Faktori rizika i kroničnog tijeka posttraumatskog stresnog poremećaja: pregled suvremenih spoznaja

/ Posttraumatic Stress Disorder Risk and Chronic Course Factors: a Review of Current Findings

Doris Čuržik

Sveučilište u Zagrebu, Filozofski fakultet, Odsjek za psihologiju, Katedra za kliničku i zdravstvenu psihologiju, Zagreb, Hrvatska

/University of Zagreb, Faculty of Humanities and Social Sciences, Department of Psychology, Chair of Clinical and Health Psychology, Zagreb, Croatia

https://orcid.org/0000-0002-5166-1250

Posttraumatski stresni poremećaj je produljena/odgođena reakcija na proživljeni traumatski događaj, koja se očituje psihološkim posljedicama poput izbjegavanja podsjetnika na traumu, ponovnog proživljavanja traumatskog događaja te pojačane pobudljivosti organizma. Desetljeća istraživanja ovog poremećaja rezultirala su proširivanjem spoznaja identifikacijom različitih faktora rizika, zaštitnih faktora te odrednica i korelata akutnog i kroničnog tijeka. Cilj ovog rada pružiti je što obuhvatniji pregled suvremenih spoznaja bioloških, psiholoških i socijalnih faktora rizika i korelata poremećaja kao i odrednica njegovog akutnog i kroničnog tijeka. Nalazi istraživanja ovog područja dotiču područja bioloških, psiholoških te socijalnih faktora u podlozi razvoja ovog poremećaja te ukazuju na potrebu za integrativnim pristupom razumijevanju njegova nastanka. Spoznaje nedovoljno istraženoga, no veoma relevantnoga područja socioekonomskih faktora rizika, također su prikazane. Zaključci mnogih istraživanja ovog područja, posebice njegovog kroničnog tijeka, odnose se na zapreke u smislu poteškoća razlučivanja čimbenika koji su premorbidni faktori od onih koji su posljedica samog tijeka poremećaja.

/ *Posttraumatic stress disorder (PTSD) presents a prolonged/delayed reaction to a traumatized event that manifests itself in psychological consequences such as avoiding traumatic reminders, re-experiencing a traumatic event, and intensifying body arousal. Decades of research of this disorder resulted in the expansion of knowledge through the identification of various risk and protective factors, and the determinants and correlates of its acute and chronic course. The aim of this paper is to provide a more comprehensive overview of the current discoveries of biological, psychological, and social factors of risk and correlates of the disorder as well as the determinants of its acute and chronic course. Research findings in this area address the areas of biological, psychological, and social factors underlying the development of this disorder and suggest the need for an integrative approach to understanding its origin. The findings from the understudied, but highly-relevant field of socio-economic risk factors are also presented. The conclusions of a lot of research in this area, particularly its chronic course, are related to obstacles in terms of the difficulty of distinguishing factors that represent premorbid factors than those resulting from the course of the disturbance itself.*

ADRESA ZA DOPISIVANJE / *ADDRESS FOR CORRESPONDENCE*:
Dr. sc. Doris Čuržik, dipl. psih.
Sveučilište u Zagrebu
Filozofski fakultet
Odsjek za psihologiju
Katedra za kliničku i zdravstvenu psihologiju
Ivana Lučića 3
10 000 Zagreb, Hrvatska
E-pošta: doriscrzk@yahoo.co.uk

KLJUČNE RIJEČI / *KEY WORDS*:
PTSP / *PTSD*
Faktori rizika / *Risk factors*
Kronični PTSP / *Chronic PTSD*
PTSP i parničenje / *PTSD and reimbursement*
Prospektivne i retrospektivne studije PTSP-a / *PTSD prospective and retrospective studies*

TO LINK TO THIS ARTICLE:

UVOD

PTSP i definicija traumatskoga iskustva

Posttraumatski stresni poremećaj je stanje koje predstavlja neposrednu ili odgođenu/produljenu reakciju na proživljeni traumatski događaj koja se manifestira psihološkim posljedicama poput izbjegavanja podsjetnika na traumu, ponovnog proživljavanja traumatskog događaja (npr. uznemirujući snovi) te pojačane autonomne pobuđenosti organizma. U općoj populaciji prevalencija ovog poremećaja kreće se od 1 % do 14 %, ovisno o metodi prikupljanja podataka i uzorku, no i važećim dijagnostičkim kriterijima. Dijagnostički kriteriji kao i definicija samoga traumatskog događaja u kontekstu ovoga poremećaja, radi višestrukih implikacija, nerijetko su tematika znanstvenih debata o čemu će biti više rečeno u sljedećem poglavlju.

U znanstvenom smislu ovaj se poremećaj spominje već od 19. stoljeća kada ga u svojoj opširnoj studiji Građanskog rata objavljenoj 1871. Jacob Mendez DaCosta spominje pod terminom *vojničko srce* (1). DaCosta je, naime, pokušao dokučiti koji su to čimbenici koji dovode do tako zamjetnih promjena u ratnih veterana kao rezultat bivanja u situaciji intenzivnog stresora odnosno traume. Traumatske situacije koje mogu inducirati ovaj poremećaj gotovo su

INTRODUCTION

PTSD and the traumatic experience definition

Posttraumatic stress disorder PTSD is a condition that presents an immediate or delayed/prolonged reaction to a traumatic event, manifested in psychological consequences such as avoiding traumatic reminders, re-experiencing traumatic events (e.g. disturbing dreams), and increased autonomic reactivity of the body. In the general population, the prevalence of this disorder ranges from 1 to 14%, depending on the method of participant sampling and data collection, but also valid diagnostic criteria. Diagnostic criteria as well as the definition of the traumatic event itself in the context of this disorder, for multiple implications, are often subject to scientific debates, which will be further discussed in the following chapter. In scientific terms, PTSD has been mentioned since the 19th century. In his extensive study of the Civil War published in 1871, Jacob Mendez DaCosta describes this condition under the terms of *the soldier's heart* (1). DaCosta was interested in the factors that lead to such noticeable changes in war veterans as a result of experiencing intense stress or a traumatic situation. Traumatic situations that usually induce this disorder almost always represent some form of life-threatening

uvijek neki oblik životne ugroženosti ili prijetnje sigurnosti traumatizirane osobe pri čemu je nerijetko važan i doživljaj bespomoćnosti ili nedostatka kontrole. Situaciju traume često predstavljaju ratna zbivanja, zatočeništvo, logori, silovanje, prirodne katastrofe te prometne nesreće. Nerijetko se radi o situacijama koje su izvan uobičajenog iskustva osobe, odnosno koje bi bile većinu ljudi trauma, stoga ne čudi kako se ovaj poremećaj često naziva „normalnom reakcijom na abnormalne događaje".

Sama definicija traume predmetom je znanstvenih debata. Točnije, nerijetko se postavlja pitanje kriterija kojim neki događaj zadovoljava kvalitetu traumatskoga. U okviru nove inačice Međunarodne klasifikacije bolesti (MKB-11) trauma je definirana *kao ekstremno prijeteći ili užasavajući događaj ili serija događaja* (2). S druge strane, DSM-5 nudi temeljitiju definiciju u kojoj je *osoba izložena smrti, prijetnjom smrti, ozljedi ili prijetnjom ozlijedi te prijetnjom ili stvarnom seksualnom nasilju putem izravne izloženosti, osobnim svjedočenjem te indirektno putem bliske osobe koja je bila izložena traumi*. Prepoznata je i ponavljana ili intenzivna izloženost traumatskim detaljima u okviru profesionalne dužnosti kao okidač posttraumatske reakcije (3).

U novije vrijeme, nova kategorija potencijalnih traumatskih događaja za PTSP sve se više istražuje, a odnosi se na zdravstvena stanja poput poroda te dijagnoza životno ugrožavajućih bolesti poput karcinoma ili kardiovaskularnih bolesti (npr. infarkt). Prema *Modelu dugoročne somatske ugroze* D. Edmonsona (*Enduring Somatic Threat model*) nakon neugodnih iskustava u okviru zdravstvenih smetnji potencijalno se razvija specifični oblik PTSP-a koji je orijentiran na zbivanja u budućnosti, posebice u kognitivnoj domeni. Prema ovome modelu, trauma je tjelesnog podrijetla, nerijetko je kroničnoga tijeka, dok su intruzivne misli povezane sa zbivanjima u budućnosti (npr. povratak maligne bolesti) te visoko kognitivne u svojoj kvaliteti (4). Iako se podatci u okviru ovog područja tek prikupljaju,

situation or a threat to the safety of an individual, with an emphasis on the experience of helplessness or lack of control. These events usually refer to war events, detention, war camps, sexual violence, natural disasters, and traffic accidents. Such experiences often represent events that are beyond the usual experience of a person, or that would be traumatic for most people, so it's no surprise that this disorder is often termed as a "normal reaction to abnormal events". However, the definition of trauma itself is often a subject of scientific debates. More specifically, the question of establishing the criteria by which an event satisfies the quality of a trauma is often discussed by researchers. Within the new version of the International Classification of Diseases (MKB-11), a traumatic event is defined as *an extreme threatening or horrifying event or series of events* (2). On the other hand, DSM 5 provides a more elaborate definition of a traumatic event in a way that *a person is exposed to death, threatened death, actual or threatened serious injury, or actual or threatened sexual violence by direct exposure, witnessing in person or indirectly through a close traumatized person*. Repetitive or intense exposure to traumatic details within professional duty as a trigger of a post-traumatic reaction (3) is also recognized.

More recently, a new category of potential traumatic events for PTSD that refer to health conditions such as birth or diagnoses of potentially life-threatening diseases such as cancer or cardiovascular disease (e.g. cardiac arrest) are increasingly investigated. According to the *Enduring Somatic Threat model* by D. Edmonds, a specific form of PTSD oriented towards future events, especially in the cognitive domain, potentially develops after unpleasant experiences within health disturbances. According to this model, trauma is of bodily origin, it is often chronic, while intrusive thoughts are related to future events (e.g. the return of a malignant disease) and highly cognitive in their quality (4). Although the data regarding this area is

istraživanja posljednjega desetljeća potvrđuju smjer ovih pretpostavki posebice u području kardiovaskularnih bolesti (5,6), malignih bolesti (7,8) te poroda, kao i njegove vrste (9-11). Iz svega navedenoga nameće se zaključak kako nove kategorije kao i podvrste ovoga poremećaja čekaju svoju temeljitiju znanstvenu potvrdu te ih možemo očekivati u okviru nekih budućih inačica dijagnostičkih klasifikacija bolesti. Upravo su kriteriji novoobjavljenih dijagnostičkih kategorizacija DSM-5 i MKB-11 te njihova problematika za utvrđivanje što sačinjava i uzrokuje ovaj poremećaj predmetom sljedećega poglavlja.

PTSP i problematika dijagnostičkih kriterija

Opis same kliničke slike ovoga poremećaja ovisi uvelike i o aktualnim dijagnostičkim klasifikacijama koje se tijekom godina usklađuju s nalazima suvremenih istraživanja. Tako je u novoj inačici američke DSM klasifikacije (DSM-5) iz 2013. godine PTSP iz kategorije anksioznih premješten u novu kategoriju *Poremećaja vezanih uz traumu i stresore*. Nadalje, iznenadan gubitak bliske osobe zbog prirodne smrti više se ne smatra jednim od traumatskih čimbenika nastanka ovoga poremećaja kao ni nužan doživljaj emocija straha, bespomoćnosti ili strave. Nova klasifikacija donosi i dvije zasebne skupine simptoma koje se odnose na *izbjegavajuće ponašanje* te *negativne promjene u mišljenju i raspoloženju*. Naposlijetku, uključena su i tri nova simptoma koja se javljaju nakon traume u razmatranju dijagnoze PTSP-a. Spomenuti se odnose na *pretjerano negativne misli i pretpostavke o sebi i svijetu* kao i *negativan afekt* te *nesmotreno* ili *destruktivno ponašanje*. Što se samoga tijeka poremećaja tiče DSM-5 ne čini više razliku između kroničnoga te akutnoga tijeka PTSP-a, no zahtijeva da smetnje traju duže od mjesec dana za postavljanje dijagnoze (3).

Iako prema novoj DSM kategorizaciji akutni i kronični tijek ovoga poremećaja više ne po-

still being collected, findings of recent research confirm the direction of these assumptions, particularly in the field of cardiovascular disease (5,6), malignant diseases (7,8), and different kinds of birth (9-11). Therefore, it can be concluded that new categories and subtypes of this disorder are waiting for their more thorough scientific confirmation and we can expect for them to appear within some future versions of the diagnostic classifications of this disease.

The next chapter deals with the aforementioned issues arising from the new DSM 5 and ICD-11 PTSD diagnostic criteria.

