Predictive validity and gender differences in a biopsychosocial model of violence risk assessment in acute psychiatry

Bjørn Magne S. Eriksen[a,b,*], Ann Færden[a], Øyvind Lockertsen[a,c], Stål Bjørkly[d,e], John Olav Roaldset[e,f,g]

[a]Oslo University Hospital, Division of Mental Health and Addiction, Acute Psychiatric Department, Oslo, Norway

[b]University of Oslo, Faculty of Medicine, Institute of Clinical Medicine, Oslo, Norway

[c]Oslo and Akershus University College of Applied Sciences, Faculty of Health Sciences, Department of Nursing and Health Promotion, Oslo, Norway

[d]Molde University College, Molde, Norway

[e]Oslo University Hospital, Centre for Research and Education in Forensic Psychiatry, Oslo, Norway

[f]Ålesund Hospital, Møre & Romsdal Health Trust, Psychiatric Department, Ålesund, Norway

[g]The Norwegian University of Science and Technology, NTNU, Trondheim, Norway

[*]Corresponding author at: Oslo University Hospital, Division of Mental Health and Addiction, Acute Psychiatric Department, P.O. Box 4956 Nydalen, N-0424 Oslo, Norway.

Tel: +4722118420, Fax: +4722118470, Mob: +4797068315

E-mail address: b.m.s.eriksen@medisin.uio.no

Abstract

Current violence risk assessment methods seem to have reached an upper limit of accuracy. More comprehensive biopsychosocial models may improve on existing methods. Research on gender differences concerning risk factors of violence is scarce and inconclusive. In this prospective study from an acute psychiatric ward, all patients admitted from March 2012 to March 2013 were included. Predictive validity and potential gender differences in a biopsychosocial model of violence risk assessment consisting of a psychosocial checklist (Violence risk screening - 10, V-RISK-10), a patient's self-report risk scale (SRS), total cholesterol (TC) and high-density lipoprotein cholesterol (HDL) were examined in an inpatient (N = 348) and a 3-months follow-up (N = 101) sample. Overall increases in explained variances and predictive values were small and non-significant compared to V-RISK-10 alone. In the inpatient sample, HDL contributed significantly to the model for men but not for women. In the follow-up sample, SRS contributed significantly for the whole sample. Results indicated that the biopsychosocial model we tested partially improved accuracy of violence risk assessments in acute psychiatry and that gender differences may exist.

Keywords: risk assessment, acute psychiatry, total cholesterol, HDL, self-report, V-RISK-10, biomarkers, screening tools

1. Introduction

The interplay between biological, psychological and social factors as a basis for understanding and treating diseases has been recognized for decades in different medical disciplines. Already in the seventies, a biopsychosocial model was proposed as a patient-centred and holistic alternative to the prevailing biomedical model (Engel, 1978). A recent review highlighted the importance of using a biopsychosocial approach regarding violence related to mental health problems (Steinert and Whittington, 2013). It was suggested that a comprehensive model could include biological factors such as genes or lesions in prefrontal brain structures, along with psychological factors (e.g. cognitive and verbal skills) and social factors (e.g. victimization or social deprivation).

Current violence risk assessment methods in psychiatry are based on psychosocial risk factors such as antisocial personality traits, psychosis, substance abuse or previous violence (Cornaggia et al., 2011; Douglas et al., 2013; Hartvig et al., 2011; Monahan et al., 2001). No structured risk tool includes the wide range of potential risk factors and risk markers that might be derived from a biopsychosocial model. The importance of research on broader approaches to violence risk assessment is emphasized in recent reviews and meta-analyses indicating that today's violence risk assessment methods seem to have reached an upper limit of accuracy (Singh et al., 2011; Yang et al., 2010). A glass-ceiling effect has been suggested, beyond which further improvements cannot be achieved (Coid et al., 2011). This underlines the importance of using multi-methods assessments and taking alternative risk factors and risk markers into account (Steinert and Whittington, 2013).

The emergency nature of acute psychiatric wards limits the range of feasible assessment routines. The only tool in use today developed and validated for screening of both inpatient and post-discharge violence risk in acute psychiatric settings is the Violence risk

screening - 10 (V-RISK-10) (Eriksen et al., 2016; Hartvig et al., 2011; Roaldset et al., 2011b). This tool also consists solely of psychological and social risk factors of violence.

According to previous findings, one potential biological risk marker of violence that is easy to use in acute settings and that fits into the biological part of a biopsychosocial model is low levels of cholesterol. Total cholesterol (TC), which consists of all cholesterol fractions in the blood, has been associated with violence risk (Asellus et al., 2014; Roaldset et al., 2011a). Low levels of the cholesterol fraction high-density lipoprotein cholesterol (HDL) has been associated with aggression in previous studies (Buydens-Branchey et al., 2000; Troisi and D'Argenio, 2006) and also with future violence according to recent findings (Eriksen et al., 2017; Roaldset et al., 2011a).

Another potentially easily implementable approach to improving existing risk assessment methods is to ask for a patient's own opinion of violence risk. This fits with Engel's biopsychosocial model by taking the patient-centred perspective into account in violence risk assessments. Several types of self-report scales are part of comprehensive tools used for risk assessments in psychiatry, such as the Self-Appraisal Questionnaire (Loza et al., 2007), the Classification of Violence Risk (COVR) (Monahan et al., 2001; Monahan et al., 2005), the Early Recognition Method (ERM) (Fluttert et al., 2010) and the Personality Assessment Inventory (Gardner et al., 2015). However, we found only three previous studies addressing patients' direct judgement of violence risk (Lockertsen et al., 2017; Roaldset and Bjorkly, 2010; Skeem et al., 2013). Two of these were using a self-report risk scale (SRS) (Lockertsen et al., 2017; Roaldset and Bjorkly, 2010). Results indicated that patients' direct judgement of violence risk might inform existing risk assessment methods.

Gender differences in types of violence (Robbins et al., 2003) and in personality traits related to psychopathy (Murphy et al., 2016) have been reported. Gender differences in violence risk assessments have also been reported (Nicholls et al., 2004), although findings on

this issue, especially from acute settings, seem to be inconclusive (Brown and Langrish, 2012; Daffern, 2007). In our literature search, the majority of previous studies on violence risk assessments were conducted on male patients in forensic and long-term settings. Some studies with positive findings on HDL cholesterol as a potential risk marker of violence were only performed on male patient groups (Buydens-Branchey et al., 2000; Troisi and D'Argenio, 2006).

In a recent study from an acute psychiatric ward, a model for violence risk assessments consisting of V-RISK-10, TC and SRS showed significant incremental validity compared to the use of V-RISK-10 alone, but no gender differences were found (Roaldset et al., 2012). To our knowledge, this is the only previous study investigating such a biopsychosocial model in an acute psychiatric setting.

There seems to be a need for violence risk assessment models that include additional risk factors and risk markers than those used in current models. Previous research from the present project found that a lower percentage of inpatient violence was explained by V-RISK-10 for women than men (Eriksen et al., 2016), that SRS might be a risk marker of inpatient violence, with stronger results for females than males (Lockertsen et al., 2017), and that HDL was a potential risk marker for violence among men, but not women (Eriksen et al., 2017). Hence, our main objectives in this study were to investigate the possible incremental validity of a biopsychosocial model for violence risk assessment by combining TC, HDL and SRS with V-RISK-10 and to explore possible gender differences in the model.

2. Methods

2.1. Design, setting and sample selection

This was a prospective naturalistic observational study that included all patients admitted to the acute psychiatric ward at Oslo University Hospital, Norway, between March

21, 2012, and March 20, 2013. This ward admits adults (18 years or older) from a catchment area of 204,000 people.

A total of 558 patients were originally included. Thirty patients used their right to withdraw, resulting in an initial sample of 528 patients from 717 admissions. This initial sample overlaps with three previous studies that investigated different selections of baseline and/or outcome variables. Because different variables were missing in the records, there were slightly different selections of patients and admissions in the final samples of each of these studies (Eriksen et al., 2016; Eriksen et al., 2017; Lockertsen et al., 2017). As in the previous studies, in this one, one admission was chosen for each patient according to the presence of baseline variables and violence (see 2.2. Procedure).

In the inpatient sample, 180 of the 528 originally included patients were excluded due to missing or incomplete recordings of V-RISK-10, SRS, TC and/or HDL at admission. The final inpatient sample then consisted of 348 patients: 156 men and 192 women.

A total of 383 of the 528 originally included patients were lost during follow-up due to missing post-discharge recordings of violence (see 2.4. Outcome variables), giving an initial follow-up sample of 145 patients with such recordings. Forty-four of these were excluded due to missing or incomplete recordings of V-RISK-10 and/or SRS before discharge, and/or TC/HDL at admission. The final follow-up sample then consisted of 101 patients: 47 men and 54 women.

