Comorbidity in posttraumatic stress disorder: a structural equation modelling approach

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Abstract

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Comorbidity in PTSD: a structural equation modelling approach

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Abstract

Objective: Posttraumatic stress disorder (PTSD) is associated with high rates of psychiatric comorbidity. Existing theories consider comorbidity as a consequence of PTSD (model 1), PTSD and comorbidity as a consequence of shared factors of vulnerability (model 2), and comorbidity as a consequence of trauma-type specific mechanisms (model 3).

Method: To compare the explanatory value of these models, we assessed PTSD (model 1), sense of coherence (SOC, model 2) and satisfaction with health (SWH, model 3) and symptoms of anxiety and depression as indicators of comorbidity five days (t1) and six months (t2) post accident in 225 injured accident survivors. Structural equation models representing models 1-3 were tested separately and combined.

Results: Combined, models 1 and 3 explained 82% of the variance of comorbid symptoms at t2. PTSD and SWH (t2) exerted strong influences on comorbid symptoms.

Conclusion: Comorbidity beside PTSD is best described by an integration of competing explanatory models.

Key words: PTSD, Comorbidity, Structural equation models, Satisfaction with health, Sense of Coherence
Introduction

Posttraumatic stress disorder (PTSD) is associated with high rates of comorbid psychiatric disorders such as mood, anxiety, or substance abuse disorders [1]. Lifetime comorbidity in population-based surveys of PTSD ranges from 62% to 92% [1]. Comorbidity strongly influences quality of life [2] and is an important factor for differential treatment decisions and prognostic issues in PTSD patients [3, 4]. Four explanatory models (models A to D) have been proposed regarding the strong association between PTSD and comorbidity [5]. According to model A, comorbidity occurs “in reaction to, or as complications of, PTSD” (p. 478). This is in accordance with data from many studies reporting that comorbid disorders develop secondarily to PTSD or that further mental disorders are common in traumatized subjects with PTSD, but not in subjects exposed to traumatic events who do not develop PTSD [1, 6-10]. However, these data also support model B. Model B assumes that trauma leads to multiple disorders, implying that “the full spectrum of posttrauma psychiatric effects is not covered by the PTSD diagnosis” [5, p. 478]. Following Breslau [e.g., 7], a more general formulation of this model would be that PTSD and further psychopathological sequelae may share common vulnerability or risk factors (one of them being the experience of potentially traumatic events). PTSD would thus only be a marker of (the shared) vulnerability for comorbid disorders. According to model C, the frequent association of PTSD and at least some psychiatric diagnoses (mood, anxiety, and somatisation disorders) is a diagnostic artefact due to symptom overlap between these disorders and PTSD. However, research has shown that this assumption cannot sufficiently explain mood and anxiety disorders comorbid with PTSD [5, 11-13]. Finally, model D postulates that pre-existing psychopathology creates a vulnerability for PTSD. However, since pre-existing psychopathology has been observed in only about one third of PTSD cases [10], the common co-occurrence of PTSD and other disorders can not be satisfactorily explained by previous psychiatric history only. We suggest to subsume pre-existing psychopathology under model B as a factor of vulnerability shared by PTSD and other psychiatric disorders occurring after trauma.
In summary, the etiology of comorbidity in PTSD is posited by different competing models and the high comorbidity rates associated with PTSD are still poorly understood. Based on the above listed models, empirical findings, and clinical experience, we propose three models (which we will henceforth call models 1-3) to be tested and compared by further research. Research needs to examine the direct effects of PTSD on associated psychopathology (henceforth model 1) as well as the impact of potentially shared vulnerability factors (henceforth model 2). As one possibility of defining shared vulnerability, we chose the perceived coping resources as measured by Antonovsky’s Sense of Coherence [SOC, 14]. An association between SOC and PTSD symptoms has been observed in samples exposed to many different possibly traumatizing events [15-20]. For instance, Engelhard et al (2003) showed in a longitudinal study that SOC measured early in pregnancy predicted symptoms of PTSD and depression after eventual pregnancy loss. In a similar study [21], SOC was related to anxiety and depression during pregnancy. Since some evidence suggests that comorbidity profiles depend on the type of trauma [22], trauma type specific mechanisms should be examined as well. In accident survivors, clinical experience and research [e.g., 23, 24] suggest that injury related consequences such as disabilities or chronic pain can provoke psychopathological symptoms. Thus, in injured accident survivors, health impairment appears to be an adequate operationalisation of a trauma type specific mechanism of psychopathology. Subjectively assessed health appears to be more closely related to psychopathology than objective health status [25]. So health related satisfaction rather than physician based measures of injury severity was chosen in order to test a trauma type specific mechanism of psychopathology (henceforth model 3).

