Staring at the (sur)face of the antisocial brain

Chris Martin-Bahr/Science Photo Library

Lancet Psychiatry 2020

Published Online
February 17, 2019
http://dx.doi.org/10.1016/S2215-0366(20)30035-3

See Online/Articles
http://dx.doi.org/10.1016/S2215-0366(20)30002-X

Despite remarkable progress in the past three decades, the aetiology of antisocial behaviour remains elusive. Using the developmental taxonomy theory of antisocial behaviour as a starting point, Christina Carlisi and colleagues[1] have made an important contribution by identifying structural brain correlates of antisocial behaviour that could be used to differentiate among individuals with life-course-persistent antisocial behaviour, those with adolescence-limited antisocial behaviour, and non-antisocial controls. Specifically, the authors report a brain-wide reduction of cortical surface area in individuals with life-course-persistent antisocial behaviour relative to participants with adolescence-limited antisocial behaviour (standardised β=–0·17 [95% CI –0·26 to –0·07], p=0·0008) and controls (standardised β=–0·18 [95% CI –0·24 to –0·11], p<0·0001). Additionally, both life-course-persistent and adolescence-limited antisocial behaviour were linked to different patterns of cortical thinning in a more restricted set of paralimbic regions relative to non-antisocial controls (life-course-persistent antisocial behaviour *vs* controls standardised β=–0·10 [95% CI –0·19 to –0·02], p=0·020; adolescence-limited antisocial behaviour *vs* controls standardised β=–0·08 [95% CI –0·16 to 0·00], p=0·039). These findings offer a considerable advance to the field and also provide an opportunity to reflect on unresolved issues concerning the use of neurobiological measures to capture and explain individual variability in antisocial behaviour. Although many challenges need to be overcome before the latter can be achieved, we restrict our focus to the issue of mapping brain structure onto function, and the application of the findings to the assessment of individuals with antisocial behaviour.

Neuroimaging has become an important tool for studying the brain correlates of antisocial behaviour. There is great interest not only in understanding how alterations in brain structure can be used to characterise individuals with antisocial behaviour, but also in how disturbances in brain functioning relate to antisocial behaviour.[2] The majority of functional neuroimaging studies have used metrics that quantify the extent to which a particular set of brain regions is involved in carrying out task-relevant computations. Once identified, researchers typically generate inferences about which cognitive functions correspond to the observed pattern of activation, meaning that the interpretation could vary depending on the theoretical framework used.[3] Structural neuroimaging studies, by contrast, have the advantage of being less dependent on such inferences, but this strength can become a weakness when researchers rely too much on using cognitive frameworks to interpret results concerning brain structure. Such an approach leans heavily on the assumption that reduced brain matter in a particular region directly translates to a disturbance in the functioning of this region. Although this line of reasoning is quite prominent in the literature, it overlooks the issue that the field of cognitive neuroscience is still searching for a good strategy to determine selective associations between brain function and structure. In other words, there is still no agreement on what exactly each brain area computes and on how to best determine what a particular part of the brain does.[4,5]

A study by Darby and colleagues[6] offers an example of one approach to bridging the function–structure gap in the context of antisocial behaviour. They found that lesions in various brain areas were linked to criminal behaviour and, importantly, that the lesions were embedded in a functional network involved in moral decision making. The rich Dunedin dataset, used by Carlisi and colleagues, allows a deeper study of antisocial behaviour-related structure–function relationships. One possibility in this regard would be to use the regions showing reduced cortical thickness in individuals with antisocial behaviour as seeds for follow-up functional connectivity analyses, which would permit probing of functional networks to investigate structure-dependent alterations, in a similar manner to Darby and colleagues. Note that such an approach has already proved to be viable in the study of psychopathic traits.[7]

With regard to the practical relevance of the study, the findings might help to move the field closer to achieving the longstanding goal of incorporating neural data into assessment protocols for antisocial behaviour.[2] They point towards the possibility that metrics of brain structure can be useful tools for improving current taxonomies of individuals with antisocial behaviour. Measuring brain structure could perhaps even be better suited for tracking the development of inter-individual

differences over time than functional activation. This notion is supported by the finding that metrics of brain structure are more consistent over time than those based on haemodynamic activity.[8]

In conclusion, Carlisi and colleagues' discovery of meaningful morphological differences between individuals with life-course-persistent and adolescence-limited antisocial behaviour offers an important advance in the use of brain metrics for differentiating among individuals with antisocial dispositions. Importantly, however, it remains to be determined whether and how measuring the brain can be used to bridge the different taxometric views and theories on the aetiology of antisocial behaviour.

We declare no competing interests.

Inti A Brazil, Macià Buades-Rotger
i.brazil@donders.ru.nl

Donders Institute for Brain, Cognition and Behaviour, Radboud University, 6525Nijmegen, Netherlands (IAB); Division Diagnostics, Research, & Education, Forensic Psychiatric Centre Pompestichting, Nijmegen, Netherlands (IAB); Collaborative Antwerp Psychiatric Research Institute, University of Antwerp, Antwerp, Belgium (IAB); Brain, Belief, and Behaviour Laboratory, Coventry University, Coventry, UK (IAB); and Department of Neurology and Institute of Psychology II, University of Lübeck, Lübeck, Germany (MB-R)

1 Carlisi CO, Moffitt TE, Knodt AR, et al. Associations between life-course-persistent antisocial behaviour and brain structure in a population-representative longitudinal birth cohort. *Lancet Psychiatry* 2020; published online Feb 17. https://doi.org/10.1016/S2215-0366(20)30002-X.

2 Brazil IA, van Dongen JDM, Maes JHR, Mars RB, Baskin-Sommers AR. Classification and treatment of antisocial individuals: from behavior to biocognition. *Neurosci Biobehav Rev* 2018; **91:** 259–77.

3 Brazil IA. Considering new insights into antisociality and psychopathy. *Lancet Psychiatry* 2015; **2:** 115–16.

4 Poldrack RA. Mapping mental function to brain structure: how can cognitive neuroimaging succeed? *Perspect Psychol Sci* 2010; **5:** 753–61.

5 Mars RB, Passingham RE, Jbabdi S. Connectivity fingerprints: from areal descriptions to abstract spaces. *Trends Cogn Sci* 2018; **22:** 1026–37.

6 Darby RR, Horn A, Cushman F, Fox MD. Lesion network localization of criminal behavior. *Proc Natl Acad Sci* 2018; **115:** 601–06.

7 Korponay C, Pujara M, Deming P, et al. Impulsive-antisocial psychopathic traits linked to increased volume and functional connectivity within prefrontal cortex. *Soc Cogn Affect Neurosci* 2017; **12:** 1169–78.

8 Elliott ML, Knodt AR, Ireland D, et al. Poor test-retest reliability of task-fMRI: new empirical evidence and a meta-analysis. bioRxiv 2019; published online June 24. DOI:10.1101/681700.