The Regional Committee for Medical and Health Research Ethics approved the study, with an exemption from asking for patients' informed consent to be included on the condition that all patients could withdraw at any time. The National Police Directorate approved access to police records (the STRASAK register) for additional recordings of violent episodes (see 2.4. Outcome variables).

2.2. Procedure

All patients received verbal and written information about the project and their right to withdraw. The information was given after admission and before discharge. V-RISK-10 and SRS were recorded by the physician on duty as part of the admission procedure (See 2.3. Baseline variables). Routine blood samples (to measure TC and HDL) were taken by nurses at the ward the morning (or the first regular working day) after admission. Samples were mostly taken non-fasting, levels of TC and HDL not normally being affected by meal intake (Nakamura et al., 2016).

Episodes of threats and violence during hospital stays were recorded by the staff using the Staff Observation Aggresion Scale – Revised (SOAS-R) (Nijman et al., 1999). To compensate for possible under-reporting, researchers recorded additional information about violence in the SOAS-R taken from hospital records.

V-RISK-10 and SRS on the day of discharge were recorded as part of the discharge procedure by the physician or psychologist in charge. Episodes of violence during the first three months after discharge were coded in a recording sheet by the patient's therapist at three collaborating outpatient clinics. If a patient had been discharged and then admitted to the ward again during the project, violence during that post-discharge period was recorded in the recording sheet by the hospital staff.

If a patient was readmitted to the ward during the project period, his or her previous post-discharge follow-up period was ended, and the patient was re-included with a new file number. Patients who had more than one hospital stay or post-discharge period with complete recordings of V-RISK-10, SRS, TC and HDL were only counted once in the analyses. For patients with recorded violence in more than one period, the period with the most serious violent episode was considered most important and chosen. For non-violent patients, the first period with complete recordings of baseline variables was chosen.

2.3. Baseline variables

V-RISK-10 was recorded at admission and before discharge. This is a screening tool for violence risk developed for acute and general psychiatry and consists of the following 10 items (Eriksen et al., 2016; Hartvig et al., 2011; Hartvig et al., 2007; Roaldset et al., 2011b):

1. Previous or current violence.
2. Previous or current violent threats.
3. Previous or current substance abuse.
4. Previous or current severe mental illness.
5. Personality disorders.
6. Lack of insight into illness or behaviour.
7. Suspiciousness.
8. Lack of empathy.
9. Unrealistic planning.
10. Exposure to future stress-situations.

The first five items are 'historical' (unchangeable, if present) and the last five items are 'dynamic' (may change over time). The items are coded as No = *definitely absent* or *does not apply*; Maybe/moderate = *possibly present* or *present only to a limited extent*; Yes = *definitely present*; or Don't know = *insufficient valid information to permit coding the item*. Based on items' scores, clinical judgement and all other available information, the clinician assesses the violence risk as *low*, *moderate* or *high* and decides whether there is a basis for more detailed violence risk assessment and implementation of preventive measures. Sum-scores and cut-offs are not used in the current clinical version of the tool.

In line with a recent study on the same material (Eriksen et al., 2016), a sum score of the V-RISK-10 (range 0 - 20) to use in the analyses was coded with the following values: *No* = 0, *Maybe/moderate* and *Don't know* = 1, and *Yes* = 2.

Serum measures of TC and HDL in millimoles per litre (mmol / L) were obtained from routine blood tests at admission and analyzed at the Department of Medical Biochemistry at Oslo University Hospital with 'enzymatic colorimetric method' for TC and 'homogeneous enzymatic colorimetric method' for HDL ('Cobas 8000 c702', Roche Diagnostics, Oslo, Norway).

To obtain a patient's own assessment of violence risk, we used the SRS (Roaldset and Bjorkly, 2010). Patients were asked a similar question as part of the admission and discharge procedures: 'What is your own opinion of the risk that you will act violently against other people: during your hospital stay/during the first three months after discharge?'. The patients were asked to grade their opinion as *no risk*, *low risk*, *moderate risk*, *high risk* or *don't know the risk*. The response was recorded into one of six categories: (1) *no risk*, (2) *low risk*, (3) *moderate risk*, (4) *high risk*, (5) *don't know the risk* or (6) *will not answer about the risk* (if the patient refused to answer).

In the inpatient and follow-up samples, *low risk*, *don't know* and *will not answer* represented increased violence risk: In the inpatient sample, 25 (11%) with violence in *no risk* versus 10 (16%) in *low risk*, 8 (33%) in *don't know* and 13 (43%) in *will not answer*; and in the follow-up sample, 7 (14%) in *no risk* versus 12 (43%) in *low risk*, 5 (29%) in *don't know* and 3 (75%) in *will not answer*. The findings on increased proportions of violence for the *don't know* and *will not answer* categories are consistent with recent findings in two different studies, of which one (from 2017) was from this material (Lockertsen et al., 2017; Roaldset and Bjorkly, 2010). We have earlier reported that *don't know* scorings seem to represent increased violence risk in other types of risk assessments in acute settings (Eriksen et al.,

2016; Roaldset et al., 2017). Only three patients (one violent) in the inpatient sample and one (non-violent) in the follow-up sample reported *moderate risk*, and one (non-violent) in the inpatient sample and none in the follow-up sample reported *high risk*, but from a clinical point of view, these categories also represent increased violence risk. To increase power and decrease possibility of Type II errors, in all the analyses, SRS was therefore transformed into a dichotomous variable. *Low*, *moderate*, *high*, *don't know* and *will not answer* were merged into one category representing increased violence risk (= *risk*), with *no risk* as the reference category.

Demographic and clinical variables including age, gender, proportion involuntarily admitted, employment and educational status, and ICD-10 diagnosis (WHO, 1992) were gathered from hospital records.

2.4. Outcome variables

The outcome variable was violent behaviour, including light/moderate and severe physical violence and threats of violence. *Violence* was defined as in previous studies: *Physical violence* was defined as a physical act against another person involving the use of body parts or objects, with a clear intent to cause physical injury on that person. *Verbal threats* were defined as verbal communications that conveyed a clear intent to inflict physical injury on another person (Dean et al., 2006; Monahan et al., 2005; Swanson et al., 2006).

During the hospital stay, the SOAS-R was used to record violence to others (Nijman et al., 1999). Only physical violence and verbal threats as defined above were recorded. Nurses were familiar with the form from a prior project in the ward.

Post-discharge violence was coded in a recording sheet and categorized into (1) violent threats, (2) light/moderate violent acts (e.g. punches and kicks without serious injury), and (3) severe violent acts (e.g. use of weapons, sexual violence or other violence with intent

to inflict serious injury on another person). The patients were monitored for 'no occurred episodes', 'yes, episodes have occurred' or 'don't know if episodes have occurred' in each category. Prior to the start of the project, the therapists were briefed on how to record violence. Episodes of post-discharge violence were also recorded from out-patient hospital records and from police records.

2.5. Statistics

SPSS version 23.0 was used to analyse data. *Don't know* recordings of post-discharge violence were treated as missing and excluded. To increase statistical power and decrease the possibility of Type II errors, verbal threats and categories of physical violence were combined into one dichotomous outcome variable: *any violence*. The inpatient and follow-up samples were analysed separately. TC and HDL, which were only recorded after admission, were used in analyses for both the inpatient and follow-up samples, whereas V-RISK-10 and SRS recorded after admission were used for the inpatient sample, and V-RISK-10 and SRS recorded before discharge were used for the follow-up sample (see 2.2. Procedure). The dichotomous version of SRS was used in all analyses (see 2.3. Baseline variables). For TC and HDL, the continuous variables were used in all logistic regression analyses. A dichotomous version of HDL was used in the 'clinical version' of the biopsychosocial model (V-RISK-EXT) (see 2.5.2. Comparison of V-RISK-10 and V-RISK-EXT).

To test differences between groups of patients, the Mann-Whitney *U*-test and Student's *t*-test were used for continuous variables. Chi-square tests or Fisher's exact tests were used for categorical variables. Pearson's Correlation Coefficient (Pearson's *r*) was used to estimate the association between inpatient and post-discharge violence.

2.5.1. Logistic regression analyses

Uni-, bi- and multivariate binary logistic regression analyses were conducted to estimate odds ratios (OR). A stepwise procedure was used to monitor the progression of the chi-square test of variance to examine whether the baseline variables SRS, TC and/or HDL added significantly to the variance explained by V-RISK-10 alone. V-RISK-10 was entered in the first step and SRS, TC and/or HDL were added in the second step. Other variables with a potentially positive impact on violence were added in a third step.