In this longitudinal study, we compared the explanatory value of these three explanation models (models 1-3) for psychiatric comorbid symptoms associated with posttraumatic stress in a sample of injured accident survivors assessed five days and six months post-accident. We tested the validity of the three explanations using structural equation models based on latent variables separately and by combining empirically supported models.
Material and Methods

Participants

This study was approved by the Institutional Review Board of the Canton of Zurich. During the 12 month recruitment period our research group was informed of all recent accident or assault admissions to the trauma ward of University Hospital of Zurich. Inclusion criteria were a minimum hospitalization of two nights, age between 18 and 65, and fluency in German, Italian, Spanish, Portuguese, Serbo-Croatian, Turkish, or Albanian. Patients were excluded if they were unable to participate in an extensive interview within 30 days of the accident or if they suffered from severe traumatic brain injury (Glasgow Coma Scale score (GCS) below nine, were unconscious for more than 15 minutes, had pathological findings in the cranial CT), or had injuries due to attempted suicide. During the recruitment period 787 patients within the required age range were reported to the interviewers. Of these, 253 patients did not fulfil the selection criteria, most due to early discharge (41.1%), poor clinical condition (29.2%), a GCS score below 9 (18.2%), language difficulties (8.3%), and other reasons (3.2%) (multiple reasons possible). However, given our research staff’s limited capacity, the great number of eligible patients required a further selection. The following system was applied to ensure the recruitment of a representative sample: based on daily lists of admissions sorted by admission time, every other consecutive patient was primarily interviewed. Patients were not interviewed until a minimum stay of 32 hours. To control for a potential bias attributable to the time of day of admission, on day one the first patient on the respective list was interviewed first; on day two the last patient on the list was the first to be interviewed, and so forth. Thus, 335 patients were included in the first assessment, whereas 148 were not approached due to limited interviewing capacity. The non-contacted patients did not differ from the participating patients with regard to gender (Pearson’s $\chi^2=0.8$, df=1, p=.38), age (t=0.31, df=481, p=.75), or time of referral to the University Hospital (t=0.60, df=467, p=.55). Limitation of the sample to accident survivors
resulted in the exclusion of 12 assault victims. Being exposed to, and recovering from interpersonal trauma such as assault, is psychologically different from experiencing an accident and its sequelae [Wittmann, Moergeli, and Schnyder, 26]. Therefore, assault victims should be studied separately from patients who sustained accidental injuries. Of the remaining 323 subjects, 68 (21.1%) declined further participation or could no longer be located. Of the 255 subjects (79.0%) completing the follow-up assessment at six months, thirty patients (11.8%) had to be excluded from the current analysis due to missing data in the self-rating questionnaires which left us with a final sample of 225 patients. The drop-outs did not differ significantly from the final sample with regard to accident-related variables, acute stress disorder (ASD) symptom level, or depression (Hospital Anxiety and Depression Scale) mean scores. However, they had lower Sense of Coherence mean scores (t=-2.41, df=103.6, p<.05, unequal variances) and higher anxiety (Hospital Anxiety and Depression Scale) mean scores (t=2.42, df=108.0, p<.05, unequal variances) than completers. Furthermore, they were more often non-Swiss citizens (Pearson’s $\chi^2$=6.9, df=1, p<.01), unemployed (Pearson’s $\chi^2$=9.3, df=1, p<.01), and had a lower educational level (Mann-Whitney U=9451.5, p<.05).

**Procedure**

Two staff members of the trauma ward of the University Hospital of Zurich conducted the initial screening. They reported all potential participants to the two study physicians who made the final selection. Patients were contacted as soon as possible after admission. After obtaining the patients’ written informed consent, the first assessment (t1) was performed on average 5.1 days ($SD = 4.0$) after the accident in the patients’ hospital room by one of the two study physicians. The second assessment (t2) was performed on average 188.7 days (SD 16.0) after the accident, either in the hospital or at the patients’ home. Assessments included an extensive structured interview and a self-rating questionnaire. Interviews were conducted based on German versions of the assessment tools, translated by interpreters during the interviews, and/or professionally
translated versions of the self-rating instruments for the 26 patients (11.6%) who did not speak German fluently. Study physicians were trained and supervised by the last author (U.S.). After the training period, study physicians independently assessed the PTSD status and symptom level of 20 accident victims. They obtained a good agreement [27] for PTSD diagnosis (no vs. sub-syndromal vs. full-blown PTSD, $\kappa = .61, p < .001$) and excellent correlations between Clinician Administered PTSD Scale (CAPS) total scores ($r = .93, p < .001$), CAPS reexperiencing ($r = .91, p < .001$), avoidance ($r = .95, p < .001$), and hyperarousal scores ($r = .82, p < .001$). Each patient was assessed by the same of the two study physician at t1 and t2.

