Depression and violence: a Swedish population study

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Summary

Background Depression increases the risk of a range of adverse outcomes including suicide, premature mortality, and self-harm, but associations with violent crime remain uncertain. We aimed to determine the risks of violent crime in patients with depression and to investigate the association between depressive symptoms and violent crime in a cohort of twins.

Methods We conducted two studies. The first was a total population study in Sweden of patients with outpatient diagnoses of depressive disorders (n=47 158) between 2001 and 2009 and no lifetime inpatient episodes. Patients were age and sex matched to general population controls (n=898 454) and risk of violent crime was calculated. Additionally, we compared the odds of violent crime in unaffected half-siblings (n=15 534) and full siblings (n=33 516) of patients with the general population controls. In sensitivity analyses, we examined the contribution of substance abuse, sociodemographic factors, and previous criminality. In the second study, we studied a general population sample of twins (n=23 020) with continuous measures of depressive symptoms for risk of violent crime.

Findings During a mean follow-up period of 3·2 years, 641 (3·7%) of the depressed men and 152 (0·5%) of the depressed women violently offended after diagnosis. After adjustment for sociodemographic confounders, the odds ratio of violent crime was 3·0 (95% CI 2·8–3·3) compared with the general population controls. The odds of violent crime in half-siblings (adjusted odds ratio 1·2 [95% CI 1·1–1·4]) and full siblings (1·5, 95% CI 1·3–1·6) were significantly increased, showing some familial confounding of the association between depression and violence. However, the odds increase remained significant in individuals with depression after adjustment for familial confounding, and in those without substance abuse comorbidity or a previous violent conviction (all p<0·0001). In the twin study, during the mean follow-up time of 5·4 years, 88 violent crimes were recorded. Depressive symptoms were associated with increased risk of violent crime and a sensitivity analysis identified little difference in risk estimate when all crimes (violent and non-violent) was the outcome.

Interpretation Risk of violent crime was increased in individuals with depression after adjustment for familial, sociodemographic and individual factors in two longitudinal studies. Clinical guidelines should consider recommending violence risk assessment in certain subgroups with depression.

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Introduction

Depression is associated with increased risk of a wide range of adverse outcomes, including reduced life expectancy,[1] suicide,[2] self-harm,[3] acute myocardial infarction,[4] and a worse prognosis for comorbidities, such as heart disease and diabetes.[5,6] Clinical experience and expert opinion[7] also suggest an association with the risk of perpetrating violence, including homicide in male perpetrators.[8] Consistent with this, community surveys in the UK,[9] register-based investigations in Australia,[10] and cohort studies in the USA[11] and New Zealand[12] report a link with violent outcomes. However, this finding is not consistent and no association was identified in a recent US longitudinal study with lifetime[13] or past year[14] diagnoses. Moreover, in studies showing associations, they have been largely confounded by comorbid alcohol or drug use[13] or sociodemographic factors,[15] or primarily noted in individuals with psychotic depression.[16]

The probable reason for these inconsistencies could be that many influential studies have included large proportions of inpatients, where the actual reason for admission might have been risk of violence to others, suicidality, psychosis, or comorbid substance abuse. Because these are strong risk factors for violence,[17] they will amplify and perhaps explain any effects. Some studies have tried to control for these confounders, but none to our knowledge have also adjusted for familial effects. Familial effects could be a further explanation for the reported association with depression and could arise from common genetic predisposition or shared early environmental adversity. Mediation of mechanisms such as impulsivity and mood instability could be important as common causes of both depression and violence.[18]

To clarify these uncertainties, we conducted two complementary studies that benefit from use of databases available for research in Sweden. In the first, we longitudinally followed up patients with an index diagnosis of depression to determine risks of violent crime; only outpatients were included to avoid the probable biases associated with inpatient samples. Risks of violent crime

were also investigated in non-depressed siblings to determine the extent of familial confounding, and a comparison was made with risks from suicide mortality. In the second study, we investigated the association between depressive symptoms and violent crime in a cohort of twins. These studies accordingly control for the major confounds we identify in the existing literature. Because clinical guidelines are inconsistent about assessment and management of violence risk in major depression, and lack information about risk factors,[19,20] by contrast to self-harm and suicide for which risk assessment is clearly highlighted in guidelines[19–21] and expert opinion,[22] we investigated such rates in the same cohort to compare risks across outcomes where clinical guidelines provide differing recommendations.