Besides gender-stratification, the multivariate model was controlled for two other variables with a potentially positive impact on violence: *involuntarily admitted* and *age.* Being involuntarily admitted is known to be associated with violence in psychiatric samples (e.g. Cornaggia et al., 2011) and also showed a significant positive association with violence in our samples (see 3.1. Descriptive data and proportions of violence). *Age* had non-significant associations with violence in our samples, but was still included because younger age is known to be associated with violence in numerous other findings from psychiatric samples (e.g. Dack et al., 2013). *Unemployed* and *only primary school* were excluded because of a considerable number of missing cases (18% in total when including these two variables), in line with previous studies from the same material (Eriksen et al., 2016; Eriksen et al., 2017). *Psychosis* and *personality disorder* are overlapping with items 4 and 5 in V-RISK-10 and were therefore not included.

To test if there were significant gender differences in the components of the biopsychosocial model (V-RISK-10, SRS, TC and HDL) or in V-RISK-EXT (see 2.5.2. Comparison of V-RISK-10 and V-RISK-EXT), we performed interaction analyses. This was done by including the interaction item (gender * the test variable, e.g. HDL), gender and the test variable in a binary logistic regression analysis with violence as the outcome. *P*-value < 0.05 of the interaction item (e.g. gender * HDL) indicates a significant impact of gender on the association between HDL and violence.

2.5.2. Comparison of V-RISK-10 and V-RISK-EXT

An extended version of V-RISK-10, 'V-RISK-EXT' was constructed as a 'clinical version' of the biopsychosocial model, based on findings in uni-, bi- and multivariate logistic regression analyses, and was compared with V-RISK-10. V-RISK-EXT was computed as a sum variable similar to the V-RISK-10 sum score (see 2.3. Baseline variables).

Due to non-significant ORs close to 1 in all samples in uni-, bi- and multivariate analyses of TC (see 3. Results), TC was not used as part of V-RISK-EXT. Together with the 10 items in V-RISK-10, SRS and HDL were treated as separate items (number 11 and 12) in V-RISK-EXT, and scorings were done similar to items in V-RISK-10 (see 2.3. Baseline variables): *no* = 0 and *yes* = 2 for each item. Unlike V-RISK-10, a *maybe/moderate* score = 1 was not computed for SRS, which was transformed into a dichotomous variable (see 2.3. Baseline variables), or for HDL, where mean value was used as cut-off, respectively. Hence, for SRS, *no risk* ('negative test') = 0 and *risk* ('positive test') = 2; and for HDL, cut-off for a 'positive test' (= 2) ≤ mean value for the whole sample = 1.49 mmol/L and values above 1.49 mmol/L ('negative test') = 0, giving a range of 0 - 24 for V-RISK-EXT used for the whole inpatient and follow-up samples (as opposed to 0 - 20 for the V-RISK-10 sum score).

When examining V-RISK-EXT for each gender, only V-RISK-10 and SRS, and not HDL, was used in V-RISK-EXT for women. HDL was excluded for women because of non-significant ORs in all uni-, bi- and multivariate logistic regression analyses. Furthermore, HDL for women only had ORs > 1. For men, V-RISK-10, SRS and HDL were used in V-RISK-EXT, as for the whole sample. The HDL mean value for men = 1.32 mmol/L and lower values were used as cut-off for a 'positive test' (= 2). Hence, ranges of V-RISK-EXT for each gender were 0 - 22 for women and 0 - 24 for men.

To assess overall predictive accuracy of V-RISK-10 and V-RISK-EXT, analysis of the area under the curve (AUC) of the receiver operating characteristic (ROC) was performed. ROC-AUC is to a lesser degree dependent on the base rate of the outcome variable in a given sample and is recommended in risk assessment studies (Singh et al., 2015). The test is a sensitivity / (1-specificity) plot of all values on the checklist, ranging from 0 to 1, where 0.5 equals chance and 1.0 equals a perfect prediction.

In line with recent recommendations for violence risk assessment studies (Singh et al., 2015), other predictive values were also estimated for scores at cut-off or higher for V-RISK-10 and V-RISK-EXT: (1) sensitivity, (2) specificity, (3) positive predictive value (PPV), (4) negative predictive value (NPV), (5) number needed to assess (NNA, how many patients were needed to be assessed to identify one true violent patient, which is equal to 1/PPV) and (6) the likelihood ratio (LR). The LR determines to what extent the odds of an outcome (e.g. violence) increases when a test is positive (LR+) and decreases when a test is negative (LR–). For tests with only two outcomes, the LR+ can be expressed as sensitivity / (1 – specificity) and LR– as (1 - sensitivity) / specificity (Deeks and Altman, 2004). Cut-off for a 'positive test' was chosen as the score with the highest sensitivity and specificity.

3. Results

3.1. Descriptive data and proportions of violence

Descriptive data with distribution of clinical and demographical variables and missing analysis are displayed in Table 1. An overview of variables in the biopsychosocial model, demographic and clinical variables with comparisons between violent and non-violent patients, is displayed in Table 2.

Proportions of violence were as follows: In the inpatient sample, 57 (16%) of the whole sample, 38 (24%) of men and 19 (9.9%) of women; in the follow-up sample, 27 (27%)

of the whole sample, 14 (30%) of men and 13 (24%) of women. There was no significant correlation between inpatient and post-discharge violence (calculated for the follow-up sample, $N = 101$): Pearson's $r = 0.035$, $p = 0.728$. There were no significant differences in inpatient violence between included and missing patients in the inpatient sample, χ^2 (df) = 1.6 (1), $p = 0.204$, or in post-discharge violence in the follow-up, χ^2 (df) = 0.81 (1), $p = 0.369$. Men were significantly more violent than women in the inpatient sample, χ^2 (df) = 13 (1), $p < 0.001$, but not in the follow-up, χ^2 (df) = 0.42 (1), $p = 0.518$.

3.2. Univariate results and contributions of the components of the biopsychosocial model to V-RISK-10

Univariate findings are displayed in Table 3. Contributions of SRS, TC and HDL to V-RISK-10 in bivariate analyses are displayed in Table 4. HDL added significantly to V-RISK-10 for men in the inpatient sample.

3.3. Multivariate analyses of the biopsychosocial model

Multivariate results are displayed in Tables 5A and 5B. In the inpatient sample, V-RISK-10 remained significant. HDL was significant in the model only for men, including when controlled for *involuntarily admitted* and *age*. In the follow-up, V-RISK-10 remained significant for the whole sample and for women. SRS was significant in the model for the whole sample and also remained borderline significant when controlled for *involuntarily admitted* and *age*.

3.4. Gender differences in the biopsychosocial model

In the inpatient sample, gender difference was borderline significant for HDL ($p = 0.051$) and non-significant for V-RISK-10 ($p = 0.730$), TC ($p = 0.535$) and SRS ($p = 0.246$).

In the follow-up sample, gender difference was significant for HDL ($p = 0.030$) and non-significant for V-RISK-10 ($p = 0.961$), TC ($p = 0.482$) and SRS ($p = 0.644$).

Gender differences in ORs for V-RISK-EXT were non-significant both in the inpatient, $p = 0.968$, and follow-up, $p = 0.625$.

3.5. Comparisons between a clinical version of the biopsychosocial model (V-RISK-EXT) and V-RISK-10

See Table 6 for overall predictive values (AUC) and Table 7 for other predictive values. AUC only slightly improved for V-RISK-EXT compared to V-RISK-10 alone. Sensitivities and LR- improved slightly for V-RISK-EXT in the inpatient sample, especially for men, and specificities, PPV, NNA and LR+, improved slightly in the follow-up sample for the whole sample and gender-stratified.

In the inpatient sample, OR for V-RISK-EXT for the whole sample = 1.2, 95% CI = 1.2 - 1.3, $p < 0.001$; for men, OR = 1.2, 95% CI = 1.1 - 1.3, $p < 0.001$; and for women, OR = 1.3, 95% CI = 1.1 - 1.4, $p < 0.001$. In the follow-up sample, for the whole sample, OR = 1.2, 95% CI = 1.1 - 1.3, $p <0.001$; for men, OR = 1.2, 95% CI = 1.1 - 1.4, $p = 0.007$; and for women, OR = 1.2, 95% CI = 1.0 - 1.4, $p = 0.012$. ORs for V-RISK-EXT were similar to ORs for V-RISK-10 (Table 3).

4. Discussion

4.1. Main findings

Predictive values improved only slightly when a biopsychosocial model was used rather than the V-RISK-10 alone. V-RISK-10 remained a significant component of the biopsychosocial model, except for men in the follow-up sample. TC was not a significant component of the biopsychosocial model in any samples. In the inpatient sample, HDL was a

significant component of the model only for men, even when controlled for other variables with a potential positive impact on violence, and added a significant increase in explained variance to V-RISK-10 alone. In the follow-up sample, SRS was a significant component of the biopsychosocial model for the whole sample.

4.2. Inpatient sample

We found only a small and non-significant improvement in AUC for the clinical version of the biopsychosocial model (V-RISK-EXT) compared to V-RISK-10 alone. Other predictive values were also similar or only slightly improved. Explained variance for the biopsychosocial model also did not improve compared to V-RISK-10 alone (see Table 5A). Findings are consistent with those of Roaldset et al. (2012) who investigated a similar model.

