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**The Importance of Familial Liability for Substance Abuse and
Criminality in Young Male Violent Offenders**

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Abstract. Background: There is an interest to find out more about family background of violent offenders and how it affects the development of their problems. Aims: To investigate the association between familial liability, substance abuse disorders (SUDs) and aggressive antisocial behaviour in young violent offenders. Method: Pedigree of first, second and third degree relationship were used to measure familial liability of young, male violent offenders (n = 221) in prison. Clusters were formed based on familial liability to examine differences in amount of SUDs and antisocial aggressive behaviour. Results: Violent offenders could be differentiated into three clusters by familial liability. Familial liability had an effect on age of debut on violent crime and SUDs, life-time rates on aggression and severity of substance abuse. Conclusions: Having familial liability gives earlier age of onset and a more severe outcome regarding both aggressive antisocial behaviour and substance abuse. Some differences can also be seen between different types of familial liability.

Violent crimes are a major problem in society, of both personal consequences and social costs and the World Health Organization has recognized violent crimes as a substantial public health problem (Krug, Dahlberg, Mercy, Zwi & Lozano, 2002). Violence is naturally linked to aggression and aggressive behaviour. According to Citrome and Volavka (2003) aggression is generally considered to be multi-determined. Predisposing factors for aggression include genetic factors, the fetal environment, obstetric complications, upbringing conditions, biologic factors, and psychiatric disorders like SUDs, psychosis, depression, and personality disorders. The relationships between violence and SUDs are well known, SUDs is clearly stated to convey an increased risk for violence and violent crime (Arseneault, Moffitt, Caspi, Taylor & Silva, 2000; Pulay et al., 2008). Both criminality and SUDs has been linked to adverse experiences in childhood and can be explained by biological, sociological and psychological theories. An earlier report on the prevalence of psychiatric problems in young violent offenders showed that they had many factors of vulnerability during childhood but also present mental illness of developmental disabilities, depression, psychosis or posttraumatic stress disorder (Billstedt & Hofvander, 2013). The majority had one or more adverse factors while growing up, such as witness and experience violence, parental alcohol or drug abuse or to be separated from family of origin. Most of the young adults had been convicted before and 84 % had substance use disorders. Although violence is a noted health problem, we still lack important knowledge about the family background of violent offenders and its significance for the development of their own problems. Therefore, there is a need to further investigate risk factors in families of violent offenders and the interactions thereof.

In Sweden violent crimes constitutes over 20 % of all convictions, and even more among young offenders where they represent over 30 % of all convictions (Kriminalvården, 2014). Nine out of ten violent crimes are committed by men (Krug et

al., 2002; Moffit & Caspi, 2001; Olseryd, 2015). Individuals aged 15-17 years are most often convicted for violent crimes, followed by the age group 18-20 years (Westfelt, 2016). In addition the majority of violent crimes are committed by a small number of offenders. A national population-based study showed that 1 % of the Swedish population accounted for the majority (63 %) of all violent convictions, with a cut-off at three or more convictions (Falk, Wallinius, Lundström, Frisell, Anckarsäter & Kerekes, 2014). Violent crimes are often committed under the influence of alcohol or drugs (Olseryd, 2015). Results from a study on inmates with SUD showed that they had more convictions since minor age, and cumulating numerous convictions. They were also reported to have more violent attitudes and behaviours compared to all inmates and confirmed increased levels of lifetime aggression irrespective of sentence classified as violent or not (Cuomo, Sarchiapone, Di Giannantonio, Mancini & Roy, 2008). The strongest risk factors for persistence in violent crime has been reported as male sex, personality disorders and a first conviction for violence before age 18, followed by SUDs (Falk et al., 2014).

Research has been made of criminals and family background. Incarcerated persons, and violent offenders, have a higher rate than others of childhood trauma and a family history of mental illness, conviction or SUD (Kriminalvården, 2014). Previous research of criminals and aggressive, antisocial behaviour and their family background has mainly been focusing on parents showing linkage of parental liability and devolving own problems (Huesmann, Eron, Lefkowitz & Walder, 1984; Osborn & West, 1979; Thornberry, Freeman-Gallant, Lizotte, Krohn, Smith, 2003; Van de Rakt, 2008). One study has been made with an intergenerational assessment on five generations of parents and children using conviction data. This study does not conclude evidence for hereditary factors and raises the question of nature or nurture as explanation (Bijleveld & Wijkman, 2009). Another three generations study found intergenerational transmission, but it decreased after controlling for other factors. The only strong linkage even after controlling for other factors was between father and son (Farrington, Coind & Murray, 2009). On the other hand there are several other studies that have shown hereditary factors as an explanation of criminality and antisocial behaviour (Frisell, Pawitan, Långström, Lichtenstein, 2012; Mednick, Gabrielli & Hutchings, 1984). In another study antisocial behaviour was accounted to have 50 % genetic influence and 20 % environmental influence of factors shared by family members (Moffitt, 2005). Offenders seem to be highly concentrated in families. In a study of inter-relationships and offending in three generations the likelihood was high that if one relative had been arrested another relative had also been arrested. Arrests of all persons in the family predicted a boy's delinquency and the most significantly predicting relative was the father (Farrington, Jolliffee, Loeber, Stouthamer-Loeber & Kalb, 2001).

There are also studies that suggest and explain hereditary mechanism not only among criminals but specifically among violent offenders (Frisell, Lichtenstein & Långström, 2011; Frisell & Långström, 2014). Result has even shown a stronger intergenerational transmission of violent offending than for non-violent offending (Van der Weijer, Bijleveld & Blokland, 2014). As named earlier, risk factors for persistence in violent crime has been identified. Adding to these, also parental factors such as having a parent who have been convicted of crime or diagnosed with psychiatric disorders or SUDs were linked to persistence in violent crime. Risk factors differed between high- and low-persistence violent offenders where high-persistence offenders had a distinctly higher frequency of risk factors. The rates of parental risks for non-

