Trauma and violent offending among adolescents: a birth cohort study

Kirsi Peltonen [ID],[1] Noora Ellonen,[1] Joonas Pitkänen [ID],[2] Mikko Aaltonen,[3] Pekka Martikainen [ID][2]

ABSTRACT

Background Earlier studies, based on data collected among juvenile court clients or prisoners, suggest that there is an association between trauma and adolescent-onset offending. However, there is a lack of large-scale data on juvenile violence and clinical mental health observations with unselected participants, and a risk-factor-oriented research combining multiple variables affecting violent behaviour.

Methods We analyse the effect of trauma on violent offending using longitudinal register-linkage population data. The study is based on administrative data on all Finnish children born between 1986 and 2000, linked with their biological and adoptive parents (N=913 675). The data include annually updated demographic and socioeconomic information from Statistics Finland, hospital discharge and specialised outpatient service records as well as the data from all suspected criminal offences known to the police (1996–2017). We measured trauma diagnosis at age 12–14 and followed participants for subsequent violent criminality from age 15 to 17.

Results The population average estimates, taking into account observed substance abuse and other mental health diagnoses, shows that trauma-related disorders (adjustment problems, post-traumatic stress disorder and acute stress disorder) were associated with violent offending. The same was true in sibling fixed effect models, which take into account genetic and environmental confounding shared by siblings.

Discussion These results suggest that severe stress related to traumatic or strong negative life changes in adolescence is a risk factor for violent behaviour.

INTRODUCTION

Starting from Widom's[1] pioneering study demonstrating a clear link between trauma and antisocial behaviour, several studies have shown an interlink between traumatic experiences, such as childhood maltreatment and criminal behaviour.[2 4] There is also emerging evidence of gene–environment interaction effects in humans that alter the developing brain in ways that modify the risk of antisocial outcomes, including violence.[5 7] In neural, autonomic and information-processing systems, evolutionarily selected response patterns characterise normal responding and are constantly adapted. In psychopathology, however, these patterns deviate from the expected course. The reasons for individual differences in these responses arise from genetic polymorphisms, adverse environmental experiences early in life and their interactions.[8]

Goff, Rose, Rose and Purves[9] found in their systematic review of epidemiological studies that the prevalence of post-traumatic stress disorder (PTSD) among sentenced prisoners is higher than in the general population. In that study, PTSD, prior to detention, was most commonly related to witnessing violence, having been threatened with a weapon, or being in a situation where they thought they or someone close to them was going to be badly hurt or die. The review of Vermeiren[10] showed that although psychopathology in general was more severe in life-course persistent offenders, also adolescent-limited offenders had significant and potential harmful levels of psychopathology. In an American study of Abram et al[11] nearly all (n=1829) youth arrested and detained in the juvenile justice system have experienced traumatic events prior to their detention often leading to PTSD and comorbid disorders.

A few studies have also investigated the rates of trauma diagnosis in co-occurrence with substance abuse demonstrating that incarcerated men with substance misuse problems and PTSD are more likely to have higher recidivism rates and higher risk for remaining entrenched in the criminal justice system than men without such problems.[12 13] Assink et al[14] performed a series of multilevel meta-analyses to examine the associations of several risk domains for life-course persistent-limited and adolescence-limited offending. They included 55 studies reporting on 1014 effects of risk factors and classified each factor into one of 14 risk domains. Consistent and large effects were found for the criminal history, aggressive behaviour and alcohol/drug abuse domains, whereas more modest effects were found for the family, neurocognitive and attitude domains in both types of offending. Surprisingly, trauma diagnosis or mental health were not explicitly included, although strongly related to offending.