SRS did not contribute significantly to the model, consistent with previous findings by Roaldset et al. Increased risk awareness and prevention of violence for patients who reported *risk* at admission might be an explanation. Hospital admissions, of which 44% were involuntary (see Table 1) may also have been characterized by coercion and hostility, which might have resulted in dishonest answers. Honesty in self-reports could have been affected by inpatients' answering questions about their own risk of violence to a stranger as well as by their possible fear of being subjected to coercion or restraint.

As in our study on cholesterol and violence from the same material (Eriksen et al., 2017), but in contrast to other findings on TC (Asellus et al., 2014; Roaldset et al., 2011a), TC was not a significant risk marker of violence and did not contribute significantly to the biopsychosocial model. HDL also did not contribute significantly to the model for the whole inpatient sample. In the study by Roaldset et al. (2011a), TC, and not HDL, was used in a similar model and was significant. The non-significant results for HDL for women in our

study might explain the non-significant results for HDL for the whole sample, as women constituted 55% of the inpatient sample (see Table 1).

The biopsychosocial model only explained about 19% of inpatient violence. Results indicate that factors other than SRS, TC, HDL and items in V-RISK-10 are also important for violence risk in an inpatient setting. For example, different types of aversive interactions between patients and staff, such as denial of requests or limit-setting situations, might trigger aggression in a ward. Factors like ward culture, types of activities, number and experience of staff and number of patient beds could also be significant (Hamrin et al., 2009; Newbill et al., 2010; Tishler et al., 2013). Such factors were not included in this study, but should be topics of future research on similar models.

4.3. Follow-up sample

Increase in AUC for V-RISK-EXT was small and non-significant compared to V-RISK-10 alone, similar to inpatient findings. Explained variance of the biopsychosocial model (see Table 5B) improved some, but the increase was still non-significant. Although sensitivity of V-RISK-EXT compared to V-RISK-10 slightly decreased, specificity, PPV, NNA and LR+ improved. These improvements, although modest, might be important from a clinical point of view, as unnecessary detainment of patients due to false positive risk assessments could be prevented.

SRS, but not TC and HDL, was significant as part of the biopsychosocial model, as was also V-RISK-10. SRS became borderline significant ($p = 0.051$) when controlled for other variables with a potential positive impact on violence (see Table 5B). Self-reports as part of comprehensive models for violence risk assessments might be beneficial because they emphasize the patient's own perspective and require an interaction between therapist and patient. Inclusion of the patient in the therapeutic decision process might have a positive

impact on the development of a therapeutic relationship (Hamann et al., 2003). More honest answers at discharge than at admission might have been due to treatment of persecutory delusions or development of a more positive therapeutic relationship during the hospital stay.

Though SRS was significant, explained variance of the model was still only about 21%, indicating that other factors with an impact on violence are also important post-discharge. Such factors could, for instance, be level of aftercare, proximity to potential victims (e.g. living in a violent relationship), availability of drugs or alcohol, or specific personality traits that might trigger violent episodes in a community context (e.g. high levels of anger or impulsivity) (Doyle et al., 2012; Monahan et al., 2001).

4.4. Gender-specific findings

AUC values for the clinical version of the biopsychosocial model (V-RISK-EXT) for each gender were similar to the whole sample, although men had a slightly greater increase than women in AUC compared to V-RISK-10 alone from 0.74 to 0.78 in the follow-up. In the inpatient sample, sensitivities seemed to improve slightly more across gender than for the whole sample, and LR- also improved more, especially for men (from 0.25 to 0.11). In the follow-up, specificities, PPV, NNA and LR+ improved similarly to the whole sample. Both inpatient and follow-up explained variances of the biopsychosocial model compared to V-RISK-10 alone improved more across gender than for the whole sample, but improvements were not significant. In the inpatient sample, the model explained more of male than female violence, whereas explained variances of post-discharge violence were equal across gender (see Tables 5A and 5B). Overall, the biopsychosocial model performed similarly well, and, in some areas, slightly better, in gender-stratified analyses.

V-RISK-10 became a non-significant component of the biopsychosocial model for men in the follow-up sample, and SRS did not contribute significantly in either the inpatient

or follow-up samples across gender. These results might indicate that smaller subgroups could have produced Type II errors in gender-stratified multivariate analyses, because SRS was significant for the whole sample in the follow-up, and V-RISK-10 was significant in the larger samples.

TC was, as in the whole inpatient and follow-up samples, non-significant in all gender-stratified analyses. HDL was non-significant for women, but a significant component in the biopsychosocial model for men in the inpatient sample, even when controlled for other variables with a potential positive impact on violence. HDL also contributed significantly to V-RISK-10 alone for men. Though this was not the case in the follow-up sample, the multivariate effect size for men in the follow-up (OR = 0.21) was similar to the inpatient sample (OR = 0.25), and univariate gender difference was also significant. Findings are consistent with our previous findings on HDL from the same material, indicating that this is a biological risk marker of violence for men, but not for women (Eriksen et al., 2017).

One important constituent in the CNS is cholesterol, and because men on average have larger brain sizes than women, one possible explanation for the gender differences that our findings indicate may be increased sensitivity for men to low cholesterol levels in the CNS (Wallner and Machatschke, 2009). A link between low TC levels in the CNS and low serotonin levels, which might increase risk of both aggression against others and other types of aggression, such as suicidal behaviour, had already been proposed in the early nineties and is still a primary hypothesis (Engelberg, 1992). The possibility that HDL is a risk marker for suicidality specifically among women has also been suggested (Emet et al., 2015). Low cholesterol levels might also be linked to other psychological factors, such as anhedonia (Loas et al., 2016) or impaired psychological health (Sahebzamani et al., 2013). These aspects are not addressed in our research.

Significant findings in bi- and multivariate analyses (Tables 4 and 5A) indicate an independent contribution of HDL to the biopsychosocial model for men. This may suggest that HDL explains other aspects of violence for men than other variables in the biopsychosocial model and the other variables with a potential positive impact in violence which were controlled for. A link to impulsive types of violence may be hypothesized. One study reported a possible connection between low HDL and impulsivity (Buydens-Branchey et al., 2000). Associations between low TC, impulsivity, and violence have also been suggested (Conklin and Stanford, 2008). Other research has found the opposite results (Paavola et al., 2002). Overall, findings in this area are sparse and not yet conclusive.

4.5. Strengths and limitations

The prospective naturalistic design increases external validity, but the heterogeneous patient population in the ward might make findings difficult to interpret on an individual level. However, a broader range of risk factors and risk markers in the biopsychosocial model (SRS, TC and HDL) might counteract this and underlines multi-methods assessments as a strength of this study. Another strength is the use of different statistical methods (Singh et al., 2015). Investigating only one psychiatric ward limits generalizability. Under-reporting by the staff is known from other studies using SOAS-R (Hvidhjelm et al., 2014; Nijman et al., 2005), but our use of multiple sources of information may have mitigated possible under-reporting.

Low *n* in the follow-up sample may have increased the possibility of Type II errors, especially in multivariate analyses (Table 5B). Patients who were missing from the follow-up sample had shorter hospital stays which might have increased post-discharge violence risk, although proportions of violence between included and missing patients were not significantly different. Missing recordings were to some degree counteracted by additional information

retrieved from post-discharge records and police records; still, the majority of patients were lost during follow-up.

In studies on unwanted events like violence or self-harm, staff and therapists cannot just observe when such events happen, but are obliged to implement preventive measures (e.g. medications, increased staffing around the patient). As a result of such interventions, violent episodes may be prevented and an otherwise 'true positive' prediction might turn out to be 'false'. Furthermore, staff and therapists who recorded incidents were not blinded to risk scores. Increased use of preventive measures for patients with higher risk scores on V-RISK-10 or SRS can therefore not be ruled out and might have resulted in a further increase in false positives. For TC and HDL, which are not established as risk markers of violence in clinical settings, such bias is less likely. Results for TC and HDL could have been influenced by other potential biases in those cases where blood samples were not taken after fasting or in any other standardized ways. However, TC and HDL values are normally not influenced by food intake (Nakamura et al., 2016). Use of statins or other medications (not recorded in this project) could also have influenced cholesterol values and violence; however, use of statins did not influence results significantly in a previous similar study (Roaldset et al., 2011a).

4.6. Conclusions and future research

Findings indicate that compared to a screening tool alone, a biopsychosocial model of violence risk assessments might partly improve predictive accuracy. The model might be of clinical importance despite the small increases in predictive accuracy across gender. Results also indicate gender differences in the use of the biological part of the model. Many results, especially in the follow-up sample, were non-significant and must be interpreted within the limitations set by the relatively small sample size. As explained variances were still low, further research on other potential risk factors of violence is also necessary. For instance,

interaction effects between genes or biomarkers and environmental adversities on violent behaviour might be present. A prior study showed an impact of TC on the relationship between victimization in childhood and adult violent behaviour (Asellus et al., 2014). Interaction between the serotonin transporter gene and childhood adversities on violence among adult men has also been found (Caspi et al., 2002). Hence, research on other populations and larger sample sizes, which include, as well, other potential risk factors, is needed before similar biopsychosocial models can be implemented in violence risk assessments in ordinary clinical practice.