**Measures**

*Hospital Anxiety and Depression Scale (HADS, Zigmond & Snaith [28])*  

Symptoms of depression and anxiety were used as indicators of comorbid symptoms of mood and anxiety disorders which constitute the most common comorbidity in non-combat-related PTSD [22]. To assess post-accident state anxiety and depression, we applied the validated German version of the Hospital Anxiety and Depression Scale [29]. The HADS is a 14-item self-rating questionnaire developed by Zigmond and Snaith in 1983 to provide clinicians and scientists with a reliable, valid, and practical tool for identifying and quantifying the most common forms of psychological disturbances in medical patients. Its reliability and validity have been proven in hundreds of research projects as has its sensitivity to change during the course of diseases [30, 31]. Scores in the depression and anxiety subscales (ranges 0-21) are computed by summing the respective seven items from each subscale (0-3 points each). The HADS discriminates well between samples with high ($>10$ points), medium (8-10 points), and low ($<8$ points) prevalences of anxiety or depressive disorders [31].

Assessment of ASD and PTSD
Clinician-Administered PTSD Scale (CAPS, Blake, Weathers, Nagy, Kapoulek, Charney, & Keane [32])

To assess posttraumatic psychological symptoms, the Clinician-Administered PTSD Scale (validated German version, Schnyder & Moergeli, [33]) was used. This instrument allows quantification of the frequency and intensity of each of the 17 PTSD symptoms according to the DSM-IV. A diagnosis of PTSD can also be made based on CAPS scores. Instructions for the clinician and interview procedures were identical to the original English version (Blake et al., 1998). For assessment of the stressor criterion A1/2 see below.

Peritraumatic Dissociative Experiences Questionnaire, rater version (PDEQ, Marmar, Weiss, & Metzler, [34])

The PDEQ rater version is a 10-item interview for the assessment of peritraumatic dissociation. It has good psychometric properties [34]. A PDEQ score (range 0-10) was calculated from the 10 items.

Since no validated assessment tool for ASD was available at the time of this study, symptom criteria for ASD diagnosis were assessed using a combination of items from the CAPS and the PDEQ. By this procedure an ASD score was obtained with a maximum range from 0 to 17 which showed a satisfactory internal consistency [Cronbach's alpha =.68; for further details see 35]. Since all patients included in the analysis were accident victims confronted with an event threatening their physical integrity, all participants were considered to fulfil the objective stressor criterion (A1). The subjective stressor criterion (A2) was captured by items that assessed the patients’ possible sense of death threat during the accident and their appraisal of accident severity (Table 1). Subjects reporting a sense of death threat during the accident and/or assessing the accident as severe or very severe were considered to fulfil the A2-criterion.
Sense of Coherence Questionnaire (SOC, Antonovsky [14])

The Sense of Coherence questionnaire [14] is a measure of an individual’s resilience to stress and his or her capacity to cope with it. Individuals with high SOC scores are likely to perceive stressors as predictable and explicable, have confidence in their capacity to overcome stressors, and judge it worthwhile to rise to the challenges they face. Test properties such as test-retest reliability and internal consistency of the Sense of Coherence questionnaire scale are excellent [36]. In order to avoid a bias due to the current situation, subjects were asked to complete this questionnaire as they would have done before the accident.

Satisfaction with Health (SWH): Questions on Life Satisfaction FLZ [37]

The subscale „Satisfaction with Health“ of the self-rating instrument Questions on Life Satisfaction [37] was chosen to measure subjectively assessed health. Here, patients are asked to rate the subjective importance of eight aspects of health as well as their degree of satisfaction in each of these aspects. The two ratings are combined into a „weighted satisfaction“ score and then added up for a total score. The scale covers the following dimensions: physical fitness, ability to relax / equilibrium, energy / zest for life, mobility (e.g., walking, driving), eyesight and hearing, freedom from anxiety, freedom from complaints and pain, independence from assistance / nursing. Test properties are very good (Cronbach's alpha = .89, test-retest reliability = .85) and normative data from a representative sample of the German general population are available. We did not include the dimensions of ability to relax / equilibrium, energy / zest for life, and freedom from anxiety in the testing of the described models in order to avoid overlap with symptoms of depression or anxiety. At t1, subjects were asked to assess their pre-accident rather than their current SWH.