Overall, earlier studies confirming the association between trauma and adolescent-onset offending are typically based on data collected among persons in juvenile court, prisoners or in different kinds of youth facilities.[15 18] However, because a large part of police cases are closed before they end up in courts and result in prison convictions, such data sets possibly result in severely biased population-level estimates of trauma and criminal behaviour. Only Sariaslan et al[19] have previously used longitudinal population-based data and individual fixed effects models to show that triggers—including exposure to violence, traumatic brain injuries, unintentional injuries, self-harm, substance intoxication and parental bereavement—contributed to elevated relative risks of violent crime among patients with psychotic disorders.
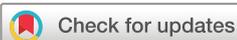
What the literature on trauma and violent offending is still missing are as follows: (1) large-scale population-level data of unselected participants followed for trauma diagnosis, clinical mental health and juvenile violent behaviour, (2) research simultaneously assessing the effects of multiple risk-factors affecting violent behaviour and (3) research that takes into account confounding factors using quasi-experimental designs. We contribute to these shortcomings by analysing the effect of trauma on violent offending using longitudinal register-linkage population data of young Finns (N=908 140), which combines data from hospital, outpatient service and police registers. We provide population averaged models to take account of observed substance abuse and other mental health diagnoses, and sibling fixed effects models to take account of all observed and unobserved confounding factors shared by siblings.

METHODS

The study is based on administrative data on all Finnish children born between 1986 and 2000 and alive in 2000 (N=936 333), linked with their biological and adoptive parents. We limit our analyses to those residing in families at the age of 12 (N=917 804). We exclude children who do not have information on mothers (N=2049), parental education (N=693) or who do not have any information between ages 15 and 17 (N=1387). The final study population is 913 675 children. The data is longitudinal and includes annually updated sociodemographic and socioeconomic information from Statistics Finland (1987–2017) and hospital discharge (1987–2017) and specialised outpatient service (1998–2017) records of the National Institute for Health and Welfare (THL). In addition, the data contains all suspected criminal offences known to the police (1996–2017). We measured trauma diagnosis at ages 12–14 and followed for subsequent violent criminality from ages 15 to 17.

We defined violent criminality from suspected offences reported to the police. We include all types of physical non-lethal and lethal violence (excluding sexual violence) and follow violent criminality from ages of 15 to 17. In Finland, the age of criminal responsibility is 15. Violent criminality was coded as a binary outcome variable (0= no violent criminality at ages 15–17, 1=at least one reported violent act at ages 15–17).

Trauma diagnosis was defined from hospital discharge records and specialised outpatient healthcare records (adjustment disorder, PTSD and acute stress disorder). Following the International Classification of Diseases Volume 10[20], all the episodes with diagnosis code F43 (reaction to severe stress and adjustment disorders) as primary or secondary diagnosis are included. This category differs from others in that it includes disorders identifiable on the basis of not only symptoms and course but also the existence of one or other of two causative influences: an exceptionally stressful life event producing an acute stress reaction or a significant life change leading to continued unpleasant circumstances that result in an adjustment disorder.[20]

We built nested linear regression models to estimate the effects of trauma on violent offending (online appendix 1). All models were fitted for the whole sample as well as stratified by gender. We estimated both standard linear probability models (LPMs) and sibling fixed effects models. In Model 1, we adjusted for gender and year of birth and prior violent crime, in Model 2 further for parental education and childhood family income at age of 12 and in Model 3, we controlled for other psychiatric diagnoses (F20–F69, F80–F99 excluding trauma diagnosis F43) and substance abuse (F10–F16, F18–F19; T40; T43; T423, T424, T426, T427 T51, R780-R784; Z502, Z504, Z715, Z715, Z721, Z722, X45, Y90, Y91) at ages 12–14.