Acknowledgements

We gratefully acknowledge nursing staff, physicians, psychologists and administrative staff at the Acute Psychiatric Department, Southern Oslo DPS, Nydalen DPS and Section of Early Psychoses Treatment, Oslo University Hospital, and especially the coordinators Kate Storm, Øyvind Ohnstad, Ingunn Verdal, Suraj Bahadur Thapa, Anniken Eid and Marit Forthun Vågsnes for their help with data collection and recordings. We also thank the head of the Division of Mental Health and Addiction, Marit Bjartveit, and the head of the Acute Psychiatric Department, Thorbjørn Harald Sundin, for their support for the project.

Funding: This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

References

Asellus, P., Nordstrom, P., Nordstrom, A.L., Jokinen, J., 2014. Cholesterol and the "cycle of violence" in attempted suicide. Psychiatry Res. 215 (3), 646-650.

Brown, S., Langrish, M., 2012. Evaluation of a risk assessment tool to predict violent behaviour by patients detained in a psychiatric intensive care unit. J. Psychiatr. Intensive Care 8 (01), 35-41.

Buydens-Branchey, L., Branchey, M., Hudson, J., Fergeson, P., 2000. Low HDL cholesterol, aggression and altered central serotonergic activity. Psychiatry Res. 93 (2), 93-102.

Caspi, A., McClay, J., Moffitt, T.E., Mill, J., Martin, J., Craig, I.W. et al., 2002. Role of genotype in the cycle of violence in maltreated children. Science 297 (5582), 851-854.

Coid, J.W., Yang, M., Ullrich, S., Zhang, T., Sizmur, S., Farrington, D. et al., 2011. Most items in structured risk assessment instruments do not predict violence. J. Forens. Psychiatry Psychol. 22 (1), 3-21.

Conklin, S.M., Stanford, M.S., 2008. Premeditated aggression is associated with serum cholesterol in abstinent drug and alcohol dependent men. Psychiatry Res. 157 (1-3), 283-287.

Cornaggia, C.M., Beghi, M., Pavone, F., Barale, F., 2011. Aggression in psychiatry wards: a systematic review. Psychiatry Res. 189 (1), 10-20.

Dack, C., Ross, J., Papadopoulos, C., Stewart, D., Bowers, L., 2013. A review and meta-analysis of the patient factors associated with psychiatric in-patient aggression. Acta Psychiatr. Scand. 127 (4), 255-268.

Daffern, M., 2007. The predictive validity and practical utility of structured schemes used to assess risk for aggression in psychiatric inpatient settings. Aggress. Violent Behav. 12 (1), 116-130.

Dean, K., Walsh, E., Moran, P., Tyrer, P., Creed, F., Byford, S. et al., 2006. Violence in women with psychosis in the community: prospective study. Br. J. Psychiatry 188, 264-270.

Deeks, J.J., Altman, D.G., 2004. Diagnostic tests 4: likelihood ratios.[comment]. BMJ 329 (7458), 168-169.

Douglas, K.S., Hart, S.D., Webster, C.D., Belfrage, H., 2013. HCR-20V3: Assessing Risk of Violence – User Guide. Burnaby, Canada: Mental Health, Law, and Policy Institute, Simon Fraser University.

Doyle, M., Carter, S., Shaw, J., Dolan, M., 2012. Predicting community violence from patients discharged from acute mental health units in England. Soc. Psychiatry Psychiatr. Epidemiol. 47 (4), 627-637.

Emet, M., Yucel, A., Ozcan, H., Akgol Gur, S.T., Saritemur, M., Bulut, N. et al., 2015. Female attempted suicide patients with low HDL levels are at higher risk of suicide re-attempt within the subsequent year: a clinical cohort study. Psychiatry Res. 225 (1-2), 202-207.

Engel, G.L., 1978. The biopsychosocial model and the education of health professionals. Ann. N. Y. Acad. Sci. 310 (1), 169-181.

Engelberg, H., 1992. Low serum cholesterol and suicide. Lancet 339 (8795), 727-729.

Eriksen, B.M.S., Bjorkly, S., Faerden, A., Friestad, C., Hartvig, P., Roaldset, J.O., 2016. Gender differences in the predictive validity of a violence risk screening tool: a prospective study in an acute psychiatric ward. Int. J. Forensic Ment. Health 15 (2), 186-197.

Eriksen, B.M.S., Bjørkly, S., Lockertsen, Ø., Færden, A., Roaldset, J.O., 2017. Low cholesterol level as a risk marker of inpatient and post-discharge violence in acute

psychiatry – a prospective study with a focus on gender differences. Psychiatry Res. 255, 1-7.

Fluttert, F.A., van Meijel, B., Nijman, H., Bjorkly, S., Grypdonck, M., 2010. Preventing aggressive incidents and seclusions in forensic care by means of the 'Early Recognition Method'. J. Clin. Nurs. 19 (11-12), 1529-1537.

Gardner, B.O., Boccaccini, M.T., Bitting, B.S., Edens, J.F., 2015. Personality Assessment Inventory scores as predictors of misconduct, recidivism, and violence: a meta-analytic review. Psychol. Assess. 27 (2), 534-544.

Hamann, J., Leucht, S., Kissling, W., 2003. Shared decision making in psychiatry. Acta Psychiatr. Scand. 107 (6), 403-409.

Hamrin, V., Iennaco, J., Olsen, D., 2009. A review of ecological factors affecting inpatient psychiatric unit violence: implications for relational and unit cultural improvements. Issues Ment. Health Nurs. 30 (4), 214-226.

Hartvig, P., Roaldset, J.O., Moger, T.A., Ostberg, B., Bjorkly, S., 2011. The first step in the validation of a new screen for violence risk in acute psychiatry: the inpatient context. Eur. Psychiatry 26 (2), 92-99.

Hartvig, P., Østberg, B., Alfarnes, S.A., Moger, T.A., Skjønberg, M., Bjørkly, S., 2007. Violence risk screening - 10 (V-RISK-10). Centre for Research and Education in Forensic Psychiatry, Oslo, Norway. http://sifer.no/files/v_risk_10_english.pdf

Hvidhjelm, J., Sestoft, D., Bjorner, J.B., 2014. The Aggression Observation Short Form identified episodes not reported on the Staff Observation Aggression Scale - Revised. Issues Ment. Health Nurs. 35 (6), 464-469.

Loas, G., Dalleau, E., Lecointe, H., Yon, V., 2016. Relationships between anhedonia, alexithymia, impulsivity, suicidal ideation, recent suicide attempt, C-reactive protein

and serum lipid levels among 122 inpatients with mood or anxious disorders. Psychiatry Res. 246, 296-302.

Lockertsen, Ø., Procter, N., Vatnar, S.K.B., Færden, A., Eriksen, B.M.S., Roaldset, J.O. et al., 2017. Screening for risk of violence using service users' self-perceptions: a prospective study from an acute mental health unit. Int. J. Ment. Health Nurs. https://doi.org/10.1111/inm.12413 [Epub ahead of print]

Loza, W., Loza-Fanous, A., Heseltine, K., 2007. The myth of offenders' deception on self-report measure predicting recidivism. J. Interpers. Violence 22 (6), 671-683.

Monahan, J., Steadman, H., Silver, E., Appelbaum, P., Robbins, P., Mulvey, E. et al., 2001. Rethinking Risk Assessment: The MacArthur Study of Mental Disorder and Violence. Oxford University Press, New York.

Monahan, J., Steadman, H.J., Robbins, P.C., Appelbaum, P., Banks, S., Grisso, T. et al., 2005. An actuarial model of violence risk assessment for persons with mental disorders. Psychiatr. Serv. 56 (7), 810-815.

Murphy, B., Lilienfeld, S., Skeem, J., Edens, J.F., 2016. Are fearless dominance traits superfluous in operationalizing psychopathy? Incremental validity and sex differences. Psychol. Assess. 28 (12), 1597-1607.

Nakamura, A., Monma, Y., Kajitani, S., Noda, K., Nakajima, S., Endo, H. et al., 2016. Effect of glycemic state on postprandial hyperlipidemia and hyperinsulinemia in patients with coronary artery disease. Heart Vessels 31 (9), 1446-1455.

Newbill, W.A., Marth, D., Coleman, J.C., Menditto, A.A., Carson, S.J., Beck, N.C., 2010. Direct observational coding of staff who are the victims of assault. Psychol. Serv. 7 (3), 177-189.