Methods

Study design and participants

In this total population study, we linked several longitudinal, nationwide Swedish Patient Register: the National Patient, the Multi-Generation, the National Cause-of-Death, the Swedish Twin, and the National Crime Registers. The Multi-Generation Register connects every person born in Sweden in or after 1933 and ever registered as living in Sweden after 1960 to their parents.[23] Similar information exists for those immigrants who became citizens of Sweden before age 18 years, together with one or both parents. Linkage of registers is possible because all residents including immigrants have a unique ten-digit personal identification number that is used in all national registers. We selected a cohort of individuals born between Jan 1, 1958, and Dec 31, 1994, who were followed from Jan 1, 2001, to the end of follow-up on Dec 31, 2009. National outpatient coverage in the Patient Register started on Jan 1, 2001, which was the reason that we started our follow-up at that time.

Using the Multi-Generation Register, we also identified patients with depression who had siblings and half-siblings without depression during the same period.

Using the Swedish Twin Register, we identified young adult to middle-aged (aged 18–47 years) twins born between Jan 1, 1959, and Dec 31, 1986, who had participated in the Study of Twin Adults: Genes and Environment (STAGE)[24] or the Swedish Twin study of CHild and Adolescent Development (TCHAD).[25] In total, 23 020 individuals from 15 298 twin pairs (5574 monozygotic and 9724 dizygotic pairs) were included in our study. We determined zygosity with DNA testing or validated zygosity questionnaires.

Cases with depression were identified from the National Patient Register as having at least two outpatient episodes between Jan 1, 2001, and Dec 31, 2009, according to International Classification of Diseases [ICD]-10 (codes F32–F33·9).[26] We excluded individuals with inpatient episodes of depression to avoid reverse causality (because violence and aggression might precipitate admission), and those with inpatient or outpatient diagnoses of schizophrenia, schizophrenia-spectrum, and bipolar disorder between Jan 1, 1969, and Dec 31, 2009.

In the twin study, we measured depressive symptoms with a short form of the Center for Epidemiologic Studies Depression (sCESD) scale.[27] The sCESD scale included 11 items, and every item was rated on a 4-point scale (0=not at all or almost not at all; 1=rather rarely; 2=quite often; 3=all the time or almost all the time). A sum score was created based on the 11 items, with good reliability (Cronbach's α=0·86). 21 451 (93·2%) of the twins answered the questionnaires in 2005 (22 in 2004 and 1547 in 2006), and they were all followed for any outcome through linkage to the Crime Register.

The Regional Ethics Committee at the Karolinska Institutet approved the study (2009/939-31/5). Data were merged and anonymised by an independent government agency (Statistics Sweden), and the code linking the personal identification numbers to the new case numbers was destroyed after merging, so informed consent was not required.

Diagnostic validity

Data from the Swedish Patient Register diagnoses have good to excellent validity for a range of conditions, including bipolar disorder[28] and schizophrenia.[29] Overall, the positive predictive value of the inpatient register, in a recent review, was identified to be 85–95% for most diagnoses.[30] For the purpose of this study, we examined the validity of diagnoses of depression in a separate sample of patients with depression, by comparing concordance rates between patient register diagnoses (as we have used) and another clinical register that provided standardised consensus diagnoses involving comprehensive court-ordered multidisciplinary evaluations during 4 weeks in inpatient settings[31]—these acted as a gold standard. In this sample of 3059 patients assessed between 1996 and 2001, we noted fair to moderate agreement (κ of 0·32; 88% full agreement).

The mean sCESD depression score of twins who had a lifetime diagnosis of depression from the National Patient Register (mean score 13·1, 95% CI 12·7–13·6) was substantially higher than that of twins without a diagnosis of depression (7·0, 6·9–7·1).

In terms of reliability, in a previous register-based study, only around 1% (13 669 of 1 421 765) had missing personal identification numbers.[32]

Measures

Data for convictions for violent crime between Jan 1, 1972, and Dec 31, 2009, were retrieved for all individuals in the cohort from the National Crime Register, which includes conviction data for everyone aged 15 years (the age of criminal responsibility) and older. These data were extracted both before (as covariate) and after diagnosis (as outcome) of depression. Conviction of a violent offence was defined as homicide and attempted