violent offenders were considerably lower (Falk et al., 2014). In a nationwide study based on all violent crimes-convictions in Sweden 1973-2004 a strong familial aggregation of interpersonal violence was found among first-degree relatives, close genetic relatives had high familial risk of violent behaviour leading to criminal convictions. For more distant relatives the aggregation was lower but still significant. Younger age at first conviction was associated with higher violence risk in siblings. The study also provided evidence of both genetic and environmental influences on development of violent behaviour (Frisell, Lichtenstein & Långström, 2011). Several studies have also shown relationship between violence and SUDs within families. In an Australian study of risk factors for death among young offenders, age 18-24, it was reported that 54 % had a family member with drug or alcohol problems, 27 % had a family member with mental illness and 52 % had a family member in prison. Of these 70 % had an offense history of violent crime (Kinner, Degenhardt, Coffey, Hearps, Spittal, Sawyer & Patton, 2015). When the association of paternal incarcerations and drug use in young adults was investigated in the United States, the study showed that having a father that has ever been incarcerated was associated with higher levels of substance use. In addition, young adults with a history of fathers being incarcerated were also more likely to have a mother with a history of binge drinking and were themselves more often arrested as juveniles (Roettger, Swisher, Kuhl & Chavez, 2010). A report on young Swedish persons who during childhood had a parent that were hospitalized for SUD or mental illness showed that in the group of persons with parental SUD or mental illness the experience of social care and criminality was higher and they were identified as a high-risk group with high levels of mortality, mental illness, SUDs, financial support and low employment. Among the parents fathers had equally both mental illness and SUD while mental illness where more frequent amongst mothers. Although, for those individuals who had mothers with SUD they had extremely much more experience of social care and higher risk of SUD. Male individuals with parents of SUD had been convicted in 30 %, and male individuals with parents with mental illness had been convicted 18-19 %, which was a higher level than the entire population (Hjern, Arat & Vinnerljung, 2014).

To prevent violent crime and to accomplish better interventions for violent offenders and their families there is still a need to find out more about the family histories of violent offenders. Since violent crime is the main crime committed by young offenders it is of particular interest. It is well known that antisocial behaviour runs in the family and that risk of SUD increases with family history. Farrington (2002) has described mechanisms that explain concentration and transmission of criminality in generations with a biosocial perspective. While previous research on family background has mainly focused on first-degree relationships, mainly parents and particularly fathers, this study expanded family to second and third degree relationships. In an article that reviewed research of behavioural-genetic studies on antisocial outcomes (Moffitts', 2005) the author suggests that relevant research benefits from a bio-social model where genetic and environmental risks coincide. Although there is no clear separation of genetic and social risk factors in this study we have made a design based on the assumption that mental disorders, SUDs and criminality all are risk factors that have a strong genetic linkage. The family liability is therefore focusing on heredity with only biological relatives in the pedigree data. Siblings, half-siblings, parents, aunts, uncles, grandparents and cousins were included. Family liability is focusing on four risk factors; criminality, SUDs, neurodevelopmental disorders and major mental disorders.

The general aim of this study was to use pedigree data to investigate the association of familial liability, SUDs and aggressive antisocial behaviour in young violent offenders. The specific aims were to;

- 1) explore whether groups of young violent offenders can be distinguished based on variables of familial liability.
- 2) analyze differences between groups focusing on lifetime prevalence of aggressive antisocial behaviour and SUD.

Method

Sample

Participants were derived from the Development of Aggressive Antisocial Behavior Study (DAABS), a study on the prevalence of early-onset behaviour disorders and mental health problems in young adult male offenders (Billstedt & Hofvander, 2013). Participants (N=270) were male offenders age 18 to 25 years, convicted for violent crimes (hands-on sexual crimes included), imprisoned in nine different prisons in the western region of the Swedish Prison and Probation Service. All participants were included between February 2010 and August 2012. Exclusion criteria were poor knowledge of Swedish or very short stay at the prison (<2 weeks). Attrition was 29 %. For this present study only participants with adequate pedigrees were included (N=221). Among them the mean age was 22 years and the mean prison sentence in months was 23. Of the cohort 87 % had been convicted prior to the ongoing sentence with a mean of 4 convictions. Most of the offenders, 75 %, were of Swedish origin, 11 % had origin of Europe, 6 % had origin of Africa, 1 % had origin of Latin America and 7 % had origin of Middle East. Origin was determined by their country of birth.

Table 1

Background of probands, N=221

	n	%
Education		
Graduated elementary school or lower	166	75.1
Graduated high school	53	23.9
Graduated college/university	1	0.5
Childhood conditions		
Positioned in family or institution*	103	46.6
LVU **	67	30.3
Mother deceased	5	2.3
Father deceased	19	8.6
Criminal record		
One or more previous convictions	192	8.9
LSU ***	25	11.5

Table 1 continuing on next page

Continuation of Table 1

	n	%
Pedigree data		
Male relatives	1300	51.7
Female relatives	1213	48.3
1 st degree relatives	885	35.2
2 nd degree relatives	1579	62.8
3 rd degree relatives	49	1.9

*Positioned before age 18.

** The Care of Young Persons (Special Provisions) Act, for age under 18.

*** Closed youth detention, juvenile sanction for age 15-17.

Measures

Information on the offenders was collected in structured protocols from file information, clinical investigations, self-rating instruments, semi-structured interviews, and parent interviews. For this study information on geographical origin, psychosocial adversities and maladjustments during upbringing, placement in institution, history of SUD, history of criminal behaviour and pedigree was gathered. DSM-IV-diagnoses of the proband were set according to the LEAD-principle (Spitzer, 1983).

Pedigree data is a family health history consisting of individuals with consanguinity covering data of diagnoses, health information and cause of death (Bennett, 1999). This pedigree information primarily relies on the prisoners' self-reports, combined with information from the other sources mentioned above. Every family member was reported dichotomously on alcohol abuse/dependence, substance abuse/dependence, criminality, autism spectrum disorder, bipolar disorder, chronic psychiatric disorder, other psychiatric disorder, abnormal personality traits, remedial education, mental retardation, dyslexia, neurological disease, miscarriage, spontaneous abortion, other somatic disease. Age of death and cause of death were also reported. This information was used to analyse the effects of genetic and environmental familial liability.