Primary Care Evaluation of Mental Disorders (Prime-MD, Spitzer et al., [38])
Pre-accident mental disorders at t1 (retrospectively assessed) and presence of disorders at t2 were assessed using Prime-MD, a screening instrument for recognition and diagnosis of the mental disorders most commonly seen in adults in primary care settings: mood disorders, anxiety, alcohol abuse, eating and somatoform disorders. This instrument proved to have good sensitivity and specificity as well as a high agreement with the independent assessments of mental health professionals [38].

**Sociodemographic and accident-related variables**

Patients’ sociodemographic characteristics (age, gender, nationality, partnership status, educational level, employment status) were also assessed during the interview. In addition, patients were asked what type of accident they had sustained (traffic accident, work related accident, household accident, sports or leisure time accident), whether they had felt during the accident that it was life threatening, and how severe they rated the accident (5-point Likert scale, 1 = *very slight*, 5 = *very severe*). To assess the immediate physical consequences of the accident, the Injury Severity Score (ISS, Baker, O’Neill, Haddon, & Long, [39]; Baker & O’Neill, [40]) and the Glasgow Coma Scale (GCS, Teasdale & Jennett, [41]) were used. Severely injured patients usually score above 9 on the ISS; patients with severe traumatic brain injury generally have a score below 9 on the GCS.

**Analyses**

Basic statistical analyses were calculated using SPSS 12.0.1. In order to test if potential symptom overlap was responsible for associations between measures, we compared patterns of correlations for several subscales. We tested if symptoms of anxiety and depression (HADS) and SWH were more closely related to avoidance or hyper-arousal than to re-experiencing (CAPS). For testing of the explanatory models of comorbidity in PTSD, we used structural equation models with latent variables which were specified using LISREL 8.72. Not meeting the
assumption of multivariate normality (PRELIS: $\chi^2$-value for skewness and kurtosis = 554.8, p < .001), our data were transformed into normal (z-) scores according to Jöreskog, Sörbom, du Toit and du Toit [42], which resulted in a distribution nearly meeting multivariate normality (PRELIS: $\chi^2$-value for skewness and kurtosis = 7.0, p = .03). Correlations between latent variables were virtually not affected by this procedure. We applied Maximum Likelihood for parameter estimation since this method is quite robust with violations of the normality assumption [43]. For the latent criterion variable comorbidity, three indicator variables were built from the HADS. Each of the indicator variables comprised items of anxiety and depressive symptoms. Thus, the latent variable comorbidity reflects symptoms of anxiety and depression continuously rather than presence or absence of a DSM-IV diagnosis. Whereas the term comorbidity typically applies to co-occurring disorders, continuous measures are more appropriate when modelling relationships between variables [44]. For the further latent variables SOC, SWH (t1/2), and ASD/PTSD, two indicator variables were built by constructing test halves of the respective scales. For instance, the two indicator variables of the latent variable sense of coherence consist of six, respectively, seven items of the SOC-questionnaire. In the specific case of the latent variable ASD, the two indicator variables each consisted of test halves of the CAPS and PDEQ items considered for ASD assessment. For evaluation of model fit, Schermelleh-Engel et al. [43] have recommended considering not only $\chi^2$ test statistics and $\chi^2$/df ratio ($0 \leq \chi^2$/df $\leq 2$ indicates a good, $2 \leq \chi^2$/df $\leq 3$ indicates an acceptable fit) as these measures are sensitive to sample size. This is especially critical for $\chi^2$ test statistics which indicate statistically significant differences between model-implied and empirical covariance matrix in almost all empirical investigations based on large sample sizes. Therefore, they should be complemented by descriptive goodness-of-fit measures. As descriptive measures of overall model fit, Schermelleh-Engel et al. [43] recommended evaluating the Root Mean Square Error of Approximation (RMSEA; $0 \leq$ RMSEA $\leq .05$ for good and $.05 \leq$ RMSEA $\leq .08$ for acceptable fit) and the Standardized Root Mean Square Residual (SRMR; $0 \leq$ SRMR $\leq .05$ for good and $.05 \leq$...

11
SRMR ≤ .10 for acceptable fit). As descriptive measures which compare the fit of the model of interest to the fit of some baseline model (e.g., the independence or null model), Schermelleh-Engel et al. [43] proposed evaluating the Nonnormed Fit Index and the Comparative Fit Index (NNFI / CFI; .97 ≤ NNFI / CFI ≤ 1.0 for good and .95 ≤ NNFI / CFI ≤ .97 for acceptable fit). In order to control if the construction of measurement models was successful, all models were first run allowing the latent variables to correlate freely. Then, the three explanatory models of comorbidity were tested separately. In a next step, the empirically supported models were combined in an integrative model in order to allow for a multivariate comparison of different predictors. Except for SOC, which is considered a trait variable, all latent variables were measured at both time points. SOC and SWH at t1 were measured retrospectively, reflecting the subjects’ appraisal before the accident. All models considered comorbidity as a dependent variable. Thus, no influences of the latent variables comorbidity at t1 and t2 onto other latent variables were allowed except for comorbidity at t1 influencing comorbidity at t2. If the directions of influence between two latent variables were not determined by one of the three underlying theories or measurement time, correlations rather than directed influences were permitted.

