 and Factor 2 scores was not significant. For female offspring with a non-resident father, the transmission of Factor 1 scores was not significant but, surprisingly, the transmission of Factor 2 scores was significant ($P=0.003$). Our results suggest that, for both male and female offspring, genetic factors may be important in the transmission of the more behavioural Factor 2 scores. However, it may be that environmental factors are more important for male offspring.

Our results agree with previous analyses of the CSDD dataset examining the intergenerational transmission of convictions. Farrington *et al*[1] found that the relationship between the convictions of same-gender intergenerational pairs was stronger than for opposite-gender pairs; father–son was stronger than father–daughter correlation, and mother–daughter was stronger than mother–son correlation.

1 Farrington DP, Barnes GC, Lambert S. The concentration of offending in families. *Legal Criminol Psychol* 1996; **1**: 47–63.

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doi: 10.1192/bjp.206.4.343a

Influences of schizophrenia risk variant rs7914558 at *CNNM2* on brain structure

A genome-wide significant variant at rs7914558, which is located in the intron of the cyclin M2 gene (*CNNM2*) on chromosome 10q24.32, has been identified in a meta-analysis of genome-wide association studies (GWAS) by the Schizophrenia Working Group of the Psychiatric Genomics Consortium (PGC).[1] Recently, the largest GWAS, combining all available schizophrenia samples in the PGC, has identified genomic loci, including the *CNNM2* gene where the genetic variant at rs11191419 ($r^2=0.608$) was the most significant.[2] Major alleles of both variants were related to risk for schizophrenia (the major G allele at rs7914558 was a risk allele).

Using Irish and Italian cohorts of patients with schizophrenia and healthy controls, Rose *et al* examined the relationships between the genome-wide significant variant at rs7914558 and neurocognition, cognitive function and brain structure.[3] They reported that the *CNNM2* risk A variant was associated with reduced self-serving bias in 256 Irish patients and 131 controls. In addition, they found the risk A allele was associated with grey matter volume in putative social cognition-related regions, such as the temporal pole and anterior cingulate cortex. The A-allele carriers had greater grey matter volume in the right temporal pole and anterior cingulate cortex in 159 Irish healthy controls, reduced grey