Sibling fixed effects models were fit based on 639 688 children in sibships to control for unobserved time-invariant genetic and environmental confounding shared by sibs. Models were estimated for the whole sample as well as for brothers and sisters. In first sibling fixed effects model, we adjust for gender and year of birth prior to violent crime and other psychiatric diagnoses and substance abuse diagnosis at ages 12–14 and in second model further for parental education and childhood family income. Essentially, these models use information on siblings discordant on the exposure and thus compare the rates of violent crime of exposed children to their sibling unexposed to trauma. By restricting the comparison to siblings, the models indirectly control for effects of all shared environmental and biological factors (including genetic factors). Given that we use both nested population-level models and sibling fixed effects models, the regression coefficients from LPM ensure that comparisons between models have the same scale (risk differences).[21] In the case of sibling fixed effects models, LPM was useful since it includes also the sibling pairs with no response variability, whereas logit models are limited to the pairs with variability in both exposures and outcomes. We present our results from linear models as regression coefficients that are interpreted as risk differences in probabilities of violent crime.

RESULTS
Descriptive statistics

The prevalence of trauma diagnosis at ages 12–14 was 0.4% (n=3860) in the population, 0.5% (n=2258) among girls and 0.3% (n=1602) among boys. Trauma prevalence decreased along with higher parental education (basic education 0.77% (n=433) to tertiary education 0.33% (n=851)). The prevalence of trauma was higher among adolescents with other mental health diagnosis (4.7%, n=1963) or substance use diagnosis (4.3%, n=165) compared to adolescents with no other mental health diagnosis (0.2%, n=1897) or substance use diagnosis (0.4%, n=3695). (table 1).

The prevalence of violent crime at ages 15–17 was 1.7% (n=15 104) in the population, 1.0% (n=4265) among girls and 2.6% (n=12 275) among boys. Rate of criminal behaviour decreased along with higher parental education (basic education 5.1% (n=2827) to tertiary education 0.79% (n=2031)). Prevalence of violent crime was higher among adolescents with other mental health diagnosis (5.7%, n=2377) or substance use diagnosis (12.2%, n=467) compared to adolescents with no other mental health diagnosis (1.6%, n=14 163) or substance use diagnosis (1.8%, n=16 073) (table 1.) Trauma diagnosis more than tripled the relative risk of violent crime. Among adolescents who had a trauma diagnosis, the prevalence of violent crime was 6.5% (n=251), whereas among those without diagnosis, the prevalence was 1.8% (n=16 289). Same descriptive information on sibling data is presented in table 2. Trauma prevalence is very similar in general and in accordance with background factors in the sibling data.

Main effects

The risk difference in violence between adolescents with and without trauma was 0.043 when the model was adjusted for year of birth and prior violent crime (model 1). The risk difference attenuated slightly (0.040, p<0.001) in model 2 that further adjusted for parental education and childhood family income at the age of 12. In the fully adjusted model (model 3), the risk

Table 1 Descriptives of Population Data

	Trauma diagnosis (ages 12–14)				Violent crime (ages 15–17)				Total	
	n	Absolute risk (%)	RD (95% CI)	RR	n	Absolute risk (%)	RD (95% CI)	RR		%
Gender										
Girl	2258	0.5	0.16 (0.13,0.19)	1.5	4265	1	−1.68 (−1.74,-1.62)	0.4	466 179	51
Boy	1602	0.3	ref.	1.0	12 275	2.6	ref.	1.0	447 496	49
Parental education										
Basic	433	0.8	0.44 (0.36,0.52)	2.3	2827	5.1	4.26 (4.06,4.46)	6.4	55 963	6.1
Upper sec.	1802	0.5	0.16 (0.12,0.19)	1.5	8796	2.4	1.59 (1.53,1.65)	3.0	369 163	40.4
Lower tertiary	774	0.3	0.00 (−0.03,0.04)	1.0	2886	1.2	0.45 (0.40,0.51)	1.6	231 766	25.4
Upper tertiary	851	0.3	ref.	1.0	2031	0.8	ref.	1.0	256 783	28.1
Other mental health diagnosis (ages 12–14)										
Yes	1963	4.7	4.50 (4.30,4.71)	21.7	2377	5.7	4.09 (3.87,4.32)	3.5	41 577	4.6
No	1897	0.2	ref.	1.0	14 163	1.6	ref.	1.0	872 098	95.5
Substance use diagnosis (ages 12–14)										
Yes	165	4.3	3.91 (3.26,4.56)	10.6	467	12.2	10.46 (9.41,11.50)	6.9	3821	0.4
No	3695	0.4	ref.	1.0	16 073	1.8	ref.	1.0	909 854	99.6
Trauma diagnosis (ages 12–14)										
Yes					251	6.5	4.71 (3.93,5.49)	3.6	3860	0.4
No					16 289	1.8	ref.	1.0	909 815	99.6