Results

Sample and accident characteristics

Of the 225 patients, 149 (66.2%) were males; mean age was 41.4 years (SD = 12.6). Further sociodemographic data and accident-related variables are presented in Table 1. It was reported by 33 patients (14.7%) that they had experienced the accident as life threatening. Mean estimation of accident severity was 3.5 (SD = 0.9) on a five point Likert scale. A sense of death threat during the accident and/or assessment of the accident as severe or very severe was reported by 122 patients (54.2%) who were therefore considered to fulfil the subjective stressor criterion (A2). Mean Injury Severity Score was 11.4 (SD 9.6). Three patients (1.3%) had a GCS
score between nine and eleven, sixteen patients (7.1%) had a GCS score of 13 or 14, the remainder had the maximum GCS score of 15. According to Prime-MD, 39 (17.3%) of the patients suffered from one or multiple mental disorders prior to the accident. Sixteen patients (7.1%) received a diagnosis of a mood disorder, twelve (5.3%) a diagnosis of an anxiety disorder. Rates of alcohol-related and somatoform disorders were 6.7% and 4.9%, respectively. SOC and SWH mean scores before the accident (retrospectively assessed at t1) were 67.2 (SD 11.2) and 88.7 (SD 36.8), respectively (see Table 2).

Posttraumatic psychiatric morbidity

The results of the psychological assessments at t1 and t2 are summarized in Table 2. Internal consistencies (Cronbach’s alpha) for the applied scales ranged from .60 to .94. Internal consistencies for the assessments based on translated instruments deviated only slightly from reliability of non-translated assessments (Tab. 2). For all three instruments assessing aspects of psychopathology (HADS, CAPS, and PDEQ), mean scores were in the low range. At t1, cut-offs for possible and probable anxiety were met by 24 (10.7%) and 13 (5.8%) subjects, respectively (t2: 20 (8.9%) and 20 (8.9%)). Cut-offs for possible and probable depression were met at t1 by 20 (8.9%) and 11 (4.9%) patients, respectively (t2: 16 (7.1%) and 12 (5.3%)). Neither the increase from 37 to 40 anxiety (probable and possible) diagnoses during the first half year after the accident (McNemar test, exact $p = .74$), nor the decrease from 31 to 28 depression diagnoses (McNemar test, exact $p = .73$) reached statistical significance. A highly significant association between the two measurement points was found regarding diagnoses (possible or probable anxiety disorder / depression vs. no anxiety disorder / depression, Fisher’s exact test, both $p < .001$). Diagnoses of anxiety and depression were highly associated at t1 (Pearson’s $\chi^2=52.6$, df=1, $p<.001$) as well as at t2 (Pearson’s $\chi^2=90.6$, df=1, $p<.001$). Anxiety diagnoses according to HADS at t1 were associated with pre-existing anxiety diagnoses according to Prime-MD (Pearson’s $\chi^2=5.9$, df=1, $p<.05$). This was also the case for diagnoses of depression according to
the HADS and pre-existing mood disorders (Prime-MD) (Pearson’s $\chi^2=8.2$, df=1, $p<.01$). At t1, seven patients (3.1%) met all criteria (A-E) for ASD. A further 23 (10.2%) met criterion A and three out of the four ASD clusters B to E and were thus diagnosed with subsyndromal ASD [35]. At t2, six patients (2.7%) met all criteria (A-D) for PTSD. Sixteen (7.1%) met criteria A and B plus either C or D, but not both, and were thus diagnosed with subsyndromal PTSD [45]. The decrease from 30 ASD diagnoses to 22 PTSD diagnoses (both full and subsyndromal) during the first half year after the accident did not reach statistical significance (McNemar test, exact $p = .22$). A highly significant association between the two measurement points was found regarding diagnoses (subsyndromal or full-blown vs no ASD/PTSD, Fisher’s exact test, $p < .001$). Rates of comorbidity with PTSD (full or subsyndromal) were 59.1% for anxiety and 45.5% for depression (possible or probable diagnosis according to the HADS). The decrease in satisfaction with health from 88.7 (SD 36.8) pre-accident to 69.2 (SD 41.2) at six months post-accident was highly significant ($t=7.18$, df=224, $p<.001$). Finally, correlations of subscales did not support the hypothesis of symptom overlap being responsible for associations of measures. For instance, no substantial differences were found between correlations of anxiety (HADS) with the PTSD clusters re-experiencing, avoidance, and hyper-arousal ($r= .53$, .55, and .62, respectively).