Life History of Aggression (LHA) and information of criminal records were used to measure aggressive antisocial behaviour. LHA is an 11-item scale developed as a self-report instrument (Brown, Elbert, Goyer, Jimerson, Klein, Bunney & Goodwin, 1982). The LHA measures aggression in a lifelong perspective where every item is rated from 0 ("no event") to 5 ("so many events that they cannot be counted"), with a range of the total score from 0 to 55. High score is reflecting persistent aggressive behaviour. The LHA has three subscales; aggression (five items on temper tantrums, physical fights, verbal aggression, physical assaults on people or animals, and assaults on property), antisocial behaviour (four items on school disciplinary problems, problems with supervisors at work, antisocial behaviour not involving the police, and antisocial behaviour involving the police) and self-directed aggression (two items on self-injurious and suicide attempts). All items are rated based on numbers of occurrences the behaviour since teenage. In this study the LHA self-report was assessed with help from clinical interview. It has been shown that the psychometric properties of LHA are

satisfying with good results on test of stability and reliability (Coccaro, Berman & Kavoussi, 1997).

Diagnoses of Substance Use Disorders (SUD) were set by senior clinical psychologists according to the Diagnostic and Statistical Manual of Mental Disorders 4th edition (DSM-IV; American Psychiatric Association, 1994). The variable “extremely destructive substance use” is defined by drug consumption pattern that consists of no special drug preference, and a severely impulsive and destructive use with loss of control and frequent over doses.

Statistical methods

Sample characteristics were given by descriptive statistics and frequency distributions. Since distributions were skewed, all statistical analyses were non-parametric.

A measure of an individual's familial risk has earlier been designed by Richard Kerber (1995) to calculate risk of disease from familial factors. This method accounts for all known biological relatives and their degree of relatedness to the proband. The original formula, FR, was the foundation for the formula used in this study. The formula developed from Kerber's FR-measure does not include information of time at risk for each member in the cohort, the formula used in this study was called Familial Liability Value, FLV. The FLV has been used as a method to obtain a continuous measure of the familial gene-environmental factors of interest for every offender. We report on four variables, considered risk factors, from family history: SUDs (alcohol and/or drugs), criminality (conviction to imprisonment or repeated convictions), major mental disorders (bipolar disorder and/or chronic psychiatric disorder) and neurodevelopmental disorder (autism spectrum disorder and/or remedial education, mental retardation, dyslexia). Every biological relative of an offender was given a weight based on degree of kinship. First-degree relationships included siblings and parents were given weight of 1. Second-degree relationships included half siblings, grandparents, aunts and uncles and were given a weight of 0.5. Third-degree relationships included cousins and they were given a weight of 0.25. All relatives were then reported positive (1) or negative (0) on every risk factor. By giving weights related to degree of kinship and adjust for number of family members the differences in offender's family structure was taken to account. All risk factors were calculated separately from each other giving a value of every offender's familial liability of SUDs, criminality, major mental disorders and neurodevelopmental disorders.

$$FLV = \frac{\sum_{j=1}^N C_j f(i, j)}{\sum_{j=1}^N f(i, j)}$$

The formula for Familial Liability Value where $f(i, j)$ is the weighted kinship between offender (i) and family member (j) and C_j is 1 if family member had positive history of risk factor and 0 if family member lacked history of risk factor.

Scores of familial liability were converted to standardized Z scores. To analyze the familial liability and association of offenders' violence and SUD hierarchical cluster analysis was performed with the four familial risk factors: SUDs, criminality, major mental disorders and neurodevelopmental disorder. Ward's method was used to identify the most relevant number of clusters, and measures of similarity between cases were calculated through Squared Euclidian distances. To check for correlation between the four liability variables tests of collinearity were made with a VIF value of between 1 and 1.291, given that no serious violation was identified. Mann-Whitney U tests were used to determine group differences between clusters for each risk factor, this was made with raw scores.

To explore group differences in level of familial liability Kruskal-Wallis H tests were conducted for ranked data. Distributions of CWWS scores were similar assessed by visual inspection of a boxplot. If median scores were statistically significantly different between groups we continued with post hoc analysis to make pairwise comparisons using Dunn's, 1964, procedure with a Bonferroni correction for multiple comparisons. If distributions of median scores were not similar between groups we checked mean ranks (Laerd Statistics, 2015). In table 3 and 4 *p*-value is presented, the post hoc analysis included adjusted *p*-value.

For dichotomous data we performed analysis with chi-square test of homogeneity and the post hoc analysis involved pairwise comparisons using the z-test of two proportions with a Bonferroni correction (Laerd Statistics, 2016). Eta squared was used to measure effect size for comparison between groups on continuous variables. Results were considered statistically significant at the $p < .05$ levels, reported *p*-values were adjusted for pairwise comparisons. Results are given as median and range.

The number of probands in the clusters varies on different reported variables, which is noted in the result tables. It can be a missing value if the data could not be verified and therefor considered as invalid data. The number of probands can also be reduced if the variable didn't apply to the proband, for example if a proband had no history of drinking alcohol the variable of "age of alcohol debut" gave no information.

All statistics were calculated, using anonymized data, with SPSS 21 and 22.

Ethical considerations

The study was approved by the Research Ethics Committees at Lund University (register # 2009/405). All inmates received oral and written information about the study from a prison staff. All inmates that agreed to participate in the study provided written informed consent. All participants were also given the opportunity to receive feedback on the preliminary results from the assessments. Participants showing indications of severe psychopathology were given the opportunity to be referred to the prison doctor (a psychiatrist) for further assessment and treatment. A small monetary compensation for time spent in the study was provided (SEK 200, approximately \$25).

Results

Familial liability groups

All persons in the pedigree data were given a weight on the different risk factors according to their degree of relationship, by that all offenders had a calculated liability. Based on familial liability of criminality, SUD, neurodevelopmental disorders and major mental disorders three clusters were formed where the offenders were similar in high or low liability. Cluster one consisted of 144 individuals (65 %) with no or very low liability on all variables, the cluster was labelled “no liability” (NL). Cluster two consisted of 49 probands (22 %) and was formed by liability in all areas except major mental disorders, labelled “liability on Criminality, SUDs and Neurodevelopmental Disorders” (C/SUD/ND). The third cluster consisted of 28 probands (13 %) who were similar in liability on two variables labelled “liability on SUDs and Major Mental Disorders (SUD/MMD). As seen in Figure 1 both cluster two and three had familial liability of SUDs, although cluster three had higher scores on the variable.

