

Editorial

Bonamy R. Oliver

Recently, a somewhat dark shadow has been cast over research in psychology in two areas: the elusive hunt for genes responsible for complex traits (e.g., Plomin, 2013; Robinson, Wray, & Visscher, 2014), and the similarly evasive replicable finding (Open Science Collaboration, 2015). Strangely motivated by this doom and gloom, when I was asked to pull together a special issue for *Psychopathology Review*, I had one main objective: to unite findings from different genetically-informed approaches to the study of psychopathology, with a view to better understanding where we might see some light.

Around one in four adults and one in ten children experience a mental health issue within a given year, with financial, physical, social and personal costs to individuals, families and society throughout the lifespan (Mental Health Foundation, 2015).

Importantly, we know that child and adult difficulties are often not independent, with early problems conferring substantial risk for adult psychopathology. In this issue, Hannigan et al. discuss how behavioural genetic approaches can, and have, informed our understanding of longitudinal continuity. Additionally, numerous, and cumulative risk factors have long been shown to have important links with psychopathology, yet, most such studies do not consider genetic propensity, despite its clear importance for the role of risk (e.g., Rutter, 2012). Here, Thomson et al. focus on internalising difficulties to critically review work on gene-environment interaction processes within both behavioural genetic and molecular genetic frameworks. Furthermore, Pingault et al. feature a relatively new approach to understanding risk for psychopathology, using Mendelian randomisation to clarify causal links between risk and outcome within their genetic context.

While the field now lies far from the predominant view of the early- to mid- 20th century that the development of psychopathology was a function of poor parent- (read 'mother-') child relationships, our knowledge about the origins of these difficulties is still scant. Understanding causation is a knotty problem in psychology, however, advances in methodology, in recognising how naturally occurring designs can help (Rutter, 2007), and the convergence and divergence of results from multiple, complementary methods do help. Behavioural genetic studies have much to offer, enabling the disentangling of genetic and environmental causes, and consistently suggesting genetic influence for psychological traits, including symptoms of psychopathology. Importantly, while replication issues blight the broader field, findings from behavioural genetic studies are remarkably stable across studies (Plomin, DeFries, Knopik, & Neiderhiser, in press). Regrettably, these studies are sometimes misconstrued as instruments for eugenic-fuelled ideas. In fact, they are one of the best tools we have for highlighting the importance of environmental factors, because they allow us to account for genetic factors in our understanding of environmental influence. As a consequence, for example, we now know that the majority of environmentally influenced variation in psychopathology is likely to lie in nonshared environments, that is experiences -- and perceptions of experiences -- that differ between siblings in the family (Plomin, 2011).

The notion of nature and nurture working together is not new (Galton, 1874), and empirical work tells a complex tale of genetic and environmental underpinnings throughout the lifespan. Gene-environment interplay is likely critical in understanding psychopathology. As presented by Thomson et al., promising advances are being made, using both behavioural and molecular genetic designs to uncover these complexities. However, mechanisms are difficult to uncover (Dick et al., 2015): few gene-environment interactions have been reliably demonstrated, and gene-environment correlation -- the selection and modification of environmental experience as a function of genetics --

requires specific study design. Promising as progress may be, the multiplicity of gene-gene, environment-environment and gene-environment interplay is likely to be quite some puzzle. However, not being able to find the mechanism is not the same as saying it doesn't exist, it simply isn't yet found. Recently scholars have posited that psychopathologies may be better explained by a general psychopathology ('p') factor analogous to the 'g' factor of general intelligence, such that underlying mechanistic similarity across disorders when examining or hunting for biomarkers may bear more fruit (see Caspi et al., 2014).

Understanding the causes and the interplaying mechanisms responsible for the development of psychopathology, leads, ultimately, to prevention and to shaping intervention. Genetic advances have made an enormous difference to prevention and treatment in medicine, most recently and dramatically in the fight against Ebola (Gire et al., 2014). There is an increasing realisation that genetic advances may also be useful for psychopathology (e.g., Moffitt, 2005; Moffitt, Caspi, & Rutter, 2006; Plomin & Haworth, 2010). For example, Mendelian Randomisation asks interesting questions about causal environmental associations we thought we knew, which in turn has critical implications for intervention. In addition, following the relatively new model of pharmacogenetics (examining molecular genetic predictors of response to pharmacological treatments), an exciting field in its infancy is therapygenetics, using molecular genetic knowledge to better understand response to psychological therapy (Lester & Eley, 2013). Moreover, behavioural genetic designs are likely to be informative for understanding intervention effectiveness, in part because they offer so much to understanding environmental influences while accounting for genetic influence.