RD, risk difference; RR, risk ratio.

Table 2 Descriptives of Sibling Data

|	Trauma diagnosis (ages 12–14)				Violent crime (ages 15–17)				Total	
	n	Absolute risk (%)	RD (95% CI) (%)	RR	n	Absolute risk (%)	RD (95% CI) (%)	RR		%
Gender										
Girl	1395	0.4	0.14 (0.11,0.17)	1.5	2693	0.9	−1.64 (−1.71,-1.58)	0.3	313 755	49
Boy	989	0.3	ref.	1.0	8147	2.5	ref.	1.0	325 933	51
Parent education										
Basic	230	0.7	0.43 (0.33,0.53)	2.4	1636	5.2	4.46 (4.18,4.74)	6.8	31 263	4.9
Upper sec.	1068	0.4	0.12 (0.08,0.16)	1.4	5712	2.3	1.50 (1.43,1.58)	2.9	250 742	39.2
Lower tertiary	505	0.3	−0.01 (−.05,0.03)	1.0	2028	1.2	0.43 (0.36,0.49)	1.6	168 783	26.4
Upper tertiary	581	0.3	ref.	1.0	1464	0.8	ref.	1.0	188 900	29.5
Other mental health diagnosis (ages 12–14)										
Yes	1200	4.5	4.35 (4.09,4.60)	23.5	9425	1.5	3.82 (3.54,4.09)	3.5	26 435	4.1
No	1184	0.2	ref.	1.0	1415	5.4	ref.	1.0	613 253	95.9
Substance use diagnosis (ages 12–14)										
Yes	105	4.3	3.96 (3.15,4.78)	12.1	288	11.8	10.19 (8.91,11.48)	7.2	2431	0.4
No	2279	0.4	ref.	1.0	10 522	1.7	ref.	1.0	637 257	99.6
Trauma diagnosis (ages 12–14)										
Yes						6.6	4.95 (3.94,5.96)	4.0	2384	0.4
No						1.7	ref.	1.0	637 304	99.6

RD, risk difference; RR, risk ratio.

difference for violent crime between adolescents with trauma and without trauma was 0.024 (p<0.001), when gender, age, parental education, income, prior violent crime, other mental health diagnosis and any substance use diagnosis are taken into account. This attenuation suggests that an important part of the bivariate association is related to other differences between exposed and non-exposed sample individuals, but even after controls, trauma is linked to a higher risk of later violent behaviour.

Among boys, risk difference for violent crime between adolescents with trauma and without trauma was 0.054 (p<0.001) in model 1 and 0.049 (p<0.001) in model 2 and finally 0.029 (p<0.001) in a full model (model 3). Among girls, risk difference

for violent crime between adolescents with trauma and without trauma was 0.035 (p<0.001) in model 1 and 0.034 (p<0.001) in model 2 and finally 0.023 (p<0.001) in a full model (model 3). Figure 1 shows the risk differences and 95% CIs of violent criminality by trauma diagnosis across all the models.