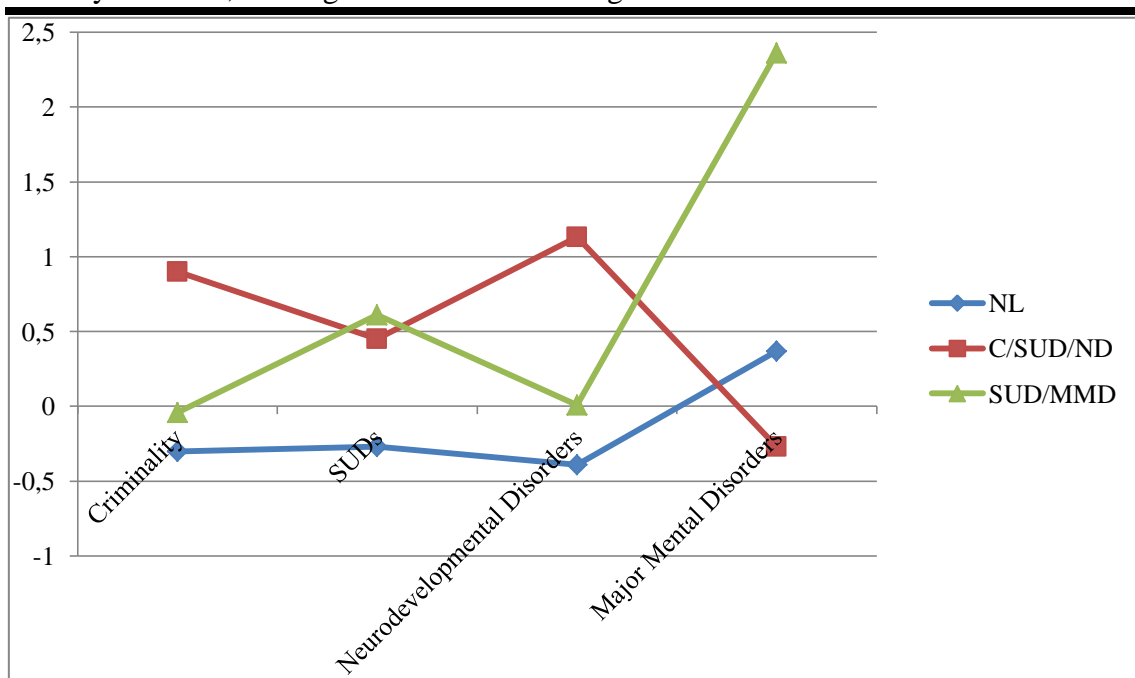


Figure 1. Mean of familial liability in groups. Z-scores are presented. Cluster one (NL) is blue and marked with rhombs, cluster two (C/SUD/ND) is red and marked with squares and cluster three (SUD/MMD) is green and marked with arrows.

Table 2

*Mann-Whitney U test comparing familial liability of risk factors**

	NL <i>Mdn</i>	C/SUD/ND <i>Mdn</i>	SUD/MMD <i>Mdn</i>
SUD	.00	.2571	.2619
C	.00	.1724	.00
ND	.00	.1250	.00
MMD	.00	.00	.1429

	NL- C/SUD/ND		NL-SUD/MMD		C/SUD/ND-SUD/MMD	
	U	<i>p</i>	U	<i>p</i>	U	<i>p</i>
SUD	2199.50	.001	1045.00	.001	615.00	.45
C	1659.50	.001	1712.00	.14	410.50	.01
ND	1261.50	.001	1577.50	.01	368.00	.001
MMD	3240.00	.001	.001	.001	12.00	.001

*Risk factors presented: SUD = Substance Abuse Disorders, C = Criminality, ND = Neurodevelopmental Disorders and MMD = Major Mental Disorders. All *p*-values are Asymp. Sig. (2-tailed) and presented significant as < .05, <.01 or <.001. If not significant exact *p*-value is presented.

Mann-Whitney U tests were run to determine if there were differences in familial liability of risk factors between the three clusters. Familial liability was not statistically significantly different between NL (*Mdn* = .00) and SUD/MMD (*Mdn* = .00) on criminality, $U = 1712.00$, $p = .14$, or between C/SUD/ND (*Mdn* = .26) and SUD/MMD (*Mdn* = .26) on substance abuse, $U = 615.00$, $p = .45$. The two comparisons that were not statistically significantly different were coherent with the shaping of clusters where different clusters were similar in one factor but not in others. Although medians were equal, or close to equal, SUD/MMD had higher mean ranks than NL on criminality and SUD/MMD had higher mean ranks than C/SUD/ND on SUDs. For all other comparisons C/SUD/ND had significantly higher familial liability on all risk factors compared to NL, SUD/MMD had significantly higher familial liability compared to NL on all factors except criminality, C/SUD/ND scored significantly higher on criminality and neurodevelopmental disorders compared to SUD/MMD, and SUD/MMD scored significantly higher on major mental disorders compared to C/SUD/ND.

Group differences in aggressive antisocial behaviour

A series of Kruskal-Wallis H tests were conducted to determine if there were differences in aggressive antisocial behaviour between groups that differed in their level of familial liability. As seen in table 3 the group NL had lower LHA scores and a somewhat later age of onset of crimes. For LHA total NL had a median of 30, C/SUD/ND had 34 and SUD/MMD had the highest score of 36 ($p < .05$). The same ranking followed for LHA aggression where ND had 17, C/SUD/ND had 20 and SUD/MMD had a median of 21 ($p < .01$). The results of pairwise comparisons were

statistically significant in both cases between NL and C/SUD/ND (LHA total *adj. p* < .05 and LHA aggression *adj. p* < .01) Results for the subscale LHA antisocial behaviour were not significant and the subscale LHA self-directed aggression had a median of 0 in all groups.

Age at onset of any crime was not significant, in contrast to age at onset of violent crime that was statistically significant (*p* < .05). C/SUD/ND had the lowest age with 15 years and SUD/MMD with the highest age of onset with 18 years. Between these two groups the pairwise comparisons were significant (*adj. p* < .05).

Table 3

*Group differences in aggressive antisocial behaviour**

	NL n=108-143 Mdn (r)	C/SUD/ND n=44-49 Mdn (r)	SUD/MMD n=19-28 Mdn (r)	<i>p</i>	df	χ^2
LHA total	30 (43)	34 (45)	36 (35)	.05	2	8.26
LHA aggression	17 (24)	20 (21)	21 (19)	.01	2	11.75
LHA antisocial behaviour	13 (19)	14 (19)	13.5 (20)	.39	2	1.99
LHA self-directed aggression	0 (6)	0 (7)	0 (8)			
Onset of any crime	14 (18)	12 (19)	13 (15)	.07	2	5.37
Onset of violent crime	17 (16)	15 (19)	18 (8)	.05	2	8.03

* Median is presented with range (r), *p* is presented significant as < .05, <.01 or <.001. If not significant exact *p*-value is presented.