**Structural equation models**

All models showed good fit indices when the latent variables were allowed to correlate freely. Thus, all measurement models can be considered as accurate.

**Model 1: Prediction of comorbidity (t2) by PTSD**

Model 1 explained 78% of the variance in comorbidity six month post-accident. All path coefficients depicted in Fig. 1 were statistically significant. At both time points, ASD and PTSD exerted a strong influence on the respective comorbidity. Comorbidity at t1 was strongly related to comorbidity at t2. A comparable association was found for ASD and PTSD. In this model,
ASD served as a negative suppressor variable [46] for PTSD, i.e., it removed variance of the variable PTSD irrelevant for the prediction of comorbidity. By this statistical suppression effect, the regression weight becomes opposite in sign to what is to be expected. The bivariate correlation between the latent variables ASD and comorbidity at t2 would be r=.48. Model parameters indicated acceptable to good model fit.

Fig. 1 about here

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Model 2: Explanation of the association between comorbidity (t2) and PTSD by pre-accident sense of coherence

Model 2 postulates that the association between comorbidity and PTSD is explained by common variance of both variables explained by sense of coherence. Thus, it was tested if the strength of the association between both measures (Fig. 1) is reduced if controlled for SOC. All path coefficients depicted in Fig. 2 were statistically significant. Higher SOC scores were associated with slightly lower psychopathological symptom scores immediately as well as six months after the accident. Compared to model 1 the reduction of the association between ASD/PTSD on the one and comorbidity at t1/t2 on the other hand was negligible. Parameters indicated an acceptable to good model fit.

Fig. 2 about here

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Model 3: Prediction of comorbidity (t2) by satisfaction with health

Model 3 explained 71% of the variance in comorbidity at t2. All path coefficients depicted in Fig. 3 were statistically significant. Pre-accident satisfaction with health exerted only a minor
influence on comorbidity at t1. Whereas the bivariate correlation between pre-accident SWH and
comorbidity at t2 would be \( r = -.27 \), pre-accident SWH served as a negative suppressor variable
for SWH at t2 with respect to comorbidity at t2. SWH at t2 played an important role, explaining
36% of the variance in comorbidity at t2. Model fit ranged from weak (RMSEA, SRMR) to
acceptable (\( \chi^2/df \) ratio, NNFI) to good (CFI).

Fig. 3 about here

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Selection of single models for an integrative model

Model 1 was characterized by a satisfying fit between model-implied and empirical covariance
matrix. Augmented by the negative suppression effect of ASD, PTSD proved to be the strongest
direct predictor of comorbidity at t2. Thus, model 1 clearly qualified for inclusion into an
integrative model. As predicted, SOC significantly contributed to all other variables considered
in model 2, although most associations were rather weak. However, as these associations did
virtually not affect the association between PTSD and comorbidity, model 2 was excluded from
the integrative model. Finally, model 3 reached almost the same variance explanation for
comorbidity at t2 as did model 1. Although its model fit did not reach the quality of model 1, it
was included into the integrative model, as those indices indicating a weak model fit (RMSEA,
SRMR) deviated only minimally from a satisfying model fit.

Integrative model

For simplification, Figure 4 depicts only the structural model of the integrative model combining
models 1 and 3. Measurement models of the latent variables were comparable to those shown in
Fig. 1 and 3. As the relationship between PTSD and SWH (t2) was not specified by any of the
underlying theories, a correlation between these variables rather than a directed influence was
permitted. Not considering the non-significant influence of SWH (t1), the integrative model explained 82% of the variance in comorbidity half a year post-accident. The most important direct predictor was PTSD, contributing 31% variance explanation. While its direct influence is obscured due to the negative suppression effect, ASD exerted a strong indirect influence on comorbidity at t2 via PTSD, satisfaction with health (t2) and comorbidity (t1). In contrast to pre-accident satisfaction with health, which was not significantly related to comorbidity, SWH at t2 significantly explained 12% of the variance in comorbidity at t2. Moderate relationships were observed between ASD/PTSD and SWH six months post accident. Most fit indices proved excellent fit between the empirical and model-implied covariance matrix.

Discussion

The aim of this study was to compare different theoretical explanations of comorbidity in PTSD with structural equation modelling. The presented analyses are based on a large, homogenous sample of accident survivors whose physical injuries required a hospitalization of at least two nights. A substantial amount (82%) of the variance in comorbidity six months post accident was explained by a structural equation model integrating two different explanations of comorbidity supported by the available literature. PTSD and satisfaction with health exerted strong direct influences onto comorbidity, whereas ASD made important indirect contributions. The following discussion elaborates the contribution of our results to the understanding of comorbidity in PTSD and points out directions for future research.