Nicholls, T.L., Ogloff, J.R., Douglas, K.S., 2004. Assessing risk for violence among male and female civil psychiatric patients: the HCR-20, PCL:SV, and VSC. Behav. Sci. Law 22 (1), 127-158.

Nijman, H.L., Muris, P., Merckelbach, H.L.G.J., Palmstierna, T., Wistedt, B., Vos, A.M. et al., 1999. The Staff Observation Aggression Scale-Revised (SOAS-R). Aggress. Behav. 25 (3), 197-209.

Nijman, H.L., Palmstierna, T., Almvik, R., Stolker, J.J., 2005. Fifteen years of research with the Staff Observation Aggression Scale: a review. Acta Psychiatr. Scand. 111 (1), 12-21.

Paavola, P., Repo-Tiihonen, E., Tiihonen, J., 2002. Serum lipid levels and violence among Finnish male forensic psychiatric patients. J. Forens. Psychiatry 13 (3), 555-568.

Roaldset, J.O., Bakken, A.M., Bjorkly, S., 2011a. A prospective study of lipids and serotonin as risk markers of violence and self-harm in acute psychiatric patients. Psychiatry Res. 186 (2-3), 293-299.

Roaldset, J.O., Bjorkly, S., 2010. Patients' own statements of their future risk for violent and self-harm behaviour: a prospective inpatient and post-discharge follow-up study in an acute psychiatric unit. Psychiatry Res. 178 (1), 153-159.

Roaldset, J.O., Hartvig, P., Bjorkly, S., 2011b. V-RISK-10: Validation of a screen for risk of violence after discharge from acute psychiatry. Eur. Psychiatry 26 (2), 85-91.

Roaldset, J.O., Hartvig, P., Bjorkly, S., 2017. Psychometric properties and predictive validity of a police version of a violence risk screen - a pilot study. Int. J. Law Psychiatry 54, 133-139.

Roaldset, J.O., Hartvig, P., Linaker, O.M., Bjorkly, S., 2012. A multifaceted model for risk assessment of violent behaviour in acutely admitted psychiatric patients. Psychiatry Res. 200 (2-3), 773-778.

Robbins, P.C., Monahan, J., Silver, E., 2003. Mental disorder, violence, and gender. Law Hum. Behav. 27 (6), 561-571.

Sahebzamani, F.M., D'Aoust, R.F., Friedrich, D., Aiyer, A.N., Reis, S.E., Kip, K.E., 2013. Relationship among low cholesterol levels, depressive symptoms, aggression, hostility, and cynicism. J. Clin. Lipidol. 7 (3), 208-216.

Singh, J.P., Serper, M., Reinharth, J., Fazel, S., 2011. Structured assessment of violence risk in schizophrenia and other psychiatric disorders: a systematic review of the validity, reliability, and item content of 10 available instruments. Schizophr. Bull. 37 (5), 899-912.

Singh, J.P., Yang, S., Mulvey, E.P., 2015. Reporting guidance for violence risk assessment predictive validity studies: The RAGEE Statement. Law Hum. Behav. 39 (1), 15-22.

Skeem, J.L., Manchak, S.M., Lidz, C.W., Mulvey, E.P., 2013. The utility of patients' self-perceptions of violence risk: consider asking the person who may know best. Psychiatr. Serv. 64 (5), 410-415.

Steinert, T., Whittington, R., 2013. A bio-psycho-social model of violence related to mental health problems. Int. J. Law Psychiatry 36 (2), 168-175.

Swanson, J.W., Swartz, M.S., Van Dorn, R.A., Elbogen, E.B., Wagner, H.R., Rosenheck, R.A. et al., 2006. A national study of violent behavior in persons with schizophrenia. Arch. Gen. Psychiatry 63 (5), 490-499.

Tishler, C.L., Reiss, N.S., Dundas, J., 2013. The assessment and management of the violent patient in critical hospital settings. Gen. Hosp. Psychiatry 35 (2), 181-185.

Troisi, A., D'Argenio, A., 2006. Apolipoprotein A-I/apolipoprotein B ratio and aggression in violent and nonviolent young adult males. J. Psychiatr. Res. 40 (5), 466-472.

Wallner, B., Machatschke, I.H., 2009. The evolution of violence in men: the function of central cholesterol and serotonin. Prog. Neuropsychopharmacol. Biol. Psychiatry 33 (3), 391-397.

WHO, 1992. The ICD-10 classification of mental and behavioural disorders, clinical description and diagnostic guidelines. World Health Organization, Geneva.

Yang, M., Wong, S.C., Coid, J., 2010. The efficacy of violence prediction: a meta-analytic comparison of nine risk assessment tools. Psychol. Bull. 136 (5), 740-767.

Table 1
Demographics with missing analysis

	Inpatient sample				**Follow-up sample**			
	Included (*n* = 348)	Missing (*n* = 180)	Test Value	*p*	Included (*n* = 101)	Missing (*n* = 427)	Test value	*p*
Mean age (range)	41 (18-83)	40 (18-95)	0.65[c]	0.514	38 (20-75)	42 (18-95)	-1.8[c]	0.073
Male gender	156 (45%)	78 (43%)	0.11 (1)[d]	0.743	47 (47%)	187 (44%)	0.25 (1)[d]	0.618
Involuntarily admitted	153 (44%)	75 (42%)	0.26 (1)[d]	0.613	41 (41%)	181 (42%)	0.11 (1)[d]	0.742
Stay days mean/median	20 / 12	9.4 / 1.0	-8.2[e]	<0.001	22 / 16	14 / 3	-4.4[e]	<0.001
Unemployed[a,b]	221 (70%)	114 (70%)	0.002 (1)[d]	0.962	72 (74%)	263 (70%)	0.74 (1)[d]	0.389
Only primary school[a]	104 (33%)	47 (32%)	0.011 (1)[d]	0.916	31 (32%)	120 (33%)	0.019 (1)[d]	0.890
Not in relationship[a]	221 (70%)	114 (70%)	0.002 (1)[d]	0.962	71 (72%)	299 (74%)	0.058 (1)[d]	0.810
F10-19 substance abuse	51 (15%)	35 (19%)	2.0 (1)[d]	0.158	12 (12%)	78 (18%)	2.4 (1)[d]	0.125
F20-29 psychosis	102 (29%)	29 (16%)	11 (1)[d]	0.001	29 (29%)	99 (23%)	1.4 (1)[d]	0.244
F30-31 bipolar disorder	44 (13%)	17 (9.4%)	1.2 (1)[d]	0.276	13 (13%)	46 (11%)	0.36 (1)[d]	0.547
F32-39 depression	51 (15%)	15 (8.3%)	4.3 (1)[d]	0.037	21 (21%)	44 (10%)	8.3 (1)[d]	0.004
F40-49 anxiety	47 (14%)	23 (13%)	0.055 (1)[d]	0.815	11 (11%)	59 (14%)	0.61 (1)[d]	0.435
F60 personality disorders	27 (7.8%)	22 (12%)	2.8 (1)[d]	0.094	10 (9.9%)	41 (9.6%)	0.008 (1)[d]	0.927
Other or no diagnoses	26 (7.5%)	39 (22%)	22 (1)[d]	<0.001	5 (5.0%)	60 (14%)	6.3 (1)[d]	0.012

Note. Proportions or means / medians are calculated from the valid *n* for variables with missing cases.
[a]Missing cases (3.6 - 13% in the individual variables in the initial *N* = 528 sample)
[b]Not working (excluding age retirement)
[c]Independent samples *t*-test (*t*-value)
[d]Chi-square test, χ^2 (*df*)
[e]Mann-Whitney *U* test (*Z*-value)