Group differences in substance abuse

For all variables the Kruskal Wallis-H tests showed that the group NL had higher age of onset of using alcohol or drugs, and the lowest score for proportion within group with specific substance abuse or severe substance abuse. Distribution for age of alcohol onset was not similar for all groups, as assessed by visual inspection of a boxplot. The mean ranks; NL 107.8, C/SUD/ND 92 and SUD/MMD 81.8, were significantly different between groups (*p* < .05). There was a significant difference between the groups for any SUD; 73 % of the probands in group NL had a SUD, 85 % of the probands in C/SUD/ND and 93 % of the probands in group SUD/MMD, although pairwise comparisons were not statistically significant. Looking at the median of total amount of SUDs NL had 3 SUDs, C/SUD/ND had 5 SUDs and SUD/MMD had 6 SUDs (*p* < .01). Pairwise comparisons were statistically significant between NL and C/SUD/ND (*adj. p* < .05).

For the specific types of drugs the chi-square test of homogeneity gave no significant results for alcohol, cannabis or opioid-analgesics. For all the other drugs the results were statistically significant that NL had lower proportion of probands with SUD compared to C/SUD/ND and SUD/MMD. For stimulants NL had 52 % while

C/SUD/ND had 73 % and SUD/MMD had 75 % ($p < .01$). The same ranking followed for heroin; NL 29 %, C/SUD/ND 38 % and SUD/MMD 54 % ($p < .05$). For hallucinogens NL had 28 %, SUD/MMD had 43 % and C/SUD/ND had 54 % ($p < .01$). The same ranking followed for sedative-hypnotic-anxiolytic; NL had 43 % SUD/MMD had 61 % and C/SUD/ND had 63 % ($p < .05$) and other substances; NL 32 %, SUD/MMD 50 % and C/SUD/ND 64 % ($p < .001$).

Table 4

*Group differences in substance abuse**

	NL n=113-143 Mdn (r)	C/SUD/ND n=46-49 Mdn (r)	SUD/MMD n=25-28 Mdn (r)	p	df	χ^2
Onset of alcohol	14 (13)	13.5 (13)	13 (7)	.05	2	6.05
Onset of drug	15 (12)	14 (14)	14 (6)	.46	2	1.55
Total SUDs	3 (11)	5 (10)	6 (10)	.01	2	10.96

	NL n=142-143 % (n)	C/SUD/ND n=47-49 % (n)	SUD/MMD n=28 % (n)	p	df
Any SUD	73 (104)	89 (42)	93 (26)	.01	2
Extremely destructive substance abuse	18 (26)	27 (13)	26 (7)	.36	2
Alcohol SUD	46 (65)	55 (27)	57 (16)	.33	2
Cannabis SUD	72 (103)	83 (40)	89 (25)	.07	2
Stimulants SUD	52 (74)	73 (35)	75 (21)	.01	2
Hallucinogens SUD	28 (39)	54 (26)	43 (12)	.01	2
Sedative-hypnotic -anxiolytic SUD	43 (61)	63 (30)	61 (17)	.05	2
Heroin SUD	29 (42)	38 (18)	54 (16)	.05	2
Opiod-analgesics SUD	39 (56)	45 (22)	61 (17)	.10	2
Other substances SUD**	32 (45)	64 (30)	50 (14)	.001	2

* Median is presented with range (r), p is presented significant as $< .05$, $< .01$ or $< .001$. If not significant exact p -value is presented.

** Steroids, volatiles, GHB/GBL.

Discussion

The aim of this study was to investigate the association of familial liability, SUDs and aggressive antisocial behaviour in young violent offenders. In the group of young violent offenders around half of them had been placed in a foster home or an institution during upbringing. The group was also very low educated. The young violent

offenders can be compared with two other groups in a survey on clients in the Swedish Prison and Probation Service (Kriminalvården, 2014); all prison inmates, or youths aged 18-21 years in prison or under probation. The level of education was lower among the violent offenders compared to all prison inmates but equal to youths in prison or under probation. The young violent offenders also had a higher rate of being positioned in institution during upbringing compared to prison inmates or youths in prison or under probation. It is known that the age groups 15-17 years, followed by 18-20 years, are most frequently convicted of violent crimes (Westfelt, 2016). This is confirmed by the young violent offenders in this study that had an extremely much higher rate of being convicted prior to the on-going sentence, and a notably higher rate of convictions according to closed youth detention, juvenile sanction for age 15-17 (LSU), compared to the other groups of inmates or youth (Kriminalvården, 2014). To sum up, this indicates that the group of violent offenders as a whole has poor conditions from young age and stands out as more weighed down than other offenders.

We know from earlier studies that violent offenders have more family related risk factors compared to other offenders. The interest has been to whether violent offenders can be differentiated based on their familial liability of criminality, SUDs, neurodevelopmental disorders and major mental disorders. To examine the familial liability we used cluster analysis that captured the natural structure of the data and discovered three clusters. The biggest group had no or very low familial liability (NL). The rest were separated in two groups, second largest were the group with liability of criminality, SUDs and neurodevelopmental disorders (C/SUD/ND). The smallest group had liability of substance abuse and major mental disorders (SUD/MMD). This showed that groups can be distinguished by familial liability, not only overall low from high, but also by different types of liability.