The sense of coherence mean score was at the upper end of the range reported in the literature [47] which is not surprising as many other studies have investigated psychiatric patients who
typically show low sense of coherence scores. With less than 20% of patients diagnosed with a mental disorder, we also found rather low rates of pre-accident psychiatric morbidity in our sample. Mean pre-accident Satisfaction With Health score conformed to norm values of the western German population [37]. In line with our expectations, satisfaction with health decreased significantly over the observation period.

A rather low proportion of our subjects (between 12 and 18%) received a possible or probable diagnosis of anxiety or depression. The low rate of ASD and PTSD replicates earlier findings of our research group [48] and has been extensively discussed elsewhere [49]. Rates of anxiety and depressive disorders comorbid with PTSD were slightly lower than reported in the literature [1].

Explaining 31% of variance, PTSD proved to be the strongest single predictor of further psychiatric disorders six months post accident. Nevertheless, considering that model 1 (comorbidity as a consequence of PTSD) is more important than the other models appears to be premature for several reasons. First, the indirect effects of ASD on comorbidity at t2 render this variable an equally important predictor of comorbidity at t2 in the integrative model, although its direct influence is obscured by the mentioned suppression effect. Thus, early symptoms of traumatic stress not only predicted chronic PTSD [49], but ASD seemed to play an important role in long-term post-traumatic psychiatric morbidity in general. Second, model 3 (comorbidity as a consequence of reduced satisfaction with health) almost reached the same level of explanation of variance as did model 1. As shown in Fig. 4, pre-accident satisfaction with health did not influence comorbidity (neither at t1 nor at t2). However, satisfaction with health at t2 appeared to account for a substantial part of anxiety and depression half a year post-accident. Therefore, trauma type specific mechanisms of psychopathological sequelae need to be considered when trying to explain the comorbidity of PTSD. In the integrative model, the explanatory value of PTSD was reduced by 25% as compared to model 1. Thus, non-
consideration of alternative explanations leads to an overestimation of the considered pathological mechanisms. Despite some influence of sense of coherence on ASD and comorbidity at t1, model 2 (PTSD and comorbidity share common vulnerability) did not reach the impact of model 1 or 3. However, it has to be taken into account that sense of coherence is only one among many other possible vulnerability factors shared by PTSD and comorbidity. Thus, only the chosen operationalisation, but not model 3 itself seemed to be less relevant for our sample. Other possible measures are shared genetic or environmental factors, or personality traits such as neuroticism. Substituting sense of coherence for a variable more strongly related to the long-term consequences such as PTSD and comorbidity at t2 might result in a reduction of the explanatory value of PTSD and satisfaction with health.

When combining models 1 and 3, an impressive variance explanation of 82% for comorbidity was achieved. Accordingly, the literature based selection of theories of comorbidity in PTSD appears to be valid. Of course, the stability of comorbidity over time implies that an essential part of variance explanation for comorbidity at t2 in all models is contributed by comorbidity at t1 (34% for the integrative model).

The following limitations should be taken into account in interpreting our results. Of subjects assessed at t1, 30.4% were lost to follow-up or had to be excluded due to missing data in the self-rating instruments. Comparison of drop-outs with completers indicated that we lost a subgroup with poorer psychosocial resources which does hamper the generalizibility of our results. To include subjects without sufficient command of the German language, professionally translated instruments were applied in 11.6% of our participants. While a strong influence on the reliability of these assessments can be ruled out, testing for influences on the validity of the respective assessments was not possible. However, we consider the inclusion of subjects without sufficient command of the locally spoken language as an essential aspect of the representativity of our
sample as currently more than 20% of Switzerland’s population are non-Swiss citizens [50].

Another limitation was that only ASD and PTSD diagnostics relied on clinician administered interviews while the other scores were obtained using self-rating instruments. However, a meaningful analysis of the respective scores is possible on the basis of available norm values and of the extensive experience from international (Hospital Anxiety and Depression Scale, Sense of Coherence questionnaire) scientific studies and those conducted in the German language (Satisfaction with Health questionnaire). As sense of coherence and satisfaction with health were assessed retrospectively at t1, a bias reflecting the current condition immediately after the accident on the scores representing the pre-accident constructs cannot be ruled out. Finally, comparison with other studies on trauma survivors are complicated by the low incidence of post-accident psychopathology in our sample.