PTSD and the diagnostic criteria issues

The clinical description of PTSD is largely dependent on current diagnostic classifications that are aligned with the findings of recent research. Thus, in the new version of the American DSM Classification (DSM 5), PTSD is removed from the category of Anxiety Disorders and has been placed to a new category of Trauma and Stress Disorders. Furthermore, the sudden loss of a close relative due to natural death is no longer considered to be one of the traumatic factors of the emergence of this disorder, nor is the experience of the emotion of fear, helplessness, or horror a necessary factor for diagnosing this disorder. The new classification also brings two separate groups of symptoms related to avoidance behaviour and negative alterations in cognitions and mood. Finally, there are three new symptoms that must occur after trauma for considering the diagnosis of PTSD. The above mentioned are related to excessively negative thoughts and assumptions about oneself and the world, negative affect, and reckless or destructive behaviour. As far as the course of the disorder itself is concerned, DSM-V does not differentiate between the chronic and acute course of PTSD but requires that the disturbances last

stoje, kliničko iskustvo te mnoga istraživanja ovoga područja ukazuju kako se ovaj poremećaj može očitovati raznoliko (duljinom i kliničkom slikom) u različitih pojedinaca. Isto potkrepljuje i zanimljiva činjenica kako nova kategorizacija unutar DSM-5 omogućava 636 120 različitih kombinacija u simptoma PTSP-a u okviru kojih je moguće postaviti dijagnozu istoga, dok je prethodna (DSM-IV-TR) omogućavala 79 794 kombinacije (12). Stoga je moguće postaviti i hipotetsko pitanje količine različitih kombinacija produljenoga tijeka ovoga poremećaja koje se mogu pojaviti u različitih pojedinaca. Istraživanja tijeka PTSP-a pokazuju kako stope spontanih oporavaka u prvoj godini nakon traume iznose oko 50 % (13,14) te da oko jedna trećina pacijenata ima PTSP s odgođenim početkom (15-17). Dok su ove studije poboljšale naše razumijevanje rizika i tijeka PTSP-a, dugoročni tijek kroničnog PTSP-a ipak ostaje nejasnijim, jer je većina ovih istraživanja usmjerena istraživanju prve godine nakon traume.

Nadalje, definicija ovog poremećaja dodatno se komplicira objavom nove inačice Međunarodne klasifikacije bolesti (MKB-11) (2). Svrha nove inačice bila je ostvarivanje razlikovanja između normalnih reakcija na stresore i PTSP-a, kao i pojednostavljenje utvrđivanja dijagnoze. Kriteriji su stoga izdvojeni u svega tri kategorije iz kojih je potrebno zadovoljiti po dva simptoma. Kategorije se odnose na (1) *ponovno proživljavanje traumatskog događaja*, (2) *izbjegavanje podsjetnika na traumu*, te (3) *intenzivni doživljaj postojeće prijetnje*. Simptomi moraju trajati mjesec dana ili duže te je potrebno narušeno funkcioniranje u barem jednom području života pojedinca. No, tolerira se i odgođena reakcija na traumatski događaj na više od 6 mjeseci nakon samog iskustva. Nadalje, MKB-11 razlikuje također i kategoriju kompleksnog PTSP-a koji je definiran kao *reakcija pojedinca nakon izloženosti višestrukim traumatskim događajima ili onima produljenoga trajanja iz kojih je izlazak nemoguć ili izrazito*

longer than one month (3). Although according to the new DSM-V categorization the acute and chronic course of this disorder no longer exist, clinical experience and a lot of research in this area indicate that this disorder can be manifested in a variety of forms in different individuals. The latter is further confirmed by the fact that the new categorization within DSM-V allows for 636,120 different combinations of PTSD symptoms within which it is possible to diagnose it, while the previous (DSM-IV-tr) enabled for 79,794 combinations (12). Therefore, a hypothetical question of the quantity of different combinations of the prolonged course of this disorder that may occur in different individuals also arises. PTSD studies show that spontaneous recovery rates in the first year after trauma account for about 50% (13,14), and about one third of patients have a delayed onset of PTSD (15-17). While these studies have improved our understanding of the risk factors and the course of PTSD, the long-term course of chronic PTSD remains unclear, as most of these studies focus on the first year following trauma. Furthermore, the definition of this disorder is further complicated by the publication of the new version of the International Classification of Diseases (ICD-11) (2). The purpose of the new diagnostic criteria concerning PTSD was to make a distinction between normal stress responses and PTSD as well as to simplify diagnosing the disorder. Criteria are therefore separated into only three categories from which two symptoms should be met. The categories are related to (1) re-experiencing a traumatic event, (2) avoidance of traumatic reminders, and (3) an intense experience of an existing threat. Symptoms must last for a month or longer and require the presence of impaired functioning in at least one area of an individual's life. However, a delayed reaction to a traumatic event for more than 6 months after the experience itself is tolerated. Furthermore, ICD-11 distinguishes between the category of complex PTSD that is defined as an individual's

težak (2). Osim zadovoljenja po dva simptoma iz prethodne tri kategodije, ova dijagnoza obuhvaća još tri koje se odnose na *teškoće samoorganizacije*. Navedeni se odnose na *teškoće regulacije raspoloženja*; *negativno samopoimanje* te *narušeno interpersonalno funkcioniranje*.

Može se reći kako je glavna tema MKB-11 kriterija u okviru dijagnostike PTSP-a prepoznavanje dijagnoza od istaknutog kliničkog značenja. Prijedlozi novih dijagnostičkih kriterija naišli su na brojne reakcije znanstvene zajednice. Istraživači ovog područja upozoravaju kako će ti prijedlozi potencijalno štetiti pojedincima s lakšim i umjerenim oblicima ovoga poremećaja kojima će potencijalna pomoć, radi izostanka zadovoljenja kriterija, možda biti uskraćena. Istraživanje Wiscoa i suradnika potvrdilo je kako predloženi PTSP kriteriji u okviru MKB-11 identificiraju značajno manje pojedinaca s ovom dijagnozom od DSM-5 i IV, posebice protokom vremena (18). Istovremeno, novi kriteriji MKB-11 nisu doveli u ovome istraživanju do smanjenja komorbidnih dijagnoza, što je bio jedan od ciljeva nove inačice MKB-11. Stoga je preporuka istraživača kliničarima uzeti u obzir cjelokupno stanje pojedinca, mimo strogoga razvrstavanja smetnji u predložene dijagnostičke kategorije (19). Potonje je veoma važno uzeti u obzir radi potencijalne zdravstvene pomoći ili naknade za koju bi pojedinci mogli biti uskraćeni.

PTSP te problematika traženja naknade i parničenja

U razmatranju različitih faktora koji doprinose održavanju PTSP-a upravo se još nedovoljno istražen proces parničenja i traženja naknade čini osobito važnim. Stoga je pružanje pregleda istraživačkih nalaza i unutar ovoga područja predstavlja jedan od važnijih motiva pisanja ovoga preglednoga rada. Naime, istraživanja pokazuju kako osobe koje traže novčanu naknadu imaju izraženije smetnje u okviru dija-

reaction after exposure to multiple traumatic events or those of prolonged duration from which the escape is impossible or extremely difficult (2). Apart from meeting the two symptoms from the first three categories, this diagnosis includes three more that relate to the difficulties of self-organization. These are related to mood regulation problems; negative self-concept and impaired interpersonal functioning. It can be argued that the main theme of the ICD-11 criteria regarding the diagnosis of PTSD is to recognize the diagnosis of clinical significance. Proposals for new PTSD diagnostic criteria within the new edition of ICD-11 have met numerous critiques and reactions of the scientific community. Researchers of this area warn that individuals with mild or moderate forms of this disorder may be potentially harmed by these criteria in a way that they could be denied potential help due to the lack of criteria for PTSD being met. Research by Wisco and associates has confirmed that the proposed PTSD criteria under ICD-11 identify significantly fewer individuals with this disease against DSM-5 and IV criteria, especially with the passage of time (18). At the same time, the new ICD-11 criteria did not lead to a reduction of comorbid diagnoses in this study, which was one of the goals of the new ICD-11 edition regarding PTSD. Therefore, the recommendation to clinicians is to consider the overall condition of an individual, rather than making a strict classification of their disturbances against the proposed diagnostic categories (19). The latter is very important to consider for the purpose of potential health care or compensation that could be denied to these individuals.

PTSD and the problem of litigation and seeking compensation

In consideration of the various factors contributing to the maintenance of PTSD, the issue of litigation and compensation seeking

gnoze PTSP-a od pojedinaca s ovim poremećajem koji su izvan procesa traženja naknade i parničenja (20-22). Znanstvenici su opažene nalaze pokušali razjasniti u okviru tri moguća razjašnjenja. Spomenuti se odnose na hipoteze *stvarne narušenosti funkcioniranja* (*disability hypothesis*), *traženja financijske dobiti* (*finantial gain hypothesis*) kao i *hipoteze pojačanoga stresa* (*stress hypothesis*) (23). Iz navedenoga jasno je kako veza između izraženosti simptoma PTSP-a i traženja financijske dobiti nije sasvim jednostavna i razjašnjena. Stoga je ovo područje važan aspekt u istraživanju faktora rizika za kronični tijek ovoga poremećaja. Dostupni nalazi istraživanja ovoga područja opisani su unutar posljednjeg poglavlja *Socioekonomskih faktora*.

Zbog složenosti kliničke slike te nerijetko dugotrajnog i multidimenzionalnog pristupa liječenju ovaj je poremećaj već dugi niz godina predmet brojnih znanstvenih studija. Istraživačka pitanja često se odnose na razmatranja različitih faktora rizika za razvoj poremećaja, poput bioloških i psiholoških ranjivosti, tijeka oporavka kao i procjene učinkovitosti različitih terapijskih tretmana PTSP-a u ovisnosti o tipu traume kao i rodu traumatizirane osobe.

Na osnovi svega navedenoga, cilj ovoga rada ponuditi je pregled suvremenih istraživanja faktora rizika te tijeka posttraumatskog stresnog poremećaja. Kako se radi o veoma kompleksnom poremećaju očigledno je kako je sam nastanak ovog poremećaja rezultat interakcije različitih elemenata, poput bioloških (anksiozni prag, interoceptivna svjesnost), psiholoških, socijalnih te nedovoljno istraženih faktora socioekonomskog statusa pregledom kojih biva zaključen cjelokupni pregled. Stoga je jasno kako utvrđivanje različitih faktora rizika za razvoj PTSP-a pruža obuhvatniji odgovor na pitanje tko će s većom vjerojatnošću razviti PTSP kao odgovor na traumatsko iskustvo, a tko neće.

still remains under investigated. Hence, exploration of research findings within this area is one of the key motives for writing this review paper. Namely, research has shown that people seeking financial compensation have more severe symptoms within the PTSD diagnosis than individuals with this disorder that are not seeking reimbursement (20-22). The researchers of this area argue that these findings could be clarified within three possible explanations. These refer to the *disability hypothesis, financial gain (hypothesis), and stress hypothesis* (23). From the above mentioned it is clear that the link between the expression of PTSD symptoms and seeking financial gain is not quite so simple or clarified. Therefore, this area represents an important aspect within the studies of various risk- and chronic course factors of this disorder. The available research findings in this area are described within the last chapter of this paper that refers to *Socioeconomic Factors*. Due to the complexity of its clinical manifestation and the often long-term and multidimensional approach to treatment, this disorder has been the subject of numerous scientific studies for many decades. Research questions often relate to consideration of various risk factors for the development of this disorder such as biological and psychological vulnerabilities, recovery rates, and assessment of the effectiveness of various PTSD therapeutic treatments depending on the type of trauma. Therefore, the aim of this paper is to provide an extensive overview of current findings on PTSD risk factors. Since PTSD is a very complex disorder, it is obvious that the very occurrence of this disorder is the result of the interaction of various elements. The above mentioned refer to biological (anxiety threshold, interoceptive sensitivity/consciousness), psychological, social, and insufficiently investigated factors of socioeconomic status. It's therefore clear that determining the various risk factors important for the development of PTSD provides a more comprehensive answer to a question of who

METODOLOGIJA

U okviru nastanka ovoga rada pretražene su internetske baze znanstvenih časopisa *Science Direct, PubMed, PsychInfo i Researchgate* tijekom razdoblja od svibnja 2013. do svibnja 2018. godine za ključne riječi koje se tiču faktora rizika i kroničnoga tijeka PTSP-a. Termini za pretraživanje uključivale su kombinaciju riječi PTSD/PTSP i sljedećih fraza i/ili termina na engleskom i hrvatskom jeziku: *risk factors*/faktori rizika, *chronic course*/kronični tijek, *anxiety sensitivity*/anksiozna osjetljivost, neuroticism/*neuroticizam*, osobine ličnosti/*personality traits*, *neurobiology*/neurobiologija, *genetics*/genetika, *brain imagery*/mozgovni zapisi, *anxiety*/anksioznost, *depression*/depresija, *comorbidity*/ komorbiditet, *reimbursement, compensation*/parničenje. Odabir radova s obzirom na relevantnost teme rezultirao je konačnim odabirom 177 publikacija čiji su rezultati spomenuti u ovom preglednom radu. S obzirom na općenito manju količinu prospektivnih longitudinalnih studija ovoga područja objedinjeni su dostupni istraživački rezultati objavljeni u razdoblju od 1988. do 2018. godine.