As a sensitivity analysis, we also estimated models excluding participants who had committed a violent crime before the age of 14, because adolescents with early criminal careers may be very different from adolescents with no experience of early criminal history. Risk differences that were found to be significant in the main analyses were also significant in the additional analyses (n=6344), but estimates for trauma diagnosis were slightly
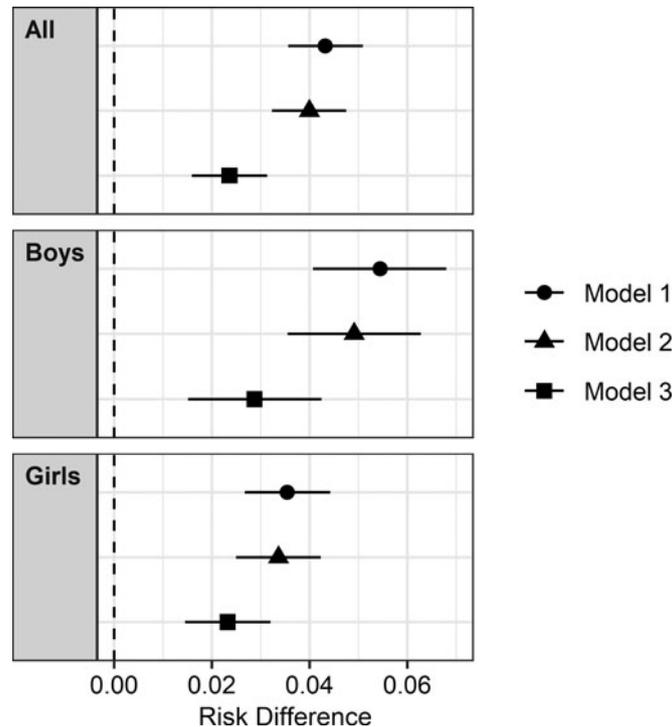
Figure 1 The risk differences and 95% confidence intervals of violent criminality by trauma diagnosis across all population models.

smaller except in sister models in sibling data, where estimates were slightly higher (results not shown).

Finally, we fitted fixed effects models based on 639 688 children in sibships to control for unobserved time-invariant genetic and environmental confounding. In these models examining only siblings discordant on the trauma exposure, the risk difference for violent crime between adolescents with trauma and without trauma was 0.014 ($p<0.05$) in a full model including all siblings and additionally adjusting for gender, age, parental education, income, prior violent crime, other mental health diagnosis and any substance use diagnosis. This suggests that the excess violence risk associated with trauma is further attenuated when we control for unobserved factors shared by the siblings.

Among brothers, the risk difference for violent crime between adolescents with trauma and without trauma was 0.024 ($p=0.055$) in the fully adjusted model, whereas among sisters the risk difference was 0.028 ($p<0.01$) in the full model. Although the p value for the risk difference among boys is slightly over the conventional threshold of statistical significance, overall the results suggest that the association between trauma and later violence is robust to controls for unobserved familial confounding, and the gender-specific associations with violence are comparable to the estimates obtained from the fully adjusted population models. Figure 2 shows the risk differences and 95% CIs of violent criminality by trauma diagnosis across all the sibling fixed effects models.

DISCUSSION

In this study, we analysed the effect of trauma diagnosis on violent offending among 15-year-old to 17-year-old adolescents in Finland. We used large-scale population-level data of unselected participants followed for trauma diagnosis, clinical mental health and juvenile violent behaviour. Our data contained all suspected criminal offences known to the police in 1996–2017. We measured trauma diagnosis at ages 12–14 and followed for subsequent violent criminality from ages 15 to 17 using large-scale, register-linkage population data of juvenile violence and clinical mental health observations.

Assessing the effects of multiple risk factors simultaneously and taking into account confounding factors revealed new information on trauma and violence. Our results showed that trauma-related disorders were strongly associated with violent offending even after controlling for demographic factors, other mental health diagnosis and substance use disorders and finally the genetic factors. The findings support the idea that severe stress related to traumatic or strong negative life changes in adolescence are risk factors for violent behaviour. A strength of the study was that we were able to identify siblings in our data and thus to estimate sibling fixed effects models that control for all confounding factors shared by siblings. We found similar results in our sibling models as in population averaged models, suggesting that the association between trauma and later violence is robust to controls for unobserved familial confounding. It is notable, however, that the excess violence risk associated with trauma was attenuated when unobserved factors shared by the siblings were controlled for.