Future research should test different operationalisations for vulnerability factors shared by PTSD and comorbidity. The suggested integrative model combining ASD/PTSD and trauma type specific mechanisms should be tested in different populations of traumatized subjects.

Satisfaction with health would have to be replaced by variables chosen according to the characteristics of the respective trauma type. Especially, traumatic events causing a higher incidence of ASD/PTSD should be considered. It might be advantageous to conduct more frequent assessments during the first half year after the traumatic event as the exact time course of symptoms of traumatic stress and those of comorbidity might allow for a refinement of the considered theories. Finally, other types of comorbidity should be considered. For instance, one might ask if the moderate association between ASD/PTSD and satisfaction with health found in our sample is a direct one or if it is mediated by somatoform disorders. In conclusion, our results suggest replacing individual, competing explanatory models of comorbidity in PTSD (models 1-3) by refining the multivariate approach of the integrative model. If confirmed by further
research, these results will be important for the treatment of PTSD as comorbidity has a strong impact on the patients’ quality of life as well as for differential treatment indication.
References


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Table 2: Psychological assessments of 225 accident survivors five days post accident (t1) and six months later (t2)

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CAPS = Clinician Administered PTSD Scale; PDEQ = Peritraumatic Dissociative Experiences Questionnaire; HADS = Hospital Anxiety and Depression Scale; SOC = Sense of Coherence; SWH = Satisfaction with Health; α<sup>a</sup> = Cronbach’s α for German assessments; α<sup>b</sup> = Cronbach’s α for translated assessment
Figures

Fig. 1: Model 1: Prediction of comorbidity (t2) by PTSD

COM=Comorbidity; ASD=Acute Stress Disorder; PTSD=Posttraumatic Stress Disorder; HADS=Hospital Anxiety and Depression Scale; CAPS=Clinician Administered PTSD Scale; Indices A to C refer to the item parcels of the respective scale (compare section Analyses); df=degrees of freedom; RMSEA=Root Mean Square Error of Approximation; SRMR=Standardized Root Mean Square Residual; NNFI=Nonnormed Fit Index; CFI=Comparative Fit Index

$\chi^2=64.2, \text{df}=30, \chi^2/\text{df}=2.1, p<.001, \text{RMSEA}=0.07, \text{SRMR}=0.06, \text{NNFI}=0.98, \text{CFI}=0.99$
**Fig. 2:** Model 2: Association of comorbidity (t2) and PTSD (t2) controlled for pre-accident Sense of Coherence

COM=Comorbidity; ASD=Acute Stress Disorder; PTSD=Posttraumatic Stress Disorder; SOC=Sense of Coherence; HADS=Hospital Anxiety and Depression Scale; CAPS=Clinician Administered PTSD Scale; Indices A to C refer to the item parcels of the respective scale (compare section Analyses); df=degrees of freedom; RMSEA=Root Mean Square Error of Approximation; SRMR=Standardized Root Mean Square Residual; NNFI=Nonnormed Fit Index; CFI=Comparative Fit Index

χ²=79.0, df=45, χ²/df=1.8, p<.01, RMSEA=0.06, SRMR=0.05, NNFI=0.98, CFI=0.99
Fig. 3: Model 3: Prediction of comorbidity (t2) by Satisfaction with Health

COM=Comorbidity; SWH=Satisfaction with Health; SOC=Sense of Coherence; HADS=Hospital Anxiety and Depression Scale; CAPS=Clinician Administered PTSD Scale; Indices A to C refer to the item parcels of the respective scale (compare section Analyses); df=degrees of freedom; RMSEA=Root Mean Square Error of Approximation; SRMR=Standardized Root Mean Square Residual; NNFI=Nonnormed Fit Index; CFI=Comparative Fit Index

χ²=82.5, df=30, χ²/df=2.8, p<.001, RMSEA=0.09, SRMR=0.12, NNFI=0.96, CFI=.97
Fig. 4: Model 4: Integration of models 1 and 3

COM=Comorbidity; ASD=Acute Stress Disorder; PTSD=Posttraumatic Stress Disorder; SWH=Satisfaction with health; HADS=Hospital Anxiety and Depression Scale; CAPS=Clinician Administered PTSD Scale; Indices A to C refer to the item parcels of the respective scale (compare section Analyses); (ns)=non significant; df=degrees of freedom; RMSEA=Root Mean Square Error of Approximation; SRMR=Standardized Root Mean Square Residual; NNFI=Nonnormed Fit Index; CFI=Comparative Fit Index

χ²=121.5, df=64, χ²/df=1.9, p<.001, RMSEA=0.06, SRMR=0.06, NNFI=0.97, CFI=.98

**Table and Graph Content**

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**Correlation Matrix**

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