EDITORIAL COMMENTARY

Crime in Huntington’s disease

Discussion of the relative contributions of nature and nurture to development of personality is an old and controversial issue that has been given new life with the development of the field of behavioural genetics. In this context criminal behaviour represents an area where debate over the role of genetic with respect to environmental aetiologies is increasingly active. The paper by Jensen et al (this volume, pp 467–471) contributes to this debate with a stated aim of evaluating “the relative importance of the psychosocial environment and of gene coding for Huntington’s disease on the development of criminal behaviour in patients with Huntington’s disease and their relatives”. The relevance of such a study in Huntington’s disease is supported by several papers which have reported behavioural disturbances to be a particular problem in this condition. A strength of the paper by Jensen et al is that their results are based on data held in Danish national registers of criminal convictions and of Huntington’s disease. The use of a nationwide register of Huntington’s disease, although still potentially prone to distortions in reporting, provides a larger and less biased study population than samples drawn from regional Huntington’s disease clinics. This is particularly important when considering criminal and associated behaviours such as alcohol consumption that display regional variations. Using a study design comparing convictions in those with Huntington’s disease to convictions in non-affected family members with a prior 50% risk and the same early perceived threat of developing Huntington’s disease, the authors sought to explore the relative contributions to criminality of growing up in an affected family and the presence of the Huntington’s disease gene or the disease process. They found a general increase in convictions and a specific increase in drunken driving in males with Huntington’s disease compared both to non-affected male first degree relatives and male controls from the general population. Unfortunately the authors did not have data allowing distinction of convictions before from those after development of clinical disease, leaving unanswered the important question of whether behavioural disturbances predate physical, psychiatric, or cognitive abnormalities. They conclude that the increased criminal behaviour in males with Huntington’s disease may be genetic in origin, mediated through the personality changes that are a recognised feature of the disease. It should be noted however that it has also been reported that cognitive decline may significantly precede apparent disease onset and that this decline is correlated with number of trinucleotide repeats, suggesting that cognitive state may also account for some of the apparently genetic basis to behavioural disturbance. Nevertheless, and not surprisingly, the findings that no significant differences in convictions were found between female patients with Huntington’s disease, female first degree relatives and female controls, and that even within the male patients, most did not have any convictions, emphasises that simply carrying the Huntington’s disease gene is at most only a partial determinant underlying criminal behaviour. The study also serves to place in context the more anecdotal reports of increased criminality in Huntington’s disease, showing that this is only an issue in a small proportion of patients.

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