Table 2
Baseline variables divided into violent and non-violent patients

	Inpatient sample (*N* = 348)				**Follow-up sample (*N* = 101)**			
	Violent	Non-violent	Test value	*p*	Violent	Non-violent	Test value	*p*
V-RISK-10	12 (11 - 13)	7.0 (6.5 - 7.5)	- 7.6[c]	<0.001	10 (8.5 - 12)	6.4 (5.4 - 7.4)	- 4.0[c]	<0.001
SRS = *risk*	32 (56%)	90 (31%)	13 (1)[d]	<0.001	20 (74%)	30 (41%)	8.9 (1)[d]	0.003
TC	4.8 (4.5 - 5.1)	5.0 (4.9 - 5.2)	1.2[c]	0.238	5.1 (4.6 - 5.6)	5.1 (4.8 - 5.4)	0.11[c]	0.914
HDL	1.4 (1.2 - 1.5)	1.5 (1.5 - 1.6)	2.1[c]	0.034	1.4 (1.2 - 1.6)	1.5 (1.4 - 1.6)	0.47[c]	0.642
Age	40 (36 - 44)	41 (40 - 43)	0.69[c]	0.491	38 (33 - 42)	39 (35 - 42)	0.32[c]	0.748
Male gender	38 (67%)	118 (41%)	13 (1)[d]	<0.001	14 (52%)	33 (45%)	0.42 (1)[d]	0.518
Involuntarily admitted	45 (79%)	108 (37%)	34 (1)[d]	<0.001	16 (59%)	25 (34%)	5.3 (1)[d]	0.021
Stay days mean/median	41/33	16/7	- 6.4[e]	<0.001	9/5	26/22	- 2.9[e]	0.003
Unemployed[a,b]	47 (90%)	174 (66%)	12 (1)[d]	<0.001	23 (89%)	49 (69%)	3.8 (1)[d]	0.052
Only primary school[a]	26 (49%)	78 (30%)	7.5 (1)[d]	0.006	9 (35%)	22 (31%)	0.12 (1)[d]	0.734
Not in relationship[a]	43 (80%)	200 (71%)	1.5 (1)[d]	0.215	21 (81%)	50 (69%)	1.2 (1)[d]	0.268
F10-19 substance abuse	12 (21%)	39 (13%)	2.2 (1)[d]	0.135	5 (19%)	7 (9.5%)	-[f]	0.295
F20-29 psychosis	27 (47%)	75 (26%)	11 (1)[d]	0.001	8 (30%)	21 (28%)	0.015 (1)[d]	0.902
F30-31 bipolar disorder	10 (18%)	34 (12%)	1.5 (1)[d]	0.223	3 (11%)	10 (14%)	-[f]	1.000
F32-39 depression	4 (7.0%)	47 (16%)	3.2 (1)[d]	0.075	0 (0.0%)	21 (28%)	9.7 (1)[d]	0.002
F40-49 anxiety	1 (1.8%)	46 (16%)	8.1 (1)[d]	0.005	3 (11%)	8 (11%)	-[f]	1.000
F60 personality disorders	2 (3.5%)	25 (8.6%)	-[f]	0.279	7 (26%)	3 (4.1%)	-[f]	0.003
Other or no diagnoses	1 (1.8%)	25 (8.6%)	-[f]	0.096	1 (3.7%)	4 (5.4%)	-[f]	1.000

Note. Means (95% CI) and / or medians for continuous variables and proportions for dichotomous variables in the violent and non-violent groups.
[a]Missing cases (3.0 - 9.5% in the individual variables)
[b]Not working (excluding age retirement)
[c]Independent samples *t*-test (*t*-value)
[d]Chi-square test, χ^2 (*df*)
[e]Mann-Whitney *U* test (*Z*-value)
[f]Fisher's exact test

Table 3

Univariate effect sizes of the components of the biopsychosocial model

	Inpatient sample (*N* = 348)		**Follow-up sample (*N* = 101)**	
	OR (95% CI)	*p*	OR (95% CI)	*p*
Whole sample				
V-RISK-10	1.3 (1.2 - 1.4)	<0.001	1.2 (1.1 - 1.3)	<0.001
SRS	2.9 (1.6 - 5.1)	<0.001	4.2 (1.6 - 11)	0.004
TC	0.86 (0.67 - 1.1)	0.238	0.98 (0.68 - 1.4)	0.913
HDL	0.49 (0.25 - 0.95)	0.035	0.79 (0.29 - 2.1)	0.639
Men				
V-RISK-10	1.2 (1.1 - 1.4)	<0.001	1.2 (1.0 - 1.4)	0.014
SRS	2.1 (1.0 - 4.4)	0.052	5.6 (1.1 - 29)	0.039
TC	0.96 (0.71 - 1.3)	0.792	0.87 (0.50 - 1.5)	0.609
HDL	0.34 (0.12 - 0.95)	0.039	0.11 (0.011 - 1.1)	0.063
Women				
V-RISK-10	1.3 (1.1 - 1.4)	<0.001	1.2 (1.0 - 1.4)	0.016
SRS	4.3 (1.6 - 12)	0.004	3.4 (0.94 - 13)	0.061
TC	0.81 (0.52 - 1.3)	0.353	1.1 (0.69 - 1.8)	0.628
HDL	1.3 (0.54 - 3.2)	0.553	2.1 (0.57 - 7.8)	0.266

Table 4
Contributions of SRS, TC and HDL to V-RISK-10 in bivariate analyses

	Inpatient sample				**Follow-up sample**			
	OR (95 % CI)	*p*	Increased variance[a] χ^2 (*df*) (%)	*p*	OR (95 % CI)	*p*	Increased variance[a] χ^2 (*df*) (%)	*p*
Whole sample								
SRS	1.4 (0.73 - 2.7)	0.306	1.0 (1) (0 - 1%)	0.308	2.7 (0.95 - 7.7)	0.063	3.6 (1) (3 - 4%)	0.059
TC	0.89 (0.68 - 1.2)	0.419	0.67 (1) (0 - 0%)	0.415	1.0 (0.69 - 1.5)	0.978	0.0 (1) (0 - 0%)	0.978
HDL	0.66 (0.32 - 1.4)	0.259	1.3 (1) (0 - 1%)	0.249	1.6 (0.50 - 4.9)	0.438	0.60 (1) (1 - 1%)	0.439
Men								
SRS	0.98 (0.41 - 2.3)	0.955	0.0 (1) (0 - 0%)	0.955	4.2 (0.76 - 23)	0.099	3.2 (1) (6 - 8%)	0.076
TC	0.97 (0.70 - 1.4)	0.878	0.024 (1) (0 - 0%)	0.878	0.84 (0.47 - 1.5)	0.556	0.36 (1) (1 - 1%)	0.549
HDL	0.27 (0.078 - 0.89)	0.032	5.2 (1) (3 - 4%)	0.022	0.18 (0.015 - 2.2)	0.178	2.1 (1) (4 - 5%)	0.151
Women								
SRS	2.5 (0.87 - 7.2)	0.088	3.0 (1) (1 - 3%)	0.086	2.1 (0.51 - 8.6)	0.308	1.0 (1) (2 - 3%)	0.309
TC	0.82 (0.50-1.3)	0.418	0.69 (1) (0 - 1%)	0.407	1.1 (0.68 - 1.9)	0.602	0.27 (1) (1 - 1%)	0.604
HDL	1.8 (0.71 - 4.8)	0.207	1.5 (1) (1 -2%)	0.214	4.3 (0.83 - 22)	0.082	3.5 (1) (6 - 8%)	0.062

Note. ORs are for each variable in bivariate analyses with V-RISK-10 as covariate. V-RISK-10 remained significant in all bivariate analyses with ORs of 1.2 - 1.3, except for women in the follow-up sample with SRS as covariate: OR = 1.2, 95 % CI = 1.0 - 1.4, $p = 0.053$.
[a]Increase from V-RISK-10 alone in total variance; χ^2 (*df*), and % increases in explained variances (lower = Cox & Snell R^2, upper = Nagelkerke R^2; estimates of the factors' contribution to the total variance).

Table 5A

Inpatient sample: Stepwise multivariate analyses of V-RISK-10, the biopsychosocial model and the biopsychosocial model controlled for other variables with a potential positive impact on violence

	OR (95% CI)	*p*	Total variance		Explained variance
			χ^2 (*df*)	*p*	
Whole sample (*N* = 348)					
Step 1: V-RISK-10 alone					
V-RISK-10	1.3 (1.2 – 1.4)	<0.001			
Variance of Step 1:			51 (1)	<0.001	14 - 23%
Step 2: the biopsychosocial model					
V-RISK-10	1.2 (1.2 - 1.4)	<0.001			
SRS	1.5 (0.75 - 2.8)	0.265			
TC	0.91 (0.69 - 1.2)	0.519			
HDL	0.68 (0.33 - 1.4)	0.303			
Increase from Step 1:			2.9 (3)	0.405	0 - 1%
Variance of Step 2:			54 (4)	<0.001	14 - 24%
Step 3: control for other variables					
V-RISK-10	1.2 (1.1 - 1.3)	<0.001			
SRS	1.4 (0.70 - 2.6)	0.362			
TC	0.93 (0.70 - 1.2)	0.639			
HDL	0.70 (0.33 - 1.5)	0.344			
Involuntarily admitted	2.5 (1.2 - 5.5)	0.018			
Age	1.0 (0.97 - 1.0)	0.750			
Increase from Step 2:			5.9 (2)	0.051	2 - 3%
Variance of Step 3:			60 (6)	<0.001	16 - 27%
Men (*n* = 156)					
Step 1: V-RISK-10 alone					
V-RISK-10	1.2 (1.1 - 1.4)	<0.001			
Variance of Step 1:			26 (1)	<0.001	15 - 23%
Step 2: the biopsychosocial model					
V-RISK-10	1.2 (1.1 - 1.4)	<0.001			
SRS	1.0 (0.42 - 2.5)	0.977			
TC	1.0 (0.74 - 1.4)	0.883			
HDL	0.26 (0.077 - 0.89)	0.032			
Increase from Step 1:			5.2 (3)	0.155	3 - 4%
Variance of Step 2:			31 (4)	<0.001	18 - 27%
Step 3: control for other variables					
V-RISK-10	1.2 (1.1 - 1.3)	0.003			
SRS	0.87 (0.35 - 2.2)	0.766			
TC	1.1 (0.77 - 1.6)	0.589			
HDL	0.25 (0.069 - 0.89)	0.032			
Involuntarily admitted	4.2 (1.5 - 12)	0.008			
Age	0.99 (0.96 - 1.0)	0.656			
Increase from Step 2:			7.8 (2)	0.020	4 - 6%
Variance of Step 3:			39 (6)	<0.001	22 - 33%