The three clusters were used to investigate aggressive antisocial behaviour and SUDs, and to see whether familial liability was associated with differences. The three clusters all had high ratings on the LHA total and the subscales aggression and antisocial behaviour. Scores on LHA total of 15 and LHA aggression of 12 is suggested as norm of when life history of aggression is abnormally high (Coccaro et al., 1997), scores which all three groups greatly exceeded. The young violent offenders had also higher scores than groups in other studies with offenders, patients with neurodevelopmental disorders or personality disorders (Coccaro et al., 1997; Coccaro, Beresford, Minar, Kaskow & Geraciotti, 2007; Hofvander et al., 2011). The self-directed aggression seemed to be non-existing for all clusters, this might be because subjects underestimate this type of aggressive behaviour. Coccaro et al. (1997) has also noted that the subscale of self-directed aggression had the poorest internal consistency and that it should be a separate consideration. Growing up with no or low familial liability was characterized by lower scores on LHA total and LHA aggression, the group had a later onset of violent crimes compared to the group with the earliest onset. The group C/SUD/ND stands out on early age of onset of violent crimes. Since this is the group with familial liability of criminality it can be interpreted in accordance with earlier reports that showed concentration of offenders in families and that violent crimes runs in families (Farrington et al., 2001; Frisell et al., 2011; Kinner et al., 2015; Kriminalvården, 2014). Familial liability of criminality might not only predict the occurrence of criminality and convictions, but also that the debut is set earlier. It is also the only group with liability of neurodevelopmental disorders, where AD/HD is included. AD/HD has been associated with higher risk of aggressive and antisocial

behaviour (Bernat, Oakes, Pettingell & Resnick, 2012; Connor, Chartier, Preen, & Kaplan, 2010; Hamshire et al., 2013; Kakouros, Maniadaki & Karaba, 2005). The SUD/MMD group scored highest on LHA total but at same time they surprisingly had the latest onset of violent crimes. A reason for that could be explained by the fact that children who grow up with mentally ill parents is commonly taking responsibility at home and might even become the caretaker (Leahy, 2014; Trondsen, 2012). In this study there were no data on offenders caretaking or responsibilities at home that might explain later onset of violent crimes, and along with a late onset of violent crime the age of debut of any crime is not comparable high although the result for debut of any crime were not significant. A Swedish report on persons in young adulthood, who grew up with parents with SUD or mental illness, showed that men with parents with SUD had a four to five times higher risk of conviction for serious crimes, and for men with parents with mental illness the risk was two to three times higher, compared to those with parents without SUD or mental illness (Hjern et al., 2014). On the contrary the population based study on violent crime convictions (Falk et al., 2014) identifies parental risk factors for persistence in violent crimes where conviction among parents is most common, followed by psychiatric inpatient diagnoses and then SUDs.

Seventy percent of the clients in the Swedish Prison and Probation Service are estimated to have a SUD, regardless of prison inmates or youth (Kriminalvården, 2014). In this study that matches the level of SUD in the NL group. The two groups with liability had clearly higher percentage of SUD. The fact that the NL group had the least severe results on all substance abuse variables showed that familial liability does have an effect on developing own problems with addiction. Looking at specific substances the gap between groups of having liability or not is the least for alcohol and cannabis, this might reflect that consumption of alcohol is legal in Sweden. Cannabis is not legal but is listed as the most common used narcotic and that the accessibility is high (Centralförbundet för alkohol- och narkotikaupplysning, 2014), this can explain why it is spread more even among all groups.

Both liability groups showed a liability of SUDs although SUD/MMD scored a little higher, that is also the group with the earliest onset of alcohol use, the biggest proportion of SUDs and highest numbers of SUDs disorders per person. From this result we can see that family liability had an effect on SUDs, and that the severity of own developed SUD might increase with amount of familial liability of SUDs. One reason is the biological vulnerability, an overview of genetics and alcoholism sets the genetic factors accountable for more than 50 % of the variance in liability (Ducci & Goldman, 2008). However we cannot make a clear separation between the two groups with liability, the mentioned risk factors is not isolated as an influence alone but can increase occurrence of problems, be amplified by multi risk factors or diminish by co-existing protective factors.

Another interesting discovery is the variable “other substances” that consists of steroids, volatiles, and GHB/GBL. If looking at steroids separately it shows that this is the only substance that the NL group does not score lowest amount of SUDs (NL 13 %, C/SUD/ND 23 %, SUD/MMD 4 %, $p = .06$). Volatiles, are often referred to as drug of choice of young teenagers, and little is known about volatile SUD among adults. Surprisingly, in this study many had a SUD of volatile (NL 11 %, C/SUD/ND 28 %, SUD/MMD 36 %, $p = .01$), which greatly exceeds levels of use from annual reports from youth in Swedish schools and reports of institutional care of adults with drug abuse or addiction (Guttormsson & Leifman, 2016; Socialstyrelsen, 2016). GHB/GBL

were quite evenly spread among the three groups (NL 20 %, C/SUD/ND 22 %, SUD/MMD 18 %, $p = .87$).

Limitations

Self-reported data from the offenders can be questioned because of the risk for under-reporting (Moffit, Caspi, Taylor, Kokaua, Milne, Polanczyk & Poulton, 2010). However, the combination of semi-structured interviews by clinicians and file information might have reduced the risk of recall bias. The examined groups are quite small in sizes and there is no control group, therefore the results should be interpreted with caution. Since all of the subjects in the study were of interest because of conviction for violent crime the variance of violence were little according to LHA and onset of crimes. It should also be pointed out that no female violent offenders in prisons of the western region of Swedish Prison and Probation Services were included. However, the result should be considered meaningful in the clinical context of male young violent offenders. This study was only focused on the familial liability, for example no socioeconomic factors is taken into account, this is considered a limitation since violence and SUDs cannot be explained by single factors but a multi factorial perspective.

Conclusions

All three groups in this study was similar regarding that the offenders had at least one present conviction for violent crime and that they before that mainly had an onset of any crime, also having at least one SUD was common. One can assume that most of the offenders have been associated with others with criminality and alcohol or drug abuse during their teens. The identified clusters were meaningful when examining the effects of familial liability. Familial liability was showed to be associated with earlier age of debut regarding violent crimes and substance use, higher scores on LHA and more severe substance abuse. It is clear that the findings of familial liability goes along with previous research on parental risk factors linked to outcome of children's SUD, conviction and persistence in violent crimes. Also it seems like distinct types of liability can be associated with different effects. To prevent and avert that youth with familial liability develop their own serious problems, and as we know that they had earlier debut in violent crime and substance use, there is a need for alertness and act from society, long before this outcome. It should also be considered that having familial liability might be closely linked to a poorer support at home for offenders in treatment.