Our results concur with Goff et al's[9] review showing higher prevalence of PTSD among sentenced prisoners compared to the general population. In our sample, the prevalence of PTSD as such was very low, which is most probably due to general trend of using adjustment disorder as a main diagnosis for under-aged children in Finland, but the fact that trauma-related disorders were over-represented among juvenile offenders is trending the same direction. All trauma disorders included in this study are thought to arise as a direct consequence of acute severe stress or continued trauma, and the disorder would not have occurred without their impact. It is notable that our study concerned 15-year-
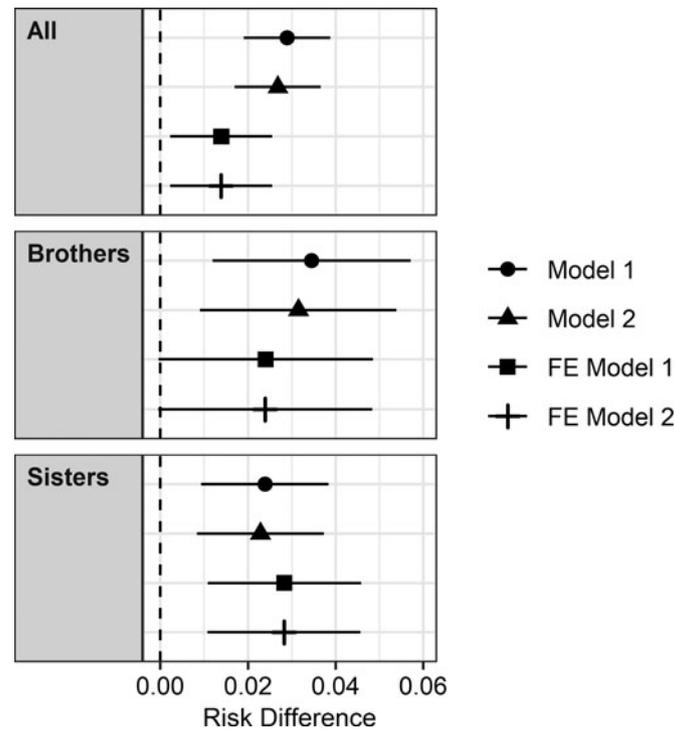
Figure 2 The risk differences and 95% CIs of violent criminality by trauma diagnosis across all sibling fixed effect models.

old to 17-year-old suspected offenders while Goff *et al*'s[9] study was conducted among adult prisoners. From youths' perspective, it is alarming that they showed a same trend.

This study had some limitations. First, the source of trauma diagnosis was not known. This means that there could be a great variety among adolescents whose trauma diagnosis is based on developmental trauma such as child abuse or neglect, compared to those who have a history of severe accidents, for example. When studying recidivism in a large sample of juvenile offenders (n=2792), Wylie and Rufino[22] found that adolescents with official records of certain kind of victimisation experiences such as abuse/neglect and personal crime victimisation were more likely to recidivate sooner than those without these victimisation experiences. Also, when examining the risk of self-harm and violent criminality among adolescents and young adults following injuries or poisonings during their childhood, Webb *et al*[23] found that the highest absolute risk was observed for violent offending among men admitted to hospital for interpersonal violence and for self-harm among women admitted to hospital following self-harm. The nature of trauma in relation to violent crimes is something that needs to be further studied.