Women (*n* = 192)					
Step 1: V-RISK-10 alone					
V-RISK-10	1.3 (1.1 - 1.4)	<0.001			
Variance of Step 1:			17 (1)	<0.001	8 - 17%
Step 2: the biopsychosocial model					
V-RISK-10	1.3 (1.1 - 1.4)	0.002			
SRS	2.5 (0.85 - 7.4)	0.096			
TC	0.72 (0.42 - 1.2)	0.231			
HDL	2.0 (0.75 - 5.6)	0.163			
Increase from Step 1:			5.8 (3)	0.123	3 - 6%
Variance of Step 2:			22 (4)	<0.001	11 - 23%
Step 3: control for other variables					
V-RISK-10	1.2 (1.0 - 1.4)	0.023			
SRS	2.5 (0.86 - 7.6)	0.092			
TC	0.70 (0.41 - 1.2)	0.213			
HDL	2.1 (0.75 - 5.8)	0.156			
Involuntarily admitted	1.7 (0.49 - 5.8)	0.408			
Age	1.0 (0.97 - 1.0)	0.841			
Increase from Step 2:			0.72 (2)	0.697	0 - 1%
Variance of Step 3:			23 (6)	0.001	11 - 24%

Note. Explained variance = Cox & Snell R^2 – Nagelkerke R^2 (lower and upper estimates of the factors' contribution to the total variance).

Table 5B

Follow-up sample: Stepwise multivariate analyses of V-RISK-10, the biopsychosocial model and the biopsychosocial model controlled for other variables with a potential positive impact on violence

	OR (95% CI)	*P*	Total variance		Explained variance
			χ^2 (*df*)	*p*	
Whole sample (*N* = 101)					
Step 1: V-RISK-10 alone					
V-RISK-10	1.2 (1.1 - 1.3)	<0.001			
Variance of Step 1:			14 (1)	<0.001	13 - 19%
Step 2: the biopsychosocial model					
V-RISK-10	1.2 (1.1 - 1.3)	0.004			
SRS	3.1 (1.0 - 9.5)	0.043			
TC	1.1 (0.70 - 1.6)	0.809			
HDL	2.0 (0.58 - 6.7)	0.279			
Increase from Step 1:			4.9 (3)	0.176	4 - 6%
Variance of Step 2:			19 (4)	0.001	17 - 25%
Step 3: control for other variables					
V-RISK-10	1.2 (1.0 - 1.3)	0.011			
SRS	3.0 (0.99 - 9.3)	0.051			
TC	1.1 (0.68 - 1.7)	0.775			
HDL	2.2 (0.61 - 7.6)	0.237			
Involuntarily admitted	1.6 (0.55 - 4.7)	0.385			
Age	0.99 (0.95 - 1.0)	0.775			
Increase from Step 2:			0.77 (2)	0.682	1 - 1%
Variance of Step 3:			20 (6)	0.003	18 - 26%
Men (*n* = 47)					
Step 1: V-RISK-10 alone					
V-RISK-10	1.2 (1.0 -1.4)	0.014			
Variance of Step 1:			7.2 (1)	0.007	14 - 20%
Step 2: the biopsychosocial model					
V-RISK-10	1.2 (0.99 - 1.4)	0.067			
SRS	3.6 (0.64 - 21)	0.145			
TC	0.86 (0.46 - 1.6)	0.648			
HDL	0.24 (0.018 - 3.2)	0.280			
Increase from Step 1:			4.8 (3)	0.188	9 - 12%
Variance of Step 2:			12 (4)	0.018	23 - 32%
Step 3: control for other variables					
V-RISK-10	1.2 (0.98 - 1.4)	0.074			
SRS	3.2 (0.55 - 19)	0.192			
TC	0.82 (0.43 - 1.6)	0.544			
HDL	0.21 (0.012 - 3.5)	0.273			
Involuntarily admitted	1.6 (0.34 - 7.6)	0.549			
Age	1.0 (0.96-1.1)	0.418			

Increase from Step 2:			1.0 (2)	0.595	1 - 2%
Variance of Step 3:			13 (6)	0.043	24 - 34%
Women (*n* = 54)					
Step 1: V-RISK-10 alone					
V-RISK-10	1.2 (1.0 - 1.4)	0.016			
Variance of Step 1:			6.6 (1)	0.010	11 - 17%
Step 2: the biopsychosocial model					
V-RISK-10	1.2 (1.0 - 1.4)	0.029			
SRS	3.8 (0.65 - 23)	0.138			
TC	1.3 (0.68 - 2.5)	0.430			
HDL	5.1 (0.89 - 29)	0.067			
Increase from Step 1:			5.8 (3)	0.119	10 - 14%
Variance of Step 2:			12 (4)	0.015	21 - 31%
Step 3: control for other variables					
V-RISK-10	1.3 (1.0 - 1.5)	0.031			
SRS	3.1 (0.49 - 20)	0.228			
TC	1.5 (0.73 - 3.1)	0.262			
HDL	5.8 (1.0 - 34)	0.051			
Involuntarily admitted	1.3 (0.25 - 6.7)	0.759			
Age	0.97 (0.91 - 1.0)	0.301			
Increase from Step 2:			1.1 (2)	0.574	1 - 2%
Variance of Step 3:			14 (6)	0.036	22 - 33%

Note. Explained variance = Cox & Snell R^2 – Nagelkerke R^2 (lower and upper estimates of the factors' contribution to the total variance)

Table 6
AUC-values for V-RISK-10 and V-RISK-EXT

	Inpatient sample			**Follow-up sample**		
	n	AUC (95 % CI)	*p*	*n*	AUC (95 % CI)	*p*
Whole sample	348			101		
V-RISK-10		0.79 (0.73 - 0.85)	<0.001		0.73 (0.63 - 0.84)	<0.001
V-RISK-EXT		0.80 (0.74 - 0.85)	<0.001		0.74 (0.63 - 0.85)	<0.001
Men	156			47		
V-RISK-10		0.77 (0.70 - 0.85)	<0.001		0.74 (0.58 - 0.89)	0.011
V-RISK-EXT		0.79 (0.71 - 0.86)	<0.001		0.78 (0.64 - 0.91)	0.003
Women	192			54		
V-RISK-10		0.77 (0.65 - 0.89)	<0.001		0.73 (0.57 - 0.88)	0.014
V-RISK-EXT		0.77 (0.66 - 0.89)	<0.001		0.74 (0.58 - 0.89)	0.011

Table 7
Predictive values for V-RISK-10 and V-RISK-EXT

	Sensitivity	Specificity	PPV	NPV	NNA	LR+	LR-
Inpatient:							
Whole sample (n = 348)							
V-RISK-10[a]	0.86	0.58	0.29	0.96	3.5	2.1	0.24
V-RISK-EXT[b]	0.88	0.61	0.31	0.96	3.3	2.2	0.20
Men (n = 156)							
V-RISK-10[c]	0.87	0.53	0.38	0.93	2.7	1.9	0.25
V-RISK-EXT[d]	0.95	0.48	0.37	0.97	2.7	1.8	0.11
Women (n = 192)							
V-RISK-10[e]	0.84	0.68	0.23	0.98	4.4	2.6	0.23
V-RISK-EXT[f]	0.90	0.64	0.21	0.98	4.7	2.5	0.17
Follow-up:							
Whole sample (n = 101)							
V-RISK-10[g]	0.78	0.60	0.41	0.88	2.4	1.9	0.37
V-RISK-EXT[h]	0.70	0.70	0.46	0.87	2.2	2.4	0.42
Men (n = 47)							
V-RISK-10[i]	0.86	0.55	0.44	0.90	2.3	1.9	0.26
V-RISK-EXT[j]	0.86	0.61	0.48	0.91	2.1	2.2	0.24
Women (n = 54)							
V-RISK-10[k]	0.92	0.42	0.33	0.94	3.0	1.6	0.19
V-RISK-EXT[l]	0.85	0.59	0.39	0.92	2.5	2.0	0.26

Note. PPV = positive predictive value. NPV = negative predictive value. NNA = number needed to assess. LR+ = positive likelihood ratio. LR- = negative likelihood ratio.
Cut-off for a positive test ≥: [a]8 [b]10 [c]8 [d]9 [e]9 [f]9 [g]7 [h]11 [i]7 [j]11 [k]4 [l]6