References

- American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders 4th edition*. Washington, DC: Author.
- Arseneault, L., Moffitt, T. E., Caspi, A., Taylor, P. J. & Silva, P. A. (2000). Mental disorders and violence in a total birth cohort: results from the Dunedin Study. *Archives of General Psychiatry* 57(10), 979–986.
- Bennett, R. L. (1999). *The Practical Guide to the Genetic Family History*. New York: Wiley-Liss.
- Bernat, D. H., Oakes, J. M., Pettingell, S. L. & Resnick, M. (2012). Risk and direct protective factors for youth violence results from the national longitudinal study of adolescent health. *American Journal of Preventive Medicine*, 2(43), 57–66. doi: 10.1016/j.amepre.2012.04.023
- Bijleveld, C. C. J. H. & Wijkman, M. (2009). Intergenerational continuity in convictions: A five-generations study. *Criminal Behaviour and Mental Health*, 19, 142-155. doi: 10.1002/cbm.714
- Billstedt, E. & Hofvander, B. (2013). *Tidigt debuterande beteendestörning: förekomst och betydelse bland vålds- och sexualbrottsdömda* (Projektnummer 2009-115). Norrköping: Kriminalvården.
- Brown, G. L., Ebert, M. H., Goyer, P. F., Jimerson, D. C., Klein, W. J., Bunney, W. E & Goodwin, F. K. (1982). Aggression, suicide, and serotonin: Relationships to CSF amine metabolites. *The American Journal of Psychiatry*, 139, 741–746.
- Centralförbundet för alkohol- och narkotikaupplysning (2014). *Drogutvecklingen i Sverige 2014* (Rapport nr 144). Stockholm: Centralförbundet för alkohol och narkotikaupplysning.
- Citrome, L. & Volavka, J. (2003). Treatment of violent behavior. In A. Tasman, J. Kay, & J. Lieberman (Eds.), *Psychiatry* (pp. 2136-2146). John Wiley & Sons, New York.
- Connor, D. F., Chartier, K. G., Preen, E. C. & Kaplan, R. F. (2010). Impulsive aggression in attention-deficit/hyperactivity disorder: symptom severity, co-morbidity and attention deficit/hyperactivity disorder subtype. *Journal of Child and Adolescent Psychopharmacology*, 2(2), 119-126. doi:10.1089/cap.2009.0076.
- Cuomo, C., Sarchiapone, M., Di Giannantonio, M., Mancini, M. & Roy, A. (2008). Aggression, impulsivity, personality traits and childhood trauma of prisoners with substance abuse and addiction. *The American Journal of Drug and Alcohol Abuse*, 34, 339–345. doi: 10.1080/00952990802010884
- Coccaro, E.F., Berman, M. E. & Kavoussi, R.J. (1997). Assessment of life history of aggression: development and psychometric characteristics. *Psychiatry Research*, 73(3), 147-157.

- Coccaro, E.F., Beresford, B., Minar, P., Kaskow, J. & Geraciotti, T. (2007). CSF testosterone: Relationship to aggression, impulsivity, and venturesomeness in adult males with personality disorder. *Journal of Psychiatric Research*, *41*, 488–492.
- Ducci, F. & Goldman, D. (2008). Genetic approaches to addiction: Genes and alcohol. *Addiction*, *103*(9), 1414-1428. doi: 10.1111/j.1360-0443.2008.02203.x
- Falk, Ö., Wallinius, M., Lundström, S., Frisell, T., Anckarsäter, H. & Kerekes, N. (2014). The 1 % of the population accountable for 63 % of all violent crime convictions. *Social Psychiatry and Psychiatric Epidemiology*, *49*, 559-571. doi: 10.1007/s00127-013-0783-y
- Farrington, D. P. (2002) Developmental criminology and risk-focused prevention. In M. Maguire, R. Morgan & R. Reiner. (Eds.), *The Oxford handbook of criminology* (pp. 657–701). Oxford: Oxford University Press.
- Farrington, D. P., Coid, J. W. & Murray, J. (2009). Family factors in the intergenerational transmission of offending. *Criminal Behaviour and Mental Health*, *19*, 109–124. doi: 10.1002/cbm.717
- Farrington, D. P., Jolliffe, D., Loeber, R., Stouthamer-Loeber, M. & Kalb, L. M. (2001). The concentration of offenders in families, and family criminality in prediction of boys' delinquency. *Journal of Adolescence*, *24*, 579-596. doi:10.1006/jado.2001.0424
- Frisell, T. & Långström, N. (2014). *Våldsbrottslighet. Totalbefolkningsstudier av gen- och miljöeffekter* (Projektnummer 20017:18). Norrköping: Kriminalvården.
- Frisell, T., Lichtenstein, P. & Långström, N. (2011). Violent crime runs in families: a total population study of 12.5 million individuals. *Psychological Medicine*, *41*, 97-105. doi:10.1017/S0033291710000462
- Frisell, T., Pawitan, Y., Långström, N. & Lichtenstein, P. (2012). Heritability, assortative mating and gender differences in violent crime: results from a total population sample using twin, adoption, and sibling models. *Behavior Genetics*, *42*, 3–18. doi: 10.1007/s10519-011-9483-0
- Guttormsson, U. & Leifman, H. (2016). *ESPAD i Sverige: Europaperspektiv på skolungdomars drogvanor 1995-2015*. Stockholm: Centralförbundet för alkohol- och narkotikaupplysning.
- Hamshere, M. L., Langley, K., Martin, J., Agha, S. S., Stergiakouli, E., Anney, R. J., Buitelaar, J., Faraone, S. V., Lesch, K. P., Neale, B. M., Franke, B., Sonuga-Barke, E., Asherson, P., Merwood, A., Kuntsi, J., Medland, S. E., Ripke, S., Steinhausen, H. C., Freitag, C., Reif, A., Renner, T. J., Romanos, M., Romanos, J., Warnke, A., Meyer, J., Palmason, H., Vasquez, A. A., Lambregts-Rommelse, N., Roeyers, H., Biederman, J., Doyle, A. E., Hakonarson, H., Rothenberger, A., Banaschewski, T., Oades, R. D., McGough, J. J., Kent, L., Williams, N., Owen, M. J., Holmans, P., O'Donovan, M. C. & Thapar, A. (2013). High loading of polygenic risk for ADHD in children with comorbid aggression. *The American Journal of Psychiatry*, *170*, 909-916.