Second, register data is unique in that it is official administrative data, which means there must be an authority contact. Our measure of criminal behaviour was based on suspicions of criminal behaviour recorded by the police. It is possible that the risk of getting caught for a violent crime varies between population groups, which may bias the results. However, other measures of delinquency (eg, self-reports) are not unbiased either, and existing studies suggest moderate agreement between different crime measures.[24 25] It thus seems unlikely that the results would only reflect differences in the risk of getting caught. It has been argued that the Nordic registries reach persons 'in need' broadly and that administrative register-linkage population data is better in

reaching marginalised individuals. They also enable retrospective designs without non-response or recall bias involved in survey or clinical sample settings.[26 27]

Third, although sibling models control indirectly for the factors shared by siblings and provide more robust estimates for the factors not shared by siblings, these models do not account for unmeasured factors not shared by siblings, such as peer groups and unique experiences.

Overall, the current study underlines the need for early identification and treatment of trauma-related symptoms. Adolescence is a time of increased risk of developing mental disorders including trauma-related disorders.[28] Early trauma treatment is crucial, not only in terms of decreasing the human suffering caused by it, but also to prevent criminal behaviour and other adverse social outcomes in later life. We need more population-based studies among under-aged young people in order to detect the effect of trauma on violent actions and to prevent the harmful cycles possibly caused by it.

What is already known on this subject

► Earlier studies confirm that there is an association between trauma and adolescent-onset offending. There is also some evidence of gene–environment interaction effects in altering the developing brain in ways that modify the risk for violence. The earlier studies have been mostly based on data collected among juvenile court clients, prisoners or clients in different kinds of youth facilities, where the youths have been placed because of own behavioural problems or parental difficulties.

What this study adds

► Using data on unselected youth, we were able to assess the effects of multiple risk -factors of trauma and violence simultaneously and while taking into account confounding factors. We show that trauma-related disorders were strongly associated with violent offending even after controlling for demographic factors, other mental health diagnosis and substance use disorders and finally the family factors shared by siblings. The current study underlines the need for early identification and treatment of trauma-related symptoms, not only in order to decrease the human suffering caused by it, but also to prevent criminal behaviour and other adverse social outcomes in later life.

ORCID iDs
Kirsi Peltonen http://orcid.org/0000-0002-5112-3823
Joonas Pitkänen http://orcid.org/0000-0002-8413-6399
Pekka Martikainen http://orcid.org/0000-0001-9374-1438