Those of us interested in using genetically-informed approaches in prevention and intervention for psychopathology are motivated by aspirations that we share with mental health practitioners: To modify psychopathological symptoms, understand

mechanisms of change, refine and redefine intervention approaches as a function of genetic understanding so as to increase response and improve the life chances of a substantial proportion of the population. Psychopathological traits are phenotypically and genetically intricate, and our understanding of their causes and the consequences for intervention still in its infancy. However, across approaches there is convergence, and growing light in the gloom.

References

Caspi, A., Houts, R. M., Belsky, D. W., Goldman-Mellor, S. J., Harrington, H., Israel, S., ... & Moffitt, T. E. (2014). The p factor one general psychopathology factor in the structure of psychiatric disorders? *Clinical Psychological Science, 2*(2), 119-137.

doi:10.1177/2167702613497473

Dick, D. M., Agrawal, A., Keller, M. C., Adkins, A., Aliev, F., Monroe, S., ... & Sher, K. J. (2015). Candidate gene–environment interaction research: Reflections and recommendations. *Perspectives on Psychological Science, 10*(1), 37-59.

doi:10.1177/1745691614556682.

Galton F. (1974). *English men of science: Their nature and nurture*. London: Macmillan.

Gire, S. K., Goba, A., Andersen, K. G., Sealfon, R. S., Park, D. J., Kanneh, L., ... & Scheffelin, J. S. (2014). Genomic surveillance elucidates Ebola virus origin and transmission during the 2014 outbreak. *Science, 345*(6202), 1369-1372.

doi:10.1126/science.1259657

Lester, K. J., & Eley, T. C. (2013). Therapygenetics: Using genetic markers to predict response to psychological treatment for mood and anxiety disorders. *Biology of Mood and Anxiety Disorders, 3*(4). doi:10.1186/2045-5380-3-4.

Mental Health Foundation (2015). *Fundamental facts about mental health*. Retrieved from <http://www.mentalhealth.org.uk/publications/fundamental-facts>.

Moffitt, T. E. (2005). The new look of behavioral genetics in developmental psychopathology: Gene-environment interplay in antisocial behaviors. *Psychological Bulletin*, 131(4), 533-554. doi: 10.1037/0033-2909.131.4.533

Open Science Collaboration. (2015). Estimating the reproducibility of psychological science. *Science*, 349(6251). doi:10.1126/science.aac4716

Plomin, R. (2011). Commentary: Why are children in the same family so different? Non-shared environment three decades later. *International Journal of Epidemiology*, 40(3), 582-592. doi:10.1093/ije/dyq144

Plomin R. (2013). Child development and molecular genetics: 14 years later. *Child Development*, 84, 104–120. doi: 10.1111/j.1467-8624.2012.01757.x

Plomin, R., DeFries, J. C., Knopik, V. S., & Neiderhiser, J. M. (in press). Top 10 replicated findings from behavioral genetics. *Perspectives on Psychological Science*.

Plomin, R., & Haworth, C. M. A. (2010). Genetics and intervention research. *Perspectives on Psychological Science*, 5(5), 557-563. doi:10.1177/1745691610383513

Robinson, M. R., Wray, N. R., & Visscher, P. M. (2014). Explaining additional genetic variation in complex traits. *Trends in Genetics*, 30(4), 124-132. doi:10.1016/j.tig.2014.02.003

Rutter, M. (2007). Proceeding from observed correlation to causal inference: The use of natural experiments. *Perspectives on Psychological Science*, 2(4), 377-395. doi: 10.1111/j.1745-6916.2007.00050.x

Rutter, M. (2012). Gene-environment interdependence. *European Journal of Developmental Psychology*, 9(4), 391-412. doi:10.1080/17405629.2012.661174