- Hjern, A., Arat, A. & Vinnerljung, B. (2014). *Att växa upp med föräldrar som har missbruksproblem eller psykisk sjukdom – hur ser livet ut i ung vuxen ålder?* (Nka, Barn som anhöriga, 2014:4). Stockholm: CHES.
- Hofvander, B., Ståhlberg, O., Nydén, A., Wentz, E., degl'Innocenti, A., Billstedt, E., Forsman, A., Gillberg, C., Nilsson, T., Rastam, M. & Anckarsäter, H. (2011) Life History of Aggression scores are predicted by childhood hyperactivity, conduct disorder, adult substance abuse, and low cooperativeness in adult psychiatric patients. *Psychiatry Research*, 185, 280–285.
- Huesmann, L. R., Eron, L. D., Lefkowitz, M. M. & Walder, L. O. (1984). The stability of aggression over time and generations. *Developmental Psychology*, 20, 1120-1134. doi: 10.1037/0012-1649.20.6.1120
- Kakouros, E., Maniadaki, K. & Karaba, R. (2005). The relationship between attention deficit/ hyperactivity disorder and aggressive behaviour in preschool boys and girls. *Early Child Development and Care*, 175(3), 203-214. doi: 10.1080/0300443042000244037
- Kerber, R. A. (1995). Method for calculating risk associated with family history of a disease. *Genetic Epidemiology*, 12, 291-301.
- Kinner, S. A., Degenhardt, L., Coffey, C., Hearps, S., Spittal, M., Sawyer, S. M. & Patton, C. (2015). Substance use and risk of death in young offenders: A prospective data linkage study. *Australasian Professional Society and other Drugs*, 34, 46-50. doi: 10.1111/dar.12179
- Kriminalvården (2014). *Kartläggning 2013 – Ett regeringsuppdrag. En presentation av bakgrundsfaktorer hos Kriminalvårdens klienter*. Norrköping: Kriminalvården.
- Krug, E. G., Dahlberg, L. L., Mercy, J. A., Zwi, A. B. & Lozano, R. (2002). *World report on violence and health*. Geneva: World Health Organization.
- Laerd Statistics (2015). Kruskal-Wallis H test using SPSS Statistics. *Statistical tutorials and software guides*. Retrieved 2016-11-20 from <https://statistics.laerd.com>
- Laerd Statistics (2016). Chi-square test of homogeneity using SPSS Statistics. *Statistical tutorials and software guides*. Retrieved 2016-11-20 from <https://statistics.laerd.com>
- Leahy, M. (2014). *Reflections of adults on their school experiences growing up with a severely mentally ill parent*. Doctoral thesis. Dissertation Abstracts International Section A: Humanities and Social Sciences, Widener University, 2014; 74, 8-A(E). ProQuest LLC.
- Mednick, S.A., Gabrielli, W.F. & Hutchings, B. (1984). Genetic influences in criminal behaviour: evidence from an adoption cohort. *Science* 224 (4651), 891-894. doi: 10.1126/science.6719119
- Moffitt, T. E. & Caspi, A. (2001). Childhood predictors differentiate life-course persistent and adolescence-limited antisocial pathways among males and females. *Development and Psychopathology* 13(2), 355–375.

- Moffitt, T. E. (2005). Genetic and environmental influences on antisocial behaviors: evidence from behavioral-genetic research. *Advances in Genetics*, 55, 41-104. doi: 10.1016/S0065-2660(05)55003-X
- Moffitt, T. E., Caspi A., Taylor, A., Kokaua J., Milne, B. J., Polanczyk, G. & Poulton, R. (2010). How common are common mental disorders? Evidence that lifetime prevalence rates are doubled by prospective versus retrospective ascertainment. *Psychological Medicine*, 40, 899-909. doi: 10.1017/S0033291709991036
- Olseryd, J. (2015). *Alkohol- och drogpåverkan vid misshandel, hot, personrån och sexualbrott* (URN:NBN:SE:BRA-590). Stockholm: Brottsförebyggande rådet.
- Osborn, S. G. & West, D. J. (1979). Conviction records of fathers and sons compared. *British Journal of Criminology*, 19(2), 120-133.
- Pulay, A. J., Dawson, D. A., Hasin, D. S., Goldstein, R. B., Ruan, W. J., Pickering, R. P., Huang, B., Chou, S. P. & Grant, B. F. (2008). Violent behavior and DSM-IV psychiatric disorders: Results from the national epidemiological survey on alcohol and related conditions. *Journal of Clinical Psychiatry*, 69(1), 12-22.
- Roettger, M. E., Swisher, R. R., Kuhl, D. C. & Chavez, J. (2010). Paternal incarceration and trajectories of marijuana and other illegal drug use from adolescence into young adulthood: evidence from longitudinal panels of males and females in the United States. *Addiction*, 106, 121-132. doi:10.1111/j.1360-0443.2010.03110.x
- Socialstyrelsen, the national board of health and welfare. (2016). *Statistics on Social Services for Adults with Drug Abuse or Addiction 2015* (Art.no: 2016-5-27). Retrieved 2017-04-13 from <http://www.socialstyrelsen.se/publikationer2016/2016-5-27>
- Spitzer, R. L. (1983). Psychiatric diagnosis: are clinicians still necessary? *Comprehensive Psychiatry*, 24, 399-411. doi:10.1016/0010-440X(83)90032-9
- Thornberry, T.P., Freeman-Gallant, A., Lizotte, A.J., Krohn, M.D. & Smith, C.A. (2003) Linked lives: the intergenerational transmission of antisocial behavior. *Journal of Abnormal Child Psychology* 31, 171–184.
- Trondsen, M. (2012). Living with a mentally ill parent: exploring adolescents' experiences and perspectives. *Qualitative Health Research*, 22(2), 174–188. doi: 10.1177/1049732311420736
- Van de Rakt, M., Nieuwbeerta, P. & Dirk de Graaf, N. (2008) Like father, like son. The relationships between conviction trajectories of fathers and their sons and daughters. *British Journal of Criminology*, 48, 538–556. doi:10.1093/bjc/azn014
- Van der Weijer, S. G. A., Bijleveld, C. J. H. & Blokland, A. A. J. (2014) The intergenerational transmission of violent offending. *Journal of Family Violence*, 29, 109–118. doi: 10.1007/s10896-013-9565-2

Westfelt, L. (2016). *Lagförda personer i befolkningen 2000–2014. En studie av andelen lagförda bland kvinnor, män, i olika åldersgrupper och för några utvalda brott* (URN: NBN: SE: BRA-661). Stockholm: Brottsförebyggande rådet.