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Institutionen för Medicinsk Epidemiologi och Biostatistik

Violent crime: Addressing causation with family-based methods

AKADEMISK AVHANDLING

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ABSTRACT

Violent crime is an important public health problem that incurs major costs for society. The effect of interventions has so far been modest, often attributed to a research focus on risk factors for crime, but a relative lack of understanding of the causal mechanisms behind these factors. The four studies in this thesis attempt to address different aspects of the etiology of violent crime by using family-based epidemiologic methods.

It has long been known that antisocial behavior runs in families. In Paper I, a nested case-control was used to quantify the familial clustering of violent crime using a linkage of several Swedish total population registers. We were able to provide precise estimates of the familial aggregation among 1st, 2nd, and 3rd degree relatives, and also adoptive relations and spouses. Familial risks were moderate to strong, and were modified by gender, socioeconomic status, type of violent crime, and age at first conviction. Familial clustering suggests that genes and/or family environment influence the propensity for violent offending. In Paper II we attempted to estimate the relative importance of these factors by calculating the heritability in mixed probit regression. Comparing results from twin, adoptee-parent, adoptee-sibling, and sibling designs, and attempting to adjust for non-random mating, we found that about half the variation in violent offending could be attributed to genetic factors. We also found significant gender differences in the etiology of violent crime.

In Paper III, we discussed the interpretation of sibling comparison designs. Sibling comparisons have been hailed for their ability to adjust for family-shared confounders, but have received little attention from a methodological standpoint. In line with previous research in economy, we showed that these models are subject to several caveats, and that they may in some situations increase rather than decrease bias. The implications of this were acknowledged in Paper IV, where we analysed the association of general cognitive ability and violent crime, and adjusted for shared family characteristics through sibling comparison analysis. Taking measurement error and non-shared confounding into account, the results indicated that the association was partly confounded by factors shared by siblings, but that most of the association could not be explained by such factors.

Together, Papers I and II suggest that violent crime runs in families due to both genetic and environmental factors, and Paper IV offer some support for the hypothesis that intelligence may be one of the factors explaining this familial aggregation. The caveats of sibling comparisons pointed out in Paper III should be taken into account in future attempts at using co-twin control and other sibling designs to address issues of causality.

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