FAKTORI RIZIKA ZA RAZVOJ POSTTRAUMATSKOG STRESNOG POREMEĆAJA

Faktore rizika za razvoj posttraumatskog stresnog poremećaja možemo radi preglednosti podijeliti u nekoliko većih skupina. To su faktori koji proizlaze iz osobina ličnosti i strategija suočavanja, psihijatrijske povijesti i komorbidnih stanja poput poremećaja ličnosti, vjerovanja i atribucija te bioloških faktora i socijalnih okolnosti.

Osobine ličnosti

Kada faktore rizika za razvoj posttraumatskog stresnog poremećaja sagledavamo iz aspekta osobina ličnosti, jedna je osobina posebice

will most likely develop PTSD in response to a traumatic experience and who will not.

METHODS

The Internet databases of Science Direct, PubMed, PsychInfo, and Researchgate were searched over the period from May 2013 to May 2018 for keywords related to risk factors and chronic PTSD. The search terms included a combination of the word PTSP and the following phrases and/or terminology in English and Croatian: risk factors, chronic course, anxiety sensitivity, neuroticism, personality traits, neurobiology, genetics, brain imagery, anxiety, depression, comorbidity, litigation, reimbursement, compensation. The selection of papers with respect to relevance of the topic resulted in the final selection of 177 publications whose results are mentioned in this review paper. Considering the generally smaller amount of prospective longitudinal studies in this area, the current review gathered the available research results published during the period from 1988 to 2018.

PTSD RISK FACTORS

Risk factors important for the development of PTSD can be divided into several major groups. These refer to the factors that arise from personality traits and coping strategies, psychiatric history, and comorbid states such as personality disorders as well as biological factors and social circumstances.

Personality traits

When considering personality traits as risk factors for the development of PTSD, one trait seems to be particularly salient within this context. The latter refers to neuroticism - a trait that represents a permanent tendency to re-

izražena u ovom kontekstu. Riječ je o neuroticizmu, odnosno osobini koja se manifestira trajnom sklonošću k reagiranju na događaje negativnim afektom, uključujući anksioznost te depresivnost (24). Neuroticizam je značajno povezan s PTSP-om (25,26) i faktor je rizika za razvoj posttraumatskog stresnog poremećaja nakon izlaganja traumatskom događaju (27). Jedan od nalaza koji pokazuje koliko je ova osobina značajna za nastanak PTSP-a je taj da je izrazito naglašen neuroticizam faktor rizika nezavisan od izloženosti borbi koji je dobiven na uzorku stotine veterana vijetnamskog rata s posttraumatskim stresnim poremećajem (28) i doprinosi više objašnjavanju varijance simptoma PTSP-a od samih ratnih iskustava (29-32). Istraživanje O'Toolea, Marshalla, Schurecka i Dobsona (33) na slučajnom uzorku australskih ratnih veterana s dijagnozom PTSP-a i bez te dijagnoze pokazalo je kako su rezultati na ljestvici neuroticizma uoči novačenja za vojsku bili kasnije povezani s razvojem PTSP-a. Slični rezultati (povezanost neuroticizma ili anksioznosti i posttraumatskih reakcija) dobivaju se i na uzorku osoba oboljelih od civilnog PTSP-a zbog različitih vrsta traumatskih događaja (34-37). Brojna istraživanja ovoga područja također ukazuju na povezanost PTSP-a i ostalih osobina ličnosti poput primjerice negativne emocionalnosti, traženja novosti, sklonosti hostilnosti i ljutnji i anksioznosti kao osobine ličnosti (38). Također, negativno je povezan s ekstraverzijom, optimizmom te kombinacijom visoke pozitivne i niske negativne emocionalnosti (39). Potonje ukazuje na potrebu obuhvatnijeg, odnosno multidimenzijskog pristupa u razmatranju odnosa i uloge različitih potencijalnih faktora rizika u nastanka PTSP-s. Istraživanja su također pokazala kako je nisko izražena osobina *smjelosti* (engl. *hardiness)* doprinosila objašnjenju veće varijance simptoma PTSP-a nego sami traumatski događaji (40). Nadalje, u prospektivnom istraživanju Tomassena i sur. (41) *smjelost* je imala indirektan utjecaj na smanjenje simptoma PTSP-a u norveških voj-

act to events with a negative affect, including anxiety and depression (24). Neuroticism is significantly associated with PTSD (25,26) and presents a risk factor for the development of PTSD after exposure to a traumatic event (27). One of the findings showing the significance of this trait for the emergence of PTSD is that high neuroticism presents a risk factor independently of exposure to combat in Vietnam veterans with PTSD (28). This trait also contributes more to the explanation of the variance of PTSD symptoms than war experiences themselves (29-32). The research of O'Toole, Marshall, Schureck, and Dobson (33) on a random sample of Australian veterans with and without PTSD showed that the results on the neuroticism scale in military recruits were later related to the development of PTSD. Similar results (association of neuroticism or anxiety and posttraumatic reactions) were also obtained on a sample of individuals with PTSD developed as a result of various types of traumatic events (34-37). Ample research in this area also points to the correlation between PTSD and other personality traits, such as negative emotionality, seeking novelty, hostility, and anger and trait anxiety (38). PTSD is also negatively associated with extraversion, optimism, and combination of high positive and low negative emotionality (39). The latter points to the need for a more comprehensive or multidimensional approach to the consideration of the relationship and role of the various potential risk factors in the emergence of PTSD. Studies have also shown that the low expression of *hardiness* contributed to the explanation of a greater variation of PTSD symptoms than traumatic events themselves (40). Furthermore, in a prospective study by Tomassen et al. (41) hardiness has had an indirect impact on reducing PTSD symptoms in Norwegian soldiers via reduced use of avoidance strategies - another risk factor significant for the emergence of PTSD. Hardiness is a feature that represents a set of personality traits that act as a protective factor in dealing

nika djelujući na smanjeno korištenje strategija izbjegavanja - još jednoga od fakora rizika za nastanak PTSP-a. Riječ je o osobini koja je skup osobina ličnosti koje djeluju kao zaštitni faktor pri suočavanju s traumatskim iskustvom (42). Ova osobina djeluje mehanizmima viđenja stresne situacije zanimljivom i punom značenja za pojedinca koji istovremeno stresore procjenjuje podložnima kontroli. Također, promjena je viđena kao mogućnost osobnog rasta te je procijenjena normalnim dijelom života. Još jednu od osobina za koju se pretpostavlja kako mnogostrukim putevima djelovanja uvećava rizik za pojavu PTSP-a je anksiozna osjetljivost.

Anksiozna osjetljivost

Anksiozna osjetljivost je veoma važan faktor u razmatranju nastanka posttraumatskog stresnog poremećaja (43). Riječ je o osobini koja predstavlja strah od unutarnjih promjena (tjelesnih, mentalnih, socijalne brige) radi uvjerenja kako su te promjene ugrožavajuće za dobrobit pojedinca. Pretpostavka je kako je ova osobina faktor rizika putem dva zasebna mehanizma djelovanja (44). Pojedinci s visokim razinama ove osobine mogu reagirati intenzivnije na traumatski stresor pri čemu će im dodatan izvor stresa biti i zabrinutost zbog vlastitih reakcija na stresor. Spomenuto potencijalno umanjuje i prag potreban za stresnu reakciju povećavajući spektar situacija koje bi kod pojedinca mogle potaknuti pojavu posttraumatskog stresa. Drugi mehanizam pretpostavlja kako traumatska situacija može potaknuti pojavu PTSP-a i anksiozne osjetljivosti koja tada povratno uvećava intenzitet simptoma. Dosadašnji istraživački napori općenito potvrđuju pretpostavku o anksioznoj osjetljivosti kao faktoru rizika za pojavu PTSP-a (45-48). Feldner i sur. (49), primjerice, u prospektivnoj su studiji utvrdili kako je anksiozna osjetljivost značajan prediktor simptoma PTSP-a u uzorku 400 mladih odraslih osoba tijekom razdoblja praćenja od 18 mjeseci. Boffa

with traumatic experience (42). This feature works via mechanisms of perceiving a stressful situation as interesting and full of meaning for an individual who at the same time perceives stressors as controllable. Also, these individuals perceive change as a potential for personal growth and view it as a normal part of life.

The next section is devoted to the trait of anxiety sensitivity - another feature that is assumed to increase the risk for PTSD in multiple ways.

Anxiety sensitivity

Anxiety sensitivity presents an important factor in considering the emergence of PTSD (43). This feature refers to fear of internal changes (physical, mental, social worries) due to a belief that an individual's wellbeing is being endangered. It's hypothesized that this feature presents a risk factor through two separate mechanisms of action (44). Individuals with high levels of this trait may react more intensively to a traumatic stressor, with worry (that arises as a result of their own reactions to the stressor) being an additional source of stress. This potentially reduces the threshold required for a stress response by increasing the range of situations that could cause an individual to develop posttraumatic stress. Another mechanism assumes that a traumatic situation may trigger the onset of PTSD and anxiety sensitivity, which then increases the intensity of the PTSD symptoms. Previous research generally confirms the assumption of anxiety sensitivity being a risk factor for the occurrence of PTSD (45-48). In prospective studies, Feldner et al. (49), for example, found that anxiety sensitivity was a significant predictor of PTSD symptoms in a sample of 400 young adults during an 18-month follow-up period. Boffa et al. (50) investigated whether elevated anxiety sensitivity before the traumatic event is a risk factor for posttraumatic stress symptoms. The results of their study showed that pretraumatic elevat-

i sur. (50) istražili su je li povišena anksiozna osjetljivost prije traumatskoga događaja faktor rizika za pojavu simptoma posttraumatskoga stresa. Rezultati njihove studije pokazali su kako je upravo pretraumatska povišena anksiozna osjetljivost predviđala simptome posttraumatskog stresa (PTSS) u skupini američkih studenata izloženih na različiti način pucnjavi na sveučilištu. Također, tjelesna komponenta anksiozne osjetljivosti djelovala je u interakciji sa stupnjem izloženosti traumatskom događaju na intenzitet izraženosti simptoma PTSS-a (50). Nadalje, Marshall, Miles i Stewart (44) istražili su longitudinalnim nacrtom vremenski odnos anksiozne osjetljivosti i intenziteta simptoma PTSP-a neposredno nakon tjelesne ozljede te nakon 6 i 12 mjeseci u 677 sudionika. Utvrđeno je kako i anksiozna osjetljivost i intenzitet simptoma PTSP-a međusobno djeluju recipročnim putem. Točnije, anksiozna osjetljivost predviđala je izraženost simptoma PTSP-a, kao što su i potonji predviđali kasniju izraženost osobine anksiozne osjetljivosti (44). Nadalje, čini se kako je anksiozna osjetljivost povezana i sa smanjenim mogućnostima samoregulacije pojedinca u kontekstu procesiranja emocionalnih podražaja (51) te sposobnostima postizanja *usredotočene svjesnosti* (engl. *mindfulness*) (52). Točnije, čini se kako su pojedini faktori unutar spomenute osobine potencijalne karakteristike pojedinca koje stvaraju veći rizik za razvoj PTSP-a. U istraživanju Schoorlove i Van Der Doesa (52) faktori usredotočene svjesnosti koji se odnose na *mogućnosti opisivanja iskustva*, *prihvaćanje bez osuđivanja* te *odsustvo reagiranja na unutarnje iskustvo* bili su u sudionika negativno povezani sa simptomima PTSP-a te depresivnom simptomatikom (52). Nadalje, ovi su faktori predviđali zasebnu varijancu simptoma PTSP-a nezavisno od varijabli anksiozne osjetljivosti i intenziteta traume (52). Faktori *mogućnosti opisivanja iskustva*, *prihvaćanje bez osuđivanja* te *djelovanje sa svjesnošću* predviđali su zasebnu varijancu simptoma depresije u osoba s PT-

ed anxiety sensitivity predicted posttraumatic stress (PTSS) symptoms in a group of American students exposed to different proximities to a shooting situation at their university. Also, the bodily concerns component of anxiety sensitivity has interacted with the degree of exposure to a traumatic event in predicting the intensity of PTSS symptoms (50). Furthermore, Marshall, Miles, and Stewart (44) investigated the temporal relationship of anxiety sensitivity and the intensity of PTSD symptoms immediately after physical injury and 6 and 12 months later on a sample of 677 participants. It was found that both anxiety sensitivity and the intensity of the PTSD symptoms interacted with each other in reciprocal ways. More specifically, anxiety sensitivity predicted the expression of PTSD symptoms, and the latter predicted a later expression of anxiety sensitivity (44). Furthermore, anxiety sensitivity seems to be associated with reduced possibilities of self-regulation of the individual in the context of processing emotional stimuli (51) and the ability to achieve mindfulness (52). Specifically, it appears that the individual variations within the aforementioned traits represent potential characteristics of an individual that pose a higher risk for PTSD development. In the research of Schoorl and Van Der Does (52), mindfulness questionnaire factors regarding the ability to Describe (experiences), Accept without Judgement, and Non-Reactivity to Inner Experience were negatively associated with symptoms of PTSD and depressive symptoms (52). Furthermore, these factors explained additional variance of PTSD symptoms independently of the anxiety sensitivity and traumatic intensity variables (52). Factors related to the ability to Describe (experiences), Accept without Judgement, and Act with Awareness accounted for a separate variance of depression symptoms in PTSD individuals independently of the cognitive reactivity variables and the amount of traumatic experiences (52). As can be seen from the described results, comorbid

SP-om nezavisno od varijabli kognitivne reaktivnosti te količine traumatskih iskustava (52). Kao što je vidljivo iz posljednjega primjera, komorbidne smetnje poput primjerice depresije, nerijetko se pojavljuju združeno sa smetnjama iz kruga PTSP-a.

Komorbiditet kao faktor rizika

Kao što je navedeno u uvodu poglavlja, osim određenih osobina ličnosti, prijašnje psihičke teškoće, odnosno psihijatrijska povijest te komorbidni poremećaji ličnosti također su faktori rizika koji doprinose razvoju PTSP-a. Prema Kessleru (53) osobe s posttraumatskim stresnim poremećajem imaju veću količinu peritraumatskih psihijatrijskih dijagnoza. U studiji Kulke i suradnika (54) prethodna dijagnoza anksioznog poremećaja pokazala se najznačajnijim faktorom rizika za razvoj PTSP-a u vijetnamskih veterana, dok je u drugom istraživanju bilo koja dijagnoza s Osi I tadašnjega DSM-IV bila povezana s dvostruko većom razinom dijagnoze PTSP-a u istraživanju Northa i suradnika (55). Čini se kako dugogodišnja dijagnoza poremećaja strukture ličnosti, posebice antisocijalnog poremećaja ličnosti, također povećava rizik od nastanka ratnog PTSP-a zbog traumatskog događaja (54). Što se ratnog PTSP-a tiče, povijest dječjeg antisocijalnog ponašanja bila je povezana s razvojem PTSP-a (56), dok je dijagnoza dječjeg poremećaja u ophođenju bila povezana s vjerojatnošću izlaganja traumatskom događaju kao i PTSP-om (57). Niža inteligencija faktor je rizika za izlaganje budućim traumatskim događajima te PTSP-a nakon izlaganja traumatskom događaju (58). Štoviše, prospektivne longitudinalne studije djece u rizičnim područjima pokazale su kako se čini da viša inteligencija reducira rizik za oboljenje od psihijatrijskog poremećaja (59,60) uključujući PTSP (61). Naposlijetku, meta-analiza Brehove i Seidlera (62) pokazala je kako je i stanje peritraumatske disocijacije rizik za kasniji razvoj PTSP-a. Ova se osobina

conditions such as depression often occur together with PTSD disturbances. Therefore, the purpose of the next paragraph is to provide relevant research findings regarding the area.

Comorbidity as a risk factor

As stated in the Introduction, apart from certain personality traits, previous mental problems, psychiatric history and comorbid personality disorders also represent risk factors that contribute to the development of PTSD. According to Kessler (53), people with PTSD have a higher amount of peritraumatic psychiatric diagnoses. In the study of Kulke et al. (54), an earlier diagnosis of an anxiety disorder was the most significant risk factor for the development of PTSD in Vietnamese veterans, while in the second study, any diagnosis from the DSM IV Axis I was associated with a twice higher probability of a PTSP diagnosis in the research of Northa et al (55). It appears that the long-term diagnosis of a personality disorder, particularly the antisocial personality disorder, also increases the risk for the emergence of PTSD due to a traumatic war event (54). As far as war PTSD is concerned, the history of child antisocial behavior was related to the development of PTSD in later life (56), while a diagnosis of childhood conduct disorder was associated to a higher probability of an exposure to a traumatic event in later life as well as PTSD (57). Lower intelligence is a risk factor for exposure to future traumatic events and post-exposure PTSD.

Moreover, prospective longitudinal studies of children in high-risk areas have shown that higher intelligence reduces the risk of psychiatric disorders (59,60), including PTSD (61). Finally, a meta-analysis by Brehove and Seidler (62) showed that the state of peritraumatic dissociation is also a risk for a later development of PTSD. This characteristic refers to the subjective experience of emotional numbing, distanc-

odnosi na subjektivni doživljaj emocionalne otupjelosti, distanciranja od drugih, smanjene reaktivnosti, depersonalizacije i derealizacije u trenutku zbivanja traumatskog događaja. Iako je u trenutku njezine pojave pojedinac trenutačno *zaštićen* od apsolutnog doživljaja traume, pretpostavka je kako ona dugoročno šteti ometajući integraciju traumatskog iskustva unutar eksplicitnog pamćenja pojedinca (63,64).

Kognitivni faktori

Posebna skupina faktora rizika za razvoj PTSP-a proizlazi iz kognitivne sfere, odnosno načina na koji ljudi pridaju značenje događajima i kako ih tumače. Pojedinčeva vjerovanja mogu djelovati raznoliko na smjer oporavka nakon traumatskog iskustva, štiteći ga ili olakšavajući nastanak smetnji. Primjerice, zaštitnim faktorima za razvoj ovog poremećaja nakon fizičkog napada pokazala su se vjerovanja o vlastitioj vrijednosti, sigurnosti i povjerenju u druge (65). Vjerovanje da svijet funkcionira na način kojeg opisuju značenje i koherentnost također predstavlja zaštitni faktor kao i pozitivna vjerovanja o vlastitoj samoefikasnosti (66-68).

S druge strane, u slučaju PTSP-a, četiri su se kognitivna elementa, odnosno vjerovanja pokazala značajnim za razvoj ovog poremećaja. To su: procjena događaja kao ugrožavajućeg; vjerovanja o osobnoj ranjivosti; pokušaji pripisivanja posebnog značenja događaju te vjerovanja o količini osobne kontrole (69). Procjena vlastite ranjivosti kao i ograničeni kapaciteti za suočavanje s izazovima (70,71), niska samoučinkovitost (72,73), procjena štetnosti te pridavanje zastrašujućih značenja događajima (74-76) vjerovanja su koja su povezana s razvojem posttraumatskih poremećaja. Još jedan od kognitivnih faktora rizika za razvoj PTSP-a je i pojedinčeva procjena štetnosti simptoma poput intruzivnih misli (77-79). Jedan od značajnih faktora u pripisivanju značenja traumatskom događaju

ing from others, reduced reactivity, depersonalization, and derealization at the moment of the occurrence of a traumatic event. Although at the time of its occurrence, peritraumatic dissociation protects an individual from the absolute trauma experience, it is assumed that it acts harmfully in the long term by hindering the integration of the traumatic experience within the explicit memory of the individual (63,64).

Cognitive factors

A particular group of risk factors for the development of PTSD results from the cognitive sphere, or the ways people ascribe meaning to events and how they interpret them. Individual beliefs can work differently on the direction of recovery after a traumatic experience, protecting it or facilitating the occurrence of disturbances. For example, beliefs about one's own value, safety and trust in others appeared to be protective factors for the development of this disorder after a physical attack (65). The belief that the world functions in a way that produces meaning and coherence is also a protective factor, as well as positive beliefs about one's self-efficacy (66-68). On the other hand, in the case of PTSD, four cognitive elements, or beliefs, have proven to be significant for the development of this disorder. These are: assessment of the event as a threat; beliefs about personal vulnerability; attempts to attribute special significance to the beliefs about the amount of personal control (69). Estimated self-vulnerability as well as limited capacity to deal with challenges (70,71), low self-efficacy (72,73), attachment of harmful or horrific meanings to events (74-76) are beliefs that are associated with the development of posttraumatic disorders. Another cognitive risk factor for the development of PTSD refers to the individual's assessment of the harmfulness produced by PTSD symptoms such as intrusive thoughts (77-79). One of the important factors in ascribing significance to a traumatic event is

je i atribucija odgovornosti te lokus kontrole, odnosno koncept koji se odnosi na pripisivanje kontrole, odgovornosti i krivnje unutarnjim ili izvanjskim faktorima. Posttraumatski stresni poremećaj povezan je s pripisivanjem kontrole izvanjskim uzrocima (80), dok druga istraživanja ukazuju na kompleksan odnos između tijeka poremećaja i lokusa kontrole u ovisnosti o vrsti traumatskog događaja što je detaljnije pojašnjeno u poglavlju koji razrađuje faktore kroničnog tijeka poremećaja.

U novije se vrijeme još jedan kognitivni faktor rizika za anksiozne poremećaje ističe unutar istraživanja ovog poremećaja. Riječ je o *kognitivnom stilu strepnje* koji se odnosi na stabilnu sklonost pojedinca viđenju prijetnje kao brzo napredujuće u vremenu i prostoru (81). Dosadašnji nalazi otkrivaju kako je simptomatika PTSP-a umjereno povezana s kognitivnim stilom strepnje (82,83). Iako tek u začetku, dosadašnji nalazi ukazuju kako manifestacije ovog kognitivnog stila u obliku pristrane obrade informacija, pažnje i pamćenja vezanih uz prijetnju podržavaju njegovu važnost kao faktora rizika za razvoj PTSP-a (84-88).

Kumulacija stresora i traumatskih iskustava

Osim bioloških faktora, osnovna pretpostavka aktualnog modela „kumulacije stresora" u nastanku PTSP-a (engl. *stressor-dose* model) je kako gomilanje proživljenih nedaća tijekom života doprinosi nastanku poremećaja te kako veća količina odnosno intenzitet traumatskog događaja uvjetuje veći intenzitet posttraumatskog stresnog poremećaja (89). Prijašnji višestruki traumatski događaji, posebice nasilje u obliku napada, su najjači faktor rizika za razvoj PTSP-a u populaciji (90), u veterana (91), silovanih žena (92) i među onima koji su razvili PTSP kao odgovor na napad u SAD-u 11. rujna (93). Iako je literatura puna nalaza koji potvrđuju povezanost količine traumatskih

the attribution of responsibility and the locus of control, that is, the concept of attributing control, responsibility and guilt to internal or external factors. PTSD is often associated with attribution of control to external agents (80) while other studies suggest a complex relationship between the course of the disturbance and the locus of control in dependence on the type of a traumatic event. The latter is explained in more detail in the chapters that deal with factors contributing to PTSD's chronic course. In recent times, one more cognitive risk factor for anxiety disorders is highlighted within the studies of this disorder. This refers to the looming cognitive style - a stable tendency of an individual to perceive a threat as rapidly rising in risk and intensity through time and space (81). Previous findings reveal that PTSD symptoms are moderately related to this cognitive factor (82,83). Although still in their beginning, the research findings indicate that the manifestations of this cognitive style in the form of biased information processing, as well as biased attention to threat and threat-related memories support its importance as a risk factor for PTSD development (84-88).

Stressor cumulation and traumatic experiences

In addition to the biological factors, the underlying assumption of the current Stressor-Dose Model is that the build-up of life-long disadvantages contributes to the emergence of a disorder. It also assumes that the greater intensity of a traumatic event will produce higher PTSD intensity (89). Previous multiple traumatic events, particularly violence in the form of attacks, represent the strongest risk factor for PTSD development in the general population (90), as well as in war veterans (91), raped women (92), and among those who developed PTSD in response to the attacks of September 11th (93). Although the literature is full of findings confirming the correlation of

događaja s vjerojatnošću rizika od PTSP-a, veliki broj istraživanja ne podržava pretpostavku o senzitizaciji u odgovoru na traumatski stresor. Primjerice, na uzorku američkih studenata Falsetti i Resnick (94) nisu utvrdili razlike u pojavnosti PTSP-a i depresije s obzirom na to je li osoba bila žrtva jednog ili nekoliko kriminalnih događaja. Isti nalaz dobiven je u slučaju usporedbe uzorka kanadskih i američkih vatrogasaca (95). Međutim, kada su Wykes i Whittington (96) u napadnutih sestara s psihijatrijskog odjela utvrdili kako se uzorak dijeli na one koje su na stres odgovorile niskom ili visokom razinom distresa postavilo se pitanje adaptiraju li ili senzitiziraju li prijašnji stresori pojedinca u odgovoru na traumatski stresor? U slučaju medicinskih sestara s reakcijom niske razine distresa autori su interpretirali ovaj nalaz adaptacijom, odnosno razvojem kognitivnih i emocionalnih vještina zbog suočavanja sa sličnim iskustvima. Iz toga je slijedio zaključak kako će određene osobe razviti adaptivne odgovore na višestruke traumatske događaje, dok će se u drugih pojačati osjetljivost na traumatske stresore (96). Neki autori povlače analogiju biološkom procesu imunizacije organizma od nekih bolesti kao odgovora na male doze otrovnih tvari (cjepivo) (89). Točnije, smatraju kako pojedinac razvija adaptaciju i strategije savladavanja većih stresora kao odgovor na prijašnje iskustvo ponavljanih stresora manjih intenziteta (89). Konzistentno s ovim modelom 90 % sudionika u istraživanju Aldwina, Suttona i Lachmana (97) izvijestilo je kako je suočavanje sa stresnim događajima potpomognuto prijašnjim iskustvima suočavanja sa stresorima. Ta su iskustva rezultirala poticanjem razvoja samopouzdanja i nekih novih i adaptivnih strategija suočavanja (97).

Osim psiholoških faktora, biologija PTSP-a opsežno je proučavana u smislu genetskih i hormonskih faktora te psihofizioloških odgovora kao i mozgovne anatomije.

the number of traumatic events and the risk for developing PTSD, a large number of studies do not support the assumption of a sensitization in response to traumatic stressors. For example, on a sample of US students, Falsetti and Resnick (94) did not determine the differences in the PTSD emergence and depression as to whether a person was a victim of one or several criminal events. The same finding was obtained in the case of a comparison of Canadian and American firefighters (95). However, when Wykes and Whittington (96) observed a dual pattern when observing attacked nurses from the psychiatric department, the authors found that the nurses either belonged to a group of those who responded to stress with a low or high level of distress. In the case of nurses with low levels of experienced distress, the authors interpreted this finding as an adaptation by developing cognitive and emotional skills through coping with similar experiences. This was further followed by the conclusion that certain individuals would develop adaptive responses to multiple traumatic events, while others would increase susceptibility to traumatic stressors (96). Some authors draw the analogy to the biological process of immunization of the organism from some diseases as a response to small doses of toxic substances (e.g. vaccine) (89). More specifically, it's assumed that the individual develops an adaptation and strategies for mastering major stressors in response to previous experiences of repeated stressors of lower intensity (89). Consistent with this model, 90% of participants in Aldwin, Sutton, and Lachman's research (97) reported that facing stressful events was backed by previous experience of dealing with stressors. The same has resulted in encouraging self-confidence and some new and adaptive coping strategies in these individuals (97). In addition to psychological factors, PTSD biology has been extensively studied in terms of genetic and hormonal factors of these psycho-physiological responses as well as brain anatomy.

Biološki faktori

Prema biološkim teorijama, trauma oštećuje noradrenergički sustav pri čemu povišene koncentracije noradrenalina uzrokuju da se osoba lakše prestraši i snažnije izražava emocije nego što bi to inače bilo normalno što podržavaju i empirijski nalazi (98,99). Nadalje, jedna od specifičnosti ovog poremećaja je i kronično poremećena regulacija hipotalamo-pituitarno-adrenalne osi (HPA-os) za koju se čini kako zbog povišene razine kortikotropin-oslobađajućeg hormona postaje pojačano osjetljiva na stres i učinke kortizola (100,101). Također, čini se kako je promijenjena aktivnost triju moždanih struktura: prefrontalnog korteksa, hipokampusa te amigdala od posebnog značenja za istraživanja karakteristika ovog poremećaja (102,103). Naime, rezultati velikog broja studija slikovnih prikaza mozga kod PTSP-a začetak su modela limbičke senzibilizacije te smanjene kortikalne inhibicije kod PTSP-a uz specifičnu disfunkciju područja uključenih u pamćenje, emocije i vidno-prostornu obradu (104). U studijama koje su inducirale simptome PTSP-a traumatskim audiozapisima utvrđena je aktivacija desnog limbičkog i paralimbičkog sustava (105) i vidnog korteksa kao i smanjeni protok krvi u medijalnom prefrontalnom korteksu, hipokampusu i vidnom asocijativnom korteksu (104). Kod veterana s PTSP-om, kao odgovor na slikovne podražaje povezane s borbom, utvrđen je povećan protok krvi u jezgrama amigdala i prednjem cingularnom korteksu, a smanjen u Brockinom području (106). Ove nalaze autori dovode u vezu s neverbalnim emocionalnim vidnim predodžbama koje su dio ponovnog proživljavanja simptoma PTSP-a (106). Nadalje, izlaganje osoba s PTSP-om dovodi do smanjenja protoka krvi u medijalnom prefrontalnom korteksu, području odgovornom za regulaciju emocionalnog odgovora preko inhibicije amigdala (104). Dugi se niz godina smatralo kako zbog traume dolazi do oštećenja moždanih struktura (npr. smanje-

Biological factors

According to biological theories, trauma causes a noradrenergic system damage, with elevated levels of norepinephrine causing a person to become frightened more easily and to express emotions more intensively than would normally be supported by empirical findings (98,99). Furthermore, one of the specificities of this disorder appears to be the chronically disturbed regulation of the hypothalamic-pituitary-adrenal axis (HPA) that is more sensitive to stress and cortisol effects as a result of elevated levels of corticosteroid-releasing hormone (100,101). It also appears that the activity of three brain regions is altered in PTSD: the prefrontal cortex, the hippocampus, and the amygdala, which is of particular importance for the research of the manifestations of this disorder (102,103). Namely, the results of a large number of brain imaging studies in PTSD presented the foundation for the lymphatic sensitization model and the reduced cortical inhibition hypothesis in PTSD with specific dysfunctions of areas involved in memory, emotions, and visuo-spatial processing (104). In studies that induced PTSD symptoms with traumatic audio, the activation of the right lymphatic and paralimbic system (105) and visual cortex as well as reduced blood flow in the medial prefrontal cortex, hippocampus, and visual associative cortex (104) were determined. In PTSD veterans, in response to images of stress-related stimuli, increased blood flow was found in amygdala nuclei and the frontal cingulate cortex, while it was decreased in Brock's area (106). These findings were linked to unrealistic emotional visual concepts that are part of the re-experiencing symptoms of PTSD (106). Furthermore, exposure therapy of individuals with PTSD leads to a decrease in blood flow in the medial prefrontal cortex, the area responsible for regulating the emotional response through amygdala inhibition (104). For a number of years, it has been thought that trauma results

ni volumen hipokampusa) koja se tada očituju i u funkcijskim deficitima verbalnog pamćenja (107). Određeno vrijeme postojala je i pretpostavka o značajnoj povezanosti između volumena hipokampusa i izraženosti posttraumatskih reakcija. Međutim, zaključak studije Gilbertsona i suradnika (108) u kojoj je proučavano 40 muških monozigotnih blizanaca različitih po izloženosti traumi u vijetnamskom ratu pokazala je nešto sasvim drugačije. Naime, traumi neizloženi, manji hipokampalni volumen bio je pretraumatski faktor rizika, umjesto da je njegov smanjeni volumen rezultat traumatskog događaja ili PTSP-a (108). Čini se kako sličan slijed prati i uočena pojačana aktivacija dorsalnog anteriornog cingularnog korteksa (dACK) u osoba s PTSP-om. Naime, studija blizanačkih potomaka osoba s PTSP-om pokazala je kako i prije izloženosti stresoru djeca ovih osoba pokazuju povišenu aktivaciju ove moždane regije važne za procesiranje emocionalnih sadržaja (109,110). Istraživanja također ukazuju kako predispoziciju za razvoj PTSP-a možemo potražiti i u premorbidnoj abnormalnoj strukturi i funkciji dACK-a (111,112), kao i u abnormalnoj povezanosti amigdala i dACK-a (113,114). Naposlijetku, Hendlerova i Admon (115) impliciraju kako je premorbidna osjetljivost na stres u osoba s PTSP-om potencijalno posredovana pretjeranom produkcijom emocije straha kao i disfunkcionalnom regulacijom straha u nastanku ovog poremećaja.

Što se genetskih istraživanja tiče, značajan utjecaj genetskih faktora utvrđen je u studijama monozigotnih i dizigotnih blizanaca te objašnjava gotovo 30 % varijance glavnih kategorija simptoma PTSP-a (116,117). Velika studija blizanaca veterana vijetnamskog rata utvrdila je kako 38 % genetske varijance doprinosi i paničnom poremećaju i PTSP-u, dok je dodatnih 14 % genetskog doprinosa bilo specifično za PTSP (118).

Glavna premisa istraživanja hormonskih utjecaja odnosila se na to da postoji vjerojatnost

in brain damage (e.g. decreased volume of the hippocampus), which is then manifested in functional deficits of verbal memory (107). For a certain time, there was also a presumption of significant correlation between the volume of the hippocampus and the expression of posttraumatic reactions. However, the conclusion of a study by Gilbertson and associates (108) in which 40 male monozygotic twins were studied against different Vietnam War traumas showed different results. Namely, trauma non-exposed, lower hippocampal volume was a pre-traumatic risk factor, rather than its reduced volume being the result of a traumatic event or PTSD (108). It seems that the same pattern can be observed in the increased activation of the dorsal anterior cingulate cortex (dACC) in PTSD individuals. Namely, the study of the twin descendants of people with PTSD has shown that prior to the stressor exposure, the children of these persons showed elevated activation of this cerebral region important for the processing of emotional content (109,110). Research also suggests that the predisposition to developing PTSD can be found in the premorbid abnormal structure and function of dACK (111,112), as well as in the abnormal communication between the amygdala and dACK (113,114). Ultimately, Hendler and Admon (115) imply that premorbid stress sensitivity in PTSD patients is potentially mediated by excessive fear production as well as dysfunctional regulation of fear in the onset of this disorder. As far as genetic research is concerned, significant influence of genetic factors has been established in monozygotic and dizygotic twin studies and explains nearly 30% of the major groups of PTSD symptoms (116,117). A large study of twin Vietnam veterans found that 38% of genetic variance contributed to panic disorder and PTSD, while an additional 14% of genetic contributions were specific to PTSD (118). The main premise of research on hormonal effects was that there is a likelihood that long-term stress hormone delivery caus-

kako dugotrajno lučenje hormona stresa uzrokuje promjene u moždanoj fiziologiji pa čak i anatomiji mozga, odnosno pokušalo se utvrditi postoje li ikakve promjene takve vrste specifične za PTSP te jesu li faktor rizika ili su posljedica traumatskog događaja, odnosno poremećaja. Središnji element ovog pitanja činili su nalazi studija hipotalamo-pituitarno-adrenalne osi (HPA os) koja kontrolira lučenje hormona, posebice kortizola zbog suočavanja sa stresorima. Točnije, studije su se bavile utvrđivanjem oslabljivanja lučenja kortizola odnosno *otupljivanja* funkcije HPA osi zbog ponavljanog izlaganja traumatskim događajima. Naime, nekolicina studija utvrdila je kako su razine kortizola neposredno nakon traume niže u pojedinaca koji kasnije razvijaju PTSP (119,120). Nadalje, dok je razina kortizola bila povišena u osoba žrtvi prvog seksualnog napada, oslabljeno lučenje kortizola zamijećeno je u žrtava višestrukih seksualnih napada (121). Razine kortizola bile su manje u veterana s aktualnom dijagnozom PTSP-a od onih koji više nemaju tu dijagnozu (122). Iz toga je proizašao zaključak kako postoji mogućnost kako ponavljanje traume uzrokuje senzitizaciju na podražaje te pretjeranu aktivaciju sustava povezanih s tjelesnom pobuđenošću što dugoročno dovodi do iscrpljivanja sustava hormonskih odgovora. S druge strane, možda se radi o faktoru rizika koji se očituje u neadekvatnoj mogućnosti davanja hormonskog odgovora na traumu, budući da novije studije ovoga područja ukazuju kako nema povezanosti između razina kortizola i osoba s PTSP-om (123).

Istraživanja psihofizioloških obrazaca povezanih s PTSP-om utvrdila su kako su pojačani srčani puls, plašljive reakcije te generalizirane reakcije na podražaje s izostankom navikavanja česti korelati PTSP-a (124,125). Studija monozigotnih parova Orra i suradnika (126) pokazala je kako pojačani srčani puls u odgovoru na zastrašujući podražaj nije faktor rizika, već znak koji nastaje nakon razvoja PTSP-a. Istra-

es changes in brain physiology and even brain anatomy. That is, it was attempted to establish whether any changes of these patterns exist that are specific to PTSD and whether they are a risk factor or a consequence of a traumatic event. The central element of this question presented the findings of the HPA axis studies, in which the secretion of hormones due to stressors, particularly cortisol, is controlled. Specifically, the studies focused on the weakening of cortisol secretion or the HPA axis function numbing due to repeated exposure to traumatic events. Specifically, several studies have determined that cortisol levels immediately after trauma are lower in individuals who subsequently develop PTSD (119,120). Furthermore, while the cortisol level was elevated in victims of the first sexual assault, impaired cortisol elevation was observed in multiple sexual assault victims (121). Cortisol levels were lower in veterans with current PTSD diagnosis than those who no longer have this diagnosis (122). The authors came to the conclusion that there is a possibility that repeated trauma experiences cause sensitization to stimuli and excessive activation of the system associated with physical excitement which in the long run leads to the exhaustion of the hormonal response system. On the other hand, this may also present a risk factor that is reflected in the inadequate ability to provide a hormonal response to trauma since recent studies in this area suggest there is no correlation between cortisol levels and PTSD (123).

The research findings on PTSD-related psycho-physiological patterns showed that increased heart rate, startle reactions, and generalized reactions without habituation to stimuli often correlate with PTSD (124,125). The study of monozygotic twins of Orr and associates (126) showed that an increased heart rate in response to a fearful stimulus is not a risk factor, but a sign that arises after the development of PTSD. Research by Keane et al.

živanje Keanea i suradnika (127) pokazalo je pak kako trećina osoba s PTSP-om ne pokazuje nikakav specifičan obrazac psihofiziološkog odgovora na znakove traume, što je podržano i u nekim drugim studijama (128).

Iz cjelokupne istraživačke građe opisane unutar ovog poglavlja može se zaključiti kako međudjelovanje različitih faktora rizika pogoduje nastanku ovog poremećaja. Točnije, proizlazi iz interakcije različitih osobina ličnosti i strategija suočavanja pojedinca, psihijatrijske povijesti i komorbidnih stanja poput poremećaja ličnosti, vjerovanja i atribucija te bioloških faktora i socijalnih okolnosti. Međutim, usprkos faktorima njegova nastanka, činjenica je kako se ovaj poremećaj neće održati jednako dugo u različitih pojedinaca. Stoga je cilj sljedećeg odlomka pružiti uvid u raznolike faktore koje dosadašnji istraživački rad ističe važnima za produljeno trajanje, odnosno kronični tijek PTSP-a.

FAKTORI KRONIČNOGA TIJEKA POSTTRAUMATSKOG STRESNOG POREMEĆAJA

Tijek PTSP-a moduliran je i različitim psihološkim i socijalnim faktorima. Nadalje, studije biologije kroničnog tijeka PTSP-a impliciraju promjene u senzitivnosti HPA-osi, potencijalno oštećenje moždanih struktura povezanih s pamćenjem te obrazac pojačanog psihofiziološkog odgovora na stresore. Međutim, istraživači ovog područja nerijetko naglašavaju poteškoće određivanja radi li se o premorbidnim deficitima ili rezultatom perzistirajućih simptoma ovog poremećaja. Nadalje, ističu potrebu za većom količinom longitudinalnih istraživanja koja bi se u što adekvatnijoj mjeri pozabavila ovim problemom. Iako postoji relativno veliki broj studija tijeka PTSP-a u smislu prevalencije i kvantifikacije zastupljenosti ove dijagnoze u vremenskom tijeku, manji ih se broj istraživao, pogotovo longitudinalnim pristupom, povezanost različitih faktora s tijekom samog

(127) showed that a third of PTSD individuals show no specific pattern of psychophysical response to signs of trauma, which is also supported in some other studies (128). From the overall research material described in this chapter, one can conclude that the interaction of different risk factors favours the emergence of this disorder. Specifically, it stems from the interaction of different personality traits and coping strategies of an individual, psychiatric history and comorbid states such as personality disorders, beliefs, and attributions as well as biological factors and social circumstances. However, despite the factors of its origin, the fact is that this disorder will have a different course both in length and intensity in different individuals. Therefore, the aim of the next section is to provide an insight into various factors that the research has shown to be important for the prolonged duration or chronic course of PTSD.

FACTORS OF THE PTSD CHRONIC COURSE DEVELOPMENT

The chronic course of PTSD is modulated by various psychological and social factors. Furthermore, the studies on the biology of chronic PTSD imply changes in HPA axis sensitivity, potentially impaired functions of memory-related brain structures, and a pattern of enhanced psychophysical response to stressors. However, researchers in this area often emphasize the difficulty of differentiating between premorbid deficiencies and the results of persistence of PTSD symptoms. Furthermore, they emphasize the need for more longitudinal research that would address the problem more adequately. Although there is a relatively large number of PTSD chronic course studies in terms of prevalence and quantification of this diagnosis over time, only a small number of them have been investigating, especially by longitudinal approach, the association of var-

poremećaja. Sljedeći odlomak sadrži pregled dosadašnjih spoznaja o tijeku ovog poremećaja u podlozi različitih vrsta traumatskih stresora.

Faktori razvoja kroničnoga tijeka

Što se kroničnog tijeka tiče, učestalost simptoma PTSP-a varira od 1 % (129) do 46 % (130) u žrtava prometnih nesreća 3-12 mjeseci nakon nesreće. U studiji Korena i suradnika (131) utvrđeno je kako otprilike 30 % ozlijeđenih žrtava prometnih nesreća ima PTSP i godinu dana nakon nesreće.

Malo je studija koje su se sustavno bavile istraživanjem prirodnog tijeka PTSP-a i odrednicama kroničnog tijeka ovog poremećaja. Kronični PTSP često je registriran u žrtava silovanja, mučenja, političkog nasilja, u izbjeglica i u ratnih veterana (132,133). Nadalje, različita istraživanja, kao i različite vrste traumatskih stresora pružaju ponešto drugačije podatke o zastupljenosti ovog poremećaja tijekom vremena. Tako prospektivna studija na australskim vatrogascima pokazuje kako 29 mjeseci nakon sudjelovanja u velikom šumskom požaru 30 % i dalje zadovoljava kriterije za prisutnost poremećaja, dok 14 godina nakon velike poplave u Buffalu 28 % preživjelih i dalje nije postiglo remisiju (54,134). Nadalje, 19 godina nakon izloženosti ratnim bitkama 15 % vijetnamskih veterana još uvijek je imalo PTSP (54), dok je 10 % žrtava prometnih nesreća imalo PTSP i 3 godine nakon nezgode (131)**.** Općenito, što se tijeka ovog poremećaja tiče, istraživanja ukazuju multifaktorski model longitudinalnog tijeka ovog poremećaja s različitim ishodima u ovisnosti o mnogobrojnim pre-, peri- i posttraumatskim faktorima rizika poput kognitivnog stila, strategija suočavanja, socijalne potpore i mnogih drugih koji potencijalno određuju njegov kronični tijek.

Simptomi PTSP-a brže se povlače tijekom prve godine, a kasnije je povlačenje postupno (53). Kronicitet ovog poremećaja olakšan je faktoious factors with the disorder itself. The next section presents an overview of the current knowledge about the different types of traumatic stressors underlying the chronic course of this disorder.

The PTSD chronic course factors

As far as PTSD chronic course is concerned, the frequency of PTSD symptoms varies from 1% (129) to 46% (130) in car accident victims 3-12 months post-accident. The study of Koren et al. (131) showed that about 30% of injured victims of traffic accidents had PTSD one year after the event. There have been a few studies that have systematically investigated the chronic course of this disorder. Chronic PTSD is often registered in victims of rape, torture, political violence, refugees, and war veterans (132,133). Furthermore, different research, as well as various types of traumatic stressors, provide somewhat different information on the presence of this disorder over time. A prospective study on Australian firefighters showed that 29 months after participating in a large forest fire, 30% of participants still met the criteria for the presence of the disorder, while 14 years after the major flood in Buffalo, 28% of survivors still didn't achieve remission (134,54). Furthermore, 19 years after exposure to war combat, 15% of Vietnamese veterans still had PTSD (54), while 10% of victims of traffic accidents had PTSD even 3 years after the accident (131). Generally, concerning the course of this disorder, research suggests a multifactorial model of a longitudinal course of this disorder with different outcomes depending on many pre- and posttraumatic risk factors such as cognitive styles, coping strategies, social support, and many others potentially determining its chronic course. PTSD symptoms diminish faster during the first year and later retreat gradually (53). The chronicity of this disorder is facilitated by factors such as high intensity traumatic experiences (e.g. concen-

rima poput traumatskog iskustva visokog intenziteta (npr. koncentracijski logori), otprije postojećim anksioznim poremećajima i poremećajima raspoloženja, socijalnim otuđenjem, većim brojem prvotnih simptoma PTSP-a, ženskim rodom, pojačanom otupjelošću ili pobuđenošću kao reakcijom na stresor te komorbidnim zdravstvenim bolestima (27). Perkonigg i suradnici (129) u svojoj prospektivnoj longitudinalnoj studiji zajednice pokušali su utvrditi po čemu se skupine potpune remisije razlikuju od skupine kod koje je zabilježen kronični tijek PTSP-a. Utvrdili su kako je iskustvo novog traumatskog događaja u razdoblju između dva mjerenja najviše razlikovalo ove dvije skupine. Nadalje, kada su iz uzorka izuzete ove retraumatizirane osobe, prediktorima kroniciteta pokazali su se veći broj izbjegavajućih ponašanja te simptoma PTSP-a te manja stopa percipirane samokompetentnosti i traženja pomoći u slučaju kronične slike PTSP-a. Kronični je tijek također povezan s neposrednom akutnom reakcijom na stres, primjerice u obliku pojačane psihomotorne napetosti (136). U studiji Davisona i suradnika (132) kronični PTSP bio je povezan sa smanjenom socijalnom podrškom, većom zastupljenošću simptoma socijalne fobije kao i većom stopom izbjegavajućih ponašanja. Zlotnick i suradnici (137) izvještavaju kako su trauma u odrasloj dobi kao i alkoholizam također povezani s nižim stopama remisije. Veliki broj studija kroničnog tijeka PTSP-a usredotočio se na istraživanja različitih kognitivnih faktora u podlozi izostanka remisije ovog poremećaja iz kojih je proizašla i kognitivna teorija kroničnog tijeka PTSP-a.

Kognitivni mehanizmi i obrada informacija

Perzistiranje simptoma PTSP-a u podskupini osoba koje su preživjele traumu objašnjava se putem čimbenika poput nepotpune emocionalne obrade (138) i disfunkcijske obrade informacija traumatskih sjećanja (139). Na-

tration camps), pre-existing anxiety disorders and mood disorders, social alienation, multiple initial symptoms of PTSD, female genital mutilation, increased numbness or excitement as a stressor response, and comorbid health illnesses (27). The prospective longitudinal community study of Perkonigg et al. (129) attempted to determine distinguished the groups that achieved complete remission from the groups that had a chronic PTSD. They found that the experience of a new traumatic event in the period between the two measurements contributed the most to explaining the differences between these two groups. Furthermore, when these retraumatised individuals were excluded from the sample, predictors of chronicity became a greater number of avoidance behaviours and PTSD symptoms, and a lower rate of perceived self-competency and seeking assistance in individuals with chronic PTSD. The chronic course is also associated with an immediate acute stress response, for example in the form of increased psychomotor tension (136). In the study of Davison et al., (132) chronic PTSD was associated with reduced social support, greater manifestation of the symptoms of social phobia, and higher rates of avoidance behaviour. Zlotnick et al. (137) reported that adult traumas and alcoholism are also associated with lower remission rates. A great number of chronic PTSD studies have focused on the research of various cognitive factors that prevented remission of its symptoms. The findings from these studies presented later a basis for the cognitive theory of PTSD.

Cognitive mechanisms and information processing

The persistence of PTSD symptoms in the subgroup of traumatic-event survivors is explained by factors such as incomplete processing of emotional stimuli (138) and dysfunctional processing of traumatic memory information (139). Furthermore, Rachman (138) completes

dalje, Rachman (138) nadopunjava koncept emocijske obrade naglašavajući važnost kognitivnih mehanizama u emocijskoj obradi. Ovaj obnovljeni fokus na individualne razlike u procjeni i sjećanja na traumu potencijalno pomaže objasniti suprotstavljene nalaze ranijih istraživanja o ulogama akutnih intruzija i simptoma izbjegavanja kao prediktora PTSP-a (139). Naime, simptomi akutnih intruzija povezivani su s uspješnim oporavkom od traume, jer su intruzije bile viđene pokazateljima emocionalne obrade (140), dok su druge studije otkrile kako su visoke razine akutnih intruzija prediktori slabijeg oporavka (142). Čini se da kognitivna teorija nudi moguće rješenje ovih naizgled proturječnih nalaza upućujući na to da simptomi PTSP-a perzistiraju kada pojedinci procesiraju traumu na način da dovodi do osjećaja intenzivne i neposredne prijetnje (143). Taj osjećaj prijetnje uvelike je potaknut negativnim procjenama i slabijom razradom i kontekstualizacijom kognitivne reprezentacije traume (143). Promjene u negativnim procjenama i sjećanjima na traumu obično su spriječene zbog nastojanja pojedinca da primijeni niz zaštitnih mehanizama i procesa kako bi se izbjegli bilo kakvi podsjetnici na traumatski događaj (142). Perzistentno visoke razine izbjegavajućih simptoma obično se zamjećuju kod nekih traumatiziranih pojedinaca tijekom vremena (144) te mogu ukazivati na smanjenu sposobnost asimilacije i potpune emocionalne obrade traumatskog događaja (138).

Atribucijski stilovi

Druga vrsta kognitivnih faktora koja doprinosi objašnjavanju kroničnog tijeka PTSP-a proizlazi iz istraživanja atribucijskih stilova i lokusa kontrole traumatiziranih osoba. Točnije, mnogo dokaza pokazuje da prihvaćanje vlastite krivnje (povezano vjerovanjima u unutarnji lokus kontrole) smanjuje rizik za nastanak PTSP-a. Prihvaćanje „umjerene krivnje“ povezano je s najznačajnijim poboljšanjem zbog

the concept of emotional processing by emphasizing the importance of cognitive mechanisms in processing emotional information. This renewed focus on individual differences in appraisals and trauma memories potentially helps in explaining the controversial findings of earlier research on the role of acute intrusions and avoidance symptoms as PTSD predictors (139). Namely, the symptoms of acute intrusions were linked to successful trauma recovery because intrusions were seen as indicators of emotional processing (140), while other studies found that high levels of acute intrusions predicted poorer recovery (142). Cognitive theory seems to offer a possible solution to these seemingly contradictory findings suggesting that PTSD symptoms persist when individuals process trauma in a way that leads to a sense of intense and immediate threat (143). This sense of threat is greatly stimulated by an individual's negative appraisals and the weaker elaboration and contextualization of traumatic cognitive representations (143). Changes in negative appraisals and memories of trauma are usually prevented by the effort of an individual to apply a series of protective mechanisms and processes to avoid any reminders of a traumatic event (142). Persistent high levels of avoidance symptoms are commonly observed in some traumatized individuals over time (144) and may indicate reduced ability to assimilate and complete emotional treatment of a traumatic event (138).

Attributional styles

Another type of cognitive factor that contributes to the explanation of chronic PTSD arises as a result of the research on attributional styles and locus of control of traumatized individuals. Specifically, ample evidence suggests that accepting one's own guilt (linked to beliefs in the internal locus of control) reduces the risk of PTSD. Acceptance of "moderate guilt" is associated with the most significant improvement

liječenja 225 veterana s PTSP-om (145). Među osobama praćenima 6 i 12 mjeseci nakon teške prometne nesreće, oni koji i dalje pokazuju simptome PTSP-a bili su oni koji su pripisivali više od polovice krivnje drugima (146). Slični rezultati dobiveni su u drugom uzorku u kojem je samookrivljavanje bilo povezano s manjim intenzitetom prvotnih simptoma i bržim oporavkom (147). Izvanjska atribucija krivnje nakon teške ozljede glave također je povezana s većom težinom simptoma PTSP-a u opsežnoj Studiji zajednice Velike Britanije (*Great Britain Community Survey*) (148). Međutim, nalazi daljnjih istraživanja ukazivali su na kompleksniji odgovor na pitanje je li samookrivljavanje ili vanjska atribucija krivnje veći rizik za razvoj PTSP-a ostavljajući otvorenom mogućnost da su ta uvjerenja u interakciji s vrstom traumatskog događaja. Kada su pojedinci s internalnim lokusom kontrole suočeni s osobito teškim ishodima, njihovo je blagostanje značajnije narušeno (149). Nadalje, u slučaju PTSP-a nastalog u odgovoru na traumatsko iskustvo silovanja, samookrivljavanje, olakšano negativnim stavovima socijalne okoline, povezano je s kroničnim tijekom ovog poremećaja te dužim oporavkom (150,151). Pesimističan atribucijski stil, koji se očituje stabilnom i globalnom unutarnjom atribucijom uzroka za negativne događaje, povezan je s depresijom (152). Ovakav je stil također bio povezan s PTSP-om u veterana (153) te u adolescenata nakon katastrofa (154). Validacijska studija inventara posttraumatskih kognicija utvrdila je samookrivljavanje jednim od tri faktora koji uspješno identificiraju PTSP, zajedno s negativnim mislima o sebi i svijetu (155). Veći intenzitet simptoma PTSP-a tipičan za žene potencijalna je posljedica povećanog samookrivljavanja (156). Na osnovi svih ovih nalaza istraživači su predložili potencijalno objašnjenje suprotstavljenih nalaza o pripisivanju odgovornosti i PTSP-a. Moguće je da uvjerenja o unutarnjem lokusu kontrole, učinkovita u širokom rasponu situacija u normalnim uvjetima, funkcioniraju drugačije

due to the treatment in 225 veterans with PTSD (145). Among those observed 6 and 12 months after a severe traffic accident, those who still exhibit PTSD symptoms were those who attributed more than half of the blame to others (146). Similar results were obtained in another sample in which self-blame was associated with lower intensity of initial symptoms and faster recovery (147). Excess attribution of guilt after severe head injury is also associated with more severe PTSD symptoms in the Great Britain Community Survey (148). However, further research has suggested a more rational answer to the question whether self-blame or external attribution of guilt poses a higher risk for PTSD development, leaving an open chance for these beliefs to interact with the type of traumatic event. When individuals with internal locus of control are faced with particularly difficult outcomes, their welfare is significantly impaired (149). Furthermore, in the case of PTSD resulting from traumatic rape experience, self-blame, facilitated by negative attitudes of the social environment, is associated with a chronic course of this disorder and longer recovery (150,151). The pessimistic attributional style, which is manifested as a stable and global internal attribution of causes for adverse events, is associated with depression (152). This style was also associated with PTSD in veterans (153) and adolescents after disasters (154). The validation study of The Posttraumatic Cognition Inventory has determined self-blame as one of three factors that successfully identify PTSD, along with negative thoughts about oneself and the world (155). The increased intensity of PTSD symptoms typical for the female gender is a potential consequence of increased self-blame (156). Based on all of these findings, researchers have suggested a potential explanation of various conflicting findings regarding attributional styles and PTSD. It is possible that beliefs about the internal locus of control, effective in a wide range of situations under normal conditions, work differently under extreme conditions. It is possible that in their core lies a

u ekstremnim uvjetima. U podlozi njih možda leži globalna osobina koja je sposobnost pojedinca da prepozna uvjete pod kojima je unutarnja atribucija odgovornosti prikladna, i one u kojima nije. Janoff-Bulman (157) smatra da su pojedinci u najvećem riziku od poremećaja vezanih uz traumu oni čija su uobičajena uvjerenja i pretpostavke o sebi ili svijetu „razdrmana" traumatskim događajem. Ova pretpostavka ukazuje da krhkost uvjerenja može biti dublji faktor rizika te postoje neki empirijski dokazi koji to i podržavaju. Među žrtvama silovanja, napadi u percipiranom sigurnom okruženju doveli su do težih simptoma PTSP-a od napada u procijenjenim opasnim okruženjima (158). Prema Bowmanu i Yehudi (89), na neki način, čini se da je razbijanje vjerovanja u sigurnost okruženja dodatni faktor rizika za PTSP. Čini se kako vjerovanja o pravednosti svijeta, osobnoj učinkovitosti i sigurnosti te unutarnjem lokusu kontrole, funkcioniraju kao faktori rizika kada ih se osoba rigidno pridržava unatoč događaju koji snažno ukazuje na suprotno. Isto tako, fleksibilnije prihvaćanje ideje da neki događaji nisu u skladu s vjerovanjima jedna je od strategija koja potencijalno pruža zaštitu kada je osoba suočena s traumom visokog intenziteta. Moguće je kako je rigidnost samog vjerovanja, više nego bilo koje specifično vjerovanje, najvažniji kognitivni faktor rizika za razvoj kroničnog PTSP-a (89).

Samoefikasnost

Osim vjerovanja i pripisivanja uzročnosti percipirana samoefikasnost konstrukt je koji se pokazao povezanim sa simptomima PTSP-a. Ovaj se konstrukt odnosi na vjerovanja u vlastite sposobnosti organizacije i izvršavanja slijeda radnji potrebnih za izvršenje nekog zadatka ili aktivnosti. Istraživanje Ginzburga i suradnika (159) pokazalo je kako veterani s kroničnim PTSP-om pokazuju značajno manji stupanj percipirane samoefikasnosti od odlikovanih veterana koji nisu razvili poremećaj.

global feature that represents the ability of an individual to recognize the conditions under which the internal attribution of responsibility is appropriate, and those in which it is not. Janoff-Bulman (157) suggests that individuals at the greatest risk of traumatic disorders are those whose common beliefs and assumptions about themselves or the world are "disrupted" by a traumatic event. This assumption suggests that the fragility of one's belief can be a deeper risk factor and there is some empirical evidence to support this. Among rape victims, attacks in a perceived safe environment led to more severe PTSD symptoms than attacks in estimated dangerous environments (158). According to Bowman and Yehuda (89), in some ways, breaking the belief in the safety of one's environment is an additional risk factor for PTSD. It seems that beliefs about the fairness of the world, personal efficacy and safety, and the internal locus of control, work as risk factors when a person is rigidly adhered to them, despite an event that strongly suggests the opposite. Likewise, the more flexible acceptance of the idea that some events are not consistent with one's beliefs is one of the strategies that potentially provide protection when a person faces a high-intensity trauma. It is possible that the rigidity of the belief, more than any specific belief itself, is the most important cognitive risk factor for the development of chronic PTSD (89).

Self-efficacy

In addition to an individual's beliefs and attributions of causality, perceived self-efficacy presents a construct that appears to be associated with the symptoms of chronic PTSD. This construct relates to beliefs in one's own organizational abilities and the execution of the sequence of actions required to carry out a task or activity. The research of Ginzburg and associates (159) showed that veterans with chronic PTSD showed a significantly lower degree of self-efficacy than veterans who received war medals and

Ovi su ispitanici pokazivali manju tendenciju od odlikovanih suboraca da pripišu neuspjeh i uspjeh unutranjim faktorima te veću tendenciju pripisivanja neuspjeha nekontrolabilnim i stabilnim faktorima, što je konzistentno s prethodnim istraživanjima (160,161). Kronični PTSP bio je povezan s lošijim funkcioniranjem na bojištu pri čemu autori ponovo naglašavaju problematiku uzročno-posljedične povezanosti pri interpretaciji ovih nalaza (159). Veterani s kroničnim PTSP-om su također imali slabiji stupanj obrazovanja od suboraca bez PTSP-a te odlikovanih suboraca. Autori ukazuju na mogućnost kako obrazovanje pomaže razvoju osobina važnih za suočavanje i prilagodbu poput osjećaja koherentnosti i samopouzdanja koji su se pokazali povezani s obrazovanjem i sposobnostima prilagodbe (160,161). Faktor dodatnih komplikacija pri oporavku od PTSP-a čini i nerijetka istovremena pojavnost i drugih psihijatrijskih dijagnoza u pojedinca s ovim poremećajem, o čemu će biti govora u idućem odlomku.

Komorbiditet kao faktor održavanja kroničnoga tijeka PTSP-a

Nekoliko je studija utvrdilo kako je klinička depresija jedan od faktora koji imaju utjecaj na tijek i liječenje PTSP-a (164). Bryant i suradnici (165) utvrdili su, primjerice, kako su osobe s PTSP-om koje su vremenom odustale od KBT-a imale izraženije simptome depresije od osoba koje su tretman privele kraju. Slične nalaze utvrdili su i McDonagh i sur. (166), no i Stein, Dickstein, Schuster, Litz i Resnick (167) koji su utvrdili i višu početnu razinu autonomne pobuđenosti u osoba s PTSP-om koje nisu pokazivale poboljšanje stanja zbog KBT tretmana. Međutim, samo pitanje komorbiditeta i tijeka oporavka od PTSP-a očito zahtijeva podrobniju istraživačku provjeru. Na potonje upućuju nalazi istraživanja koja nisu utvrdila značajne veze između simptoma velikog depresivnog po-

did not develop the disorder. These respondents showed a lesser tendency to attribute either failure or success to internal factors and a greater tendency to attribute failure to non-controllable and stable factors than participants without PTSD, consistent with previous research (160,161). Chronic PTSD was associated with inferior functioning on the battlefield, where authors again emphasize the cause-and-effect relationship in the interpretation of these findings (159). Veterans with chronic PTSD also had a lower level of education from non-PTSD veterans and veterans who received war medals. The authors suggest the possibility that education helps to develop features that are important for coping and adaptation, such as feelings of coherence and self-confidence that have shown to be related to education and adaptation skills (160,161). The simultaneous occurrence of other psychiatric diagnoses in individuals with this disorder presents another factor of potential additional complications in PTSP recovery, which will be discussed in the next section.

Comorbidity as a factor for maintenance of chronic PTSD

Several studies have established that clinical depression is one of the factors influencing the course and the treatment of PTSD (164). Bryant et al. (165) found, for example, that persons with PTSD who eventually dropped out of CBT had more pronounced symptoms of depression than the treatment recipients who endured the therapy to the planned end. Similar findings have been found by McDonagh et al. (166), however, Stein, Dickstein, Schuster, Litz, and Resnick (167) also found a higher initial autonomic arousal levels in PTSD patients who did not show improvement post-CBT. The issue of comorbidity and the recovery process from PTSD obviously requires more detailed research. The latter is indicated by research findings that have not established significant links between the symptoms of major depressive disorder and the course of PTSD

remećaja i tijeka PTSP-a (168-170). Istraživanje Gillespiea i suradnika (171) utvrdilo je kako depresija, alkoholizam te panični poremećaj nisu povezani s manje uspješnim ishodima liječenja osoba s PTSP-om. Međutim, stanje osoba s komorbidnim smetnjama zahtijevalo je veći broj susreta u okviru terapije za postizanje sličnoga uspjeha kao s osobama bez komorbidnih smetnji. Na osnovi opisanoga, očiglednom se ističe potreba za temeljitijim istraživanjima ovog područja, posebice za nacrtima koji bi utvrđvali medijatore odnosa između komorbiditeta i terapijskog napretka u osoba s PTSP-om.

Socijalna podrška

Jedan od faktora koji je također istraživan u kontekstu tijeka oporavka od PTSP-a je i uloga različitih aspekata socijalne podrške. Istraživanje Klarića i suradnika (172) na uzorku traumatiziranih mostarskih žena pokazalo je kako je percipirana socijalna podrška prijatelja i radnih kolega niža nego u kontrolnog uzorka. Ova vrsta socijalne podrške pokazala se jačim zaštitnim faktorom za sve razine intenziteta simptoma PTSP-a, jačim čak i od obiteljske podrške. Percipirane niske razine prijateljske podrške pokazale su se jedinim značajnim prediktorom simptoma PTSP-a (172). Andrews i suradnici (173) istraživali su rodne razlike u različitim aspektima percipirane socijalne pomoći na uzorku u muškaraca i žena žrtava nasilnog napada. Iako su pripadnici oba roda mjesec dana nakon traume izvještavali o jednakim stupnjevima pozitivne podrške i zadovoljstva, žene su izvijestile o višim razinama negativnih reakcija od obitelji i prijatelja koje su bile medijator povezanosti roda i simptoma PTSP-a 6 mjeseci nakon napada. Socijalna podrška pri povratku s rata i aktualna socijalna podrška pokazali su se faktorima zaštite i/ili ublažavanja simptoma kroničnog PTSP-a dok ostala istraživanja ukazuju na ulogu socijalne podrške kao zaštitnog faktora u prevenciji nastanka PTSP ili ublažavanju postojećih simptoma (174,175). Naposli-

(168-170). Research by Gillespie et al. (171) found that depression, alcoholism, and panic disorder were not associated with less successful outcome of PTSD treatment. However, the condition of people with comorbid dysfunction required a greater number of treatments within a therapy to achieve similar success as in individuals without comorbid disorders. Based on the above, the need for more thorough research in this area is evident, especially for designs that would determine mediators of the relationship between comorbidity and therapeutic progress in individuals with PTSD.

Social support

One of the factors that has also been explored in the context of the PTSD recovery process is the role of various aspects of social support. The research of Klarić and his associates (172) on a sample of traumatized Bosnian women from the city of Mostar showed that these women perceived lower levels of social support by friends and work colleagues than the comparison group. This type of social support has been shown to be a stronger protective factor for all levels of PTSD symptom intensities, even stronger than family support. Perceived low levels of support from friends have proved to be the only significant predictor of PTSD symptoms (172). Andrews et al. (173) investigated gender differences in different aspects of perceived social support on a sample of male and female victims of violent assaults. Although members of both genders reported a degree of positive support and social support satisfaction after the trauma, women reported higher levels of negative reactions from family and friends that mediated gender related PTSD symptoms 6 months after the attacks. Social support after the return from war and current social support have been shown to act as protective or mitigating factors of chronic PTSD symptoms while other studies point to the role of social support as a protective factor in the prevention of PTSD or the alleviation of

jetku, negativna usmjerenost socijalnoj mreži (engl. *negative network orientation*) definirana negativnim stavovima i očekivanjima pojedinca o koristi socijalne podrške u suočavanju sa stresom, povezana je s intenzitetom simptoma PTSP te je u negativnoj povezanosti s percipiranom socijalnom podrškom (176).

Faktori socioekonomskog statusa

Na kraju, zanimljivo, još nedovoljno istraženo područje u kontekstu trajanja simptoma PTSP-a, su istraživanja uloge socioekonomskog statusa traumatizirane osobe. Točnije, uloge parničenja i traženja odštete u učvršćivanju simptoma PTSP-a, pa čak i njegovog kroničnog oblika. Naime, stupanj u kojem na kronični PTSP utječe parničenje još je uvijek predmet rasprava u literaturi. Tradicionalni pogled, koji se očituje i u Millerovom pojmu „neuroza nezgode“ (131), ukazuje na ulogu motivacije i ponavljanja traumatskog iskustva za sudske sporove u formiranju PTSP-a. Ipak, nedavne studije koje su ispitivale ovu hipotezu daju oprečne rezultate. Dok su neki otkrili vezu između parnice i PTSP-a (177,178) drugi nisu potvrdili takav odnos (16) kao ni između nagodbe i naknade i oporavka (179).

Kao što je spomenuto u uvodu, tri su osnovne hipoteze koje se odnose na opažene razlike u izraženosti simptoma PTSP-a u osoba koje traže financijsku dobit od onih koji ju ne traže. Jedna od hipoteza odnosi se na pretpostavku o stvarnom *stvarne narušenosti funkcioniranja* (*disability hypothesis*). Druga hipoteza je pretpostavka *traženja financijske dobiti* (*finantial gain hypothesis*). Kao što ime predlaže ova hipoteza se odnosi na pretpostavku kako osobe s ovim poremećajem pretjeruju prigodom iskaza o svojim simptomima kako bi maksimizirali financijsku dobit. Ova je hipoteza najviše istraživana pri čemu većina istraživanja upućuje kako petina sudionika obično pokazuje profile na dijagnostičkoj ljestvici MMPI-2 koji ukazuju na

existing symptoms (174,175). In the end, negative network orientation, defined through negative attitudes and expectations of the individual on the benefits of social support in dealing with stress, is associated with higher intensities of PTSD symptoms and is negatively associated with perceived social support (176).

Factors of socioeconomic status

Finally, an interesting, still insufficiently explored area in the context of the duration of PTSD symptoms, refers to the role of socioeconomic status of a traumatized person. Specifically, the role of litigation and compensation seeking in alleviating the symptoms of PTSD and even its chronic forms. Namely, the degree to which chronic PTSD is affected by litigation is still a subject of discussion in literature. The traditional view, also expressed in Miller's concept of "accident neurosis" (131), suggests the role of motivation and repetition of traumatic experience during court disputes in the formation of PTSD, although recent studies that have been examining this hypothesis give contradictory results. The relationship between litigation and PTSD (177,178) did not confirm such a relationship (16) nor the relation between settlement and compensation and PTSD recovery (179). As mentioned in the introduction, there are three basic hypotheses that relate to the observed differences in the expression of PTSD symptoms in people seeking financial gain from those who do not seek it. The first hypothesis refers to the assumption of the presence of actual disability in individuals with PTSD (disability hypothesis). The second refers to the assumption of one's financial gain (financial gain hypothesis). As the name suggests, this hypothesis refers to the assumption that people with this disorder exaggerate when they report their symptoms to maximize their financial gain. This hypothesis is the most explored one, with most studies suggesting that a fifth of the participants usually show profiles on the

ekstremne rezultate na kontrolnim ljestvicama koje ukazuju na pretjerivanje u opisu izraženosti simptoma (22). Posljednja pretpostavka odnosi se na *hipotezu pojačanoga stresa* (*the stress hypothesis*). Navedena pretpostavka odnosi se na pojačavanje simptoma PTSP-a kako je pojedinac prisiljen ponovo proživljavati različite aspekte traumatskog iskustva prolazeći kroz postupke parničenja. Posljednja hipoteza nije znanstveno nikada direktno istražena, no zasada postoje indirektni istraživački nalazi koji impliciraju njezinu utemeljenost (180). Zanimljivi su i nalazi istraživanja koji ukazuju kako financijska dobit nije jedini razlog traženja kompenzacije. Za osobe s PTSP-om spomenuto predstavlja i određenu simboliku koja se odnosi na priznanje vezano uz proživljenu patnju kao i odrješenje od doživljaja krivnje posebno vezano uz žrtve seksualnog nasilja. Također, utvrđeno je kako je visina pojedinčevih prihoda često povezana s doživljajem manjeg značenja same financijske dobiti koja je rezultat parničenja i obrnuto (181).

Temeljitiji nacrti ispitivanja ovog važnog faktora rizika za kroničan tijek ovoga poremećaja svakako su nužni u budućnosti.

ZAKLJUČAK

PTSP je poremećaj čiji je nastanak uvjetovan kompleksnom interakcijom bioloških, psiholoških te socijalnih faktora koji određuju reakciju pojedinca. Dosadašnja istraživanja izdvojila su različite faktore rizika koji pogoduju nastanku ovog poremećaja, a proizlaze iz interakcije različitih osobina ličnosti i strategija suočavanja pojedinca, psihijatrijske povijesti i komorbidnih stanja poput poremećaja ličnosti, vjerovanja i atribucija te bioloških faktora i socijalnih okolnosti. Iako su ova istraživanja opterećena problematikom uzročno-posljedične povezanosti, čini se kako se ipak u nastanku PTSP-a značajnima ističu osobine poput neuroticizma te genetskih nasljednih faktora, prethodna po-

MMPI-2 diagnostic scale that have extreme results on control scales that indicate an exaggeration in the description of symptoms expression (22). The last assumption refers to the stress hypothesis. The aforesaid assumption refers to the increase of the symptoms of PTSD as an individual is forced to re-experience different aspects of a traumatic experience through litigation procedures. The last hypothesis has never been directly researched empirically, but there are indirect research findings that imply its assumptions (180). Interestingly, there are also research findings that indicate that financial gain is not the only reason for seeking compensation. For individuals with PTSD, seeking compensation also presents a certain symbol of acknowledgment regarding the suffering experienced as well as the omission of guilt - especially related to the victims of sexual violence. It has also been found that an individual's income is often negatively associated with estimations of the significance of potential financial gain from litigation and vice versa (181). Further research with more elaborate designs of this important risk factor for the chronic course of PTSD is necessary in the future.

CONCLUSION

PTSD is a result of a complex interaction of biological, psychological and social factors that determine an individual's response. Present research has identified a variety of risk factors that favour the emergence of this disorder, resulting from the interaction of different personality traits and coping strategies of an individual, psychiatric history and comorbid states such as personality disorders, beliefs and attributions as well as biological factors and social circumstances. Although these studies do not provide an answer to the issue of causality, it seems that the emergence of PTSP is significantly alleviated by specific features such as neuroticism and genetic heritability factors, the previous history of anx-

vijest anksioznih poremećaja, odnosno osobine poput povišene anksioznosti kao osobine ličnosti te posebice anksiozna osjetljivost. Značajnima su se posebno pokazali kognitivni faktori poput procjene događaja kao ugrožavajućeg; vjerovanja o osobnoj ranjivosti; pokušaja pripisivanja posebnog značenja događaju te vjerovanja o količini osobne kontrole, a potencijalno i kognitivni stil strepnje. Čini se kako faktori rizika također proizlaze i iz stupnja intelektualnih sposobnosti pojedinca, razine socijalne podrške kao i iz višestrukih iskustava traume za koje postoji pretpostavka kako putem senzitizacije HPA-osi djeluju na smanjenje adaptivne spremnosti odgovora na stresor.

Što se kroničnog tijeka PTSP-a tiče čini se kako stupanj trajanja simptoma varira s obzirom na vrstu traumatskog stresora te je uvjetovan interakcijom višestrukih faktora poput kognitivnog stila, strategija suočavanja, socijalne potpore i mnogih drugih koji potencijalno određuju njegov kronični tijek. Ponovna traumatizacija te intenzitet prvotnog odgovora na traumu prediktori su kroničnog tijeka. Način na koji pojedinac procesira traumu (osjećaj intenzivne i neposredne prijetnje te izbjegavanje) te rigidnost vjerovanja uzdrmanog traumom, više nego bilo koje specifično vjerovanje o sebi, svijetu i okolini, predstavlja istaknuti kognitivni faktor rizika za razvoj kroničnoga tijeka PTSP-a. Naposlijetku, socijalna podrška, pogotovo ona negativna u slučaju ženskoga roda, pokazala se faktorom rizika kroničnoga tijeka ovog poremećaja.

Iako se naizgled radi o veoma temeljito istraženom području, daljnja istraživanja, pogotovo ona longitudinalne prirode nužna su kako bi se u što boljoj mjeri razumjeli faktori u podlozi nastanka i kroniciteta ovog poremećaja. Također, čini se kako je potrebno podrobnije istražiti doprinos procesa parničenja, rentnih faktora te društveno-ekonomskih okolnosti u kontekstu formiranja identiteta *traumatiziranoga* te održavanja ovoga poremećaja.

iety disorders, or features such as elevated trait anxiety and particularly anxiety sensitivity. Special contributors to the development of this disorder appear to be cognitive factors. The latter refer to the assessment of an event as a threatening one; beliefs about personal vulnerability; attempts to attribute special significance to the beliefs about the amount of personal control and potentially – the looming cognitive style. It also appears that risk factors arise from the degree of an individual's intellectual abilities, the level of social support, as well as from multiple trauma experiences, for which there is a hypothesis that HPA-axis sensitization acts to reduce adaptive responsiveness to the stressor. As far as the chronic course of PTSD is concerned, it seems that the duration of the symptoms varies with regard to the type of a traumatic stressor experienced. The PTSD chronic course also appears to be conditioned by the interaction of multiple factors such as cognitive styles, coping strategies, social support, and many others that potentially determine its chronic course. Re-traumatization and the intensity of the first response to trauma are also predictors of the chronic course. The way in which an individual processes trauma (intense and immediate threats and avoidance) and the rigidity of traumatic beliefs, more than any specific beliefs about oneself, the world and the environment, is a prominent cognitive risk factor for the development of chronic PTSD. Ultimately, social support, especially negative in the case of the female gender, has been shown to be the risk factor of the chronic course of this disorder. Although this area of research appears to be thoroughly explored, further research, especially of longitudinal quality, is necessary to better understand the underlying factors of the development and chronicity of this disorder. Also, it seems more necessary to explore the contribution of the process of litigation, compensation seeking factors and socio-economic circumstances in the context of *the traumatized* identity formation and maintenance of this disorder.