REFERENCES

1 Widom CS. Child abuse, neglect, and adult behavior: research design and findings on criminality, violence, and child abuse. *Am J Orthopsychiatry* 1989;59:355–67.
2 Ardino V. Offending behaviour: the role of trauma and PTSD. *Eur J Psychotraumatol* 2012;3:18968.
3 Foy DW, Ritchie IK, Conway AH. Trauma exposure, posttraumatic stress, and comorbidities in female adolescent offenders: findings and implications from recent studies. *Eur J Psychotraumatol* 2012;3:17247.
4 Ariga M, Uehara T, Takeuchi K, *et al.* Trauma exposure and posttraumatic stress disorder in delinquent female adolescents. *J Child Psychol Psychiatry* 2008;49:79–87.
5 Caspi A, McClay J, Moffitt TE, *et al.* Role of genotype in the cycle of violence in maltreated children.1072290. *Science* 2002;297:851–4.
6 Byrd AL, Manuck SB. MAOA, childhood maltreatment, and antisocial behavior: meta-analysis of a gene-environment interaction. *Biol Psychiatry* 2014;75:9–17.
7 Williams WH, Chitsabesan P, Fazel S, *et al.* Traumatic brain injury: a potential cause of violent crime? *The Lancet Psychiatry* 2018;5:836–44.
8 Dodge KA. Mechanisms of gene-environment interaction effects in the development of conduct disorder. *Perspect Psychol Sci* 2009;4:408–14.
9 Goff A, Rose E, Rose S, *et al.* Does PTSD occur in sentenced prison populations? A systematic literature review. *Criminal Behav Mental Health* 2007;17:152–62.
10 Vermeiren R. Psychopathology and delinquency in adolescents: a descriptive and developmental perspective. *Clin Psychol Rev* 2003;23:277–318.
11 Abram KM, Teplin LA, King DC, *et al.* PTSD, trauma, and comorbid psychiatric disorders in detained youth. *Juvenile Justice Bull* 2013; Washington, DC: U.S. Department of Justice, Office of Juvenile Justice and Delinquency Prevention. http://www.ojjdp.gov/pubs/239603.pdf (accessed 1 Feb 2019)
12 Kubiak SP. The effects of PTSD on treatment adherence, drug relapse, and criminal recidivism in a sample of incarcerated men and women. *Res Soc Work Pract* 2004;14:424–33.
13 Ouimette PC, Finney JW, Moos RH. Two-year posttreatment functioning and coping of substance abuse patients with posttraumatic stress disorder. *Psychol Addict Behav* 1999;13:105–14.
14 Assink ME, van der Put C, Hoeve M, *et al.* Risk factors for persistent delinquent behavior among juveniles: a meta-analytic review. *Clin Psychol Rev* 2015;42:47–61.
15 DeLisi M, Fox BH, Fully M, *et al.* The effects of temperament, psychopathy, and childhood trauma among delinquent youth: a test of DeLisi and Vaughn's temperament-based theory of crime. *Int J Law Psychiatry* 2018;57:53–60.
16 Dierkhising CB, Ko SJ, Woods-Jaege B, *et al.* Trauma histories among justice-involved youth: findings from the national child traumatic stress network. *Eur J Psychotraumatol* 2013;4:20274.
17 Hoeve M, Colins OF, Mulder EA, *et al.* Trauma and mental health problems in adolescent males: differences between childhood-onset and adolescent-onset offenders. *Crim Justice Behav* 2015;42:685–702.
18 Marsiglio MC, Chronister KM, Gibson B, *et al.* Examining the link between traumatic events and delinquency among juvenile delinquent girls: a longitudinal study. *J Child Adolesc Trauma* 2014;7:217–25.
19 Sariaslan A, Lichtenstein P, Larsson H, *et al.* Triggers for violent criminality in patients with psychotic disorders. *JAMA Psychiatry* 2016;7:796–803.
20 World Health Organization. *ICD-10: international statistical classification of diseases and related health problems: tenth revision*, 2nd Edition. 2004. https://apps.who.int/iris/handle/10665/42980 (accessed 1 Feb 2019).
21 Breen R, Karlson KB, Holm A. Interpreting and understanding logits, probits, and other nonlinear probability models. *Annu Rev Sociol* 2018;44:39–54.
22 Wylie LE, Rufino KA. The impact of victimization and mental health symptoms on recidivism for early system-involved juvenile offenders. *Law Hum Behav* 2018;42:558–69.
23 Webb RT, Antonsen S, Carr MJ, *et al.* Self-harm and violent criminality among young people who experienced trauma-related hospital admission during childhood: a Danish national cohort study. *Lancet Public Health* 2017;2:E314–E322-.
24 Gilman AB, Hill KG, Kim BK, *et al.* Understanding the relationship between self-reported offending and official criminal charges across early adulthood. *Criminal Behav Mental Health: CBMH* 2014;24:229–40.
25 Piquero AR, Schubert CA, Brame R. Comparing official and self-report records of offending across gender and race/ethnicity in a longitudinal study of serious youthful offenders. *J Res Crime Delinquency* 2014;5:526–56.
26 Andersen LH. Danish register data: flexible administrative data and their relevance for studies of intergenerational transmission. In: Eichelsheim VI, van de Weijer SGA, eds. *Intergenerational continuity of criminal and antisocial behaviour: an international overview of studies.* Cambridge, UK: Routledge, 2018.
27 Skardhamar T, Skirbekk V. Relative mortality among criminals in Norway and the relation to drug and alcohol related offenses. *PLoS One* 2013;8:e78893.
28 Casey BJ, Jones RM, Hare TA. The adolescent brain. *Ann N Y Acad Sci* 2018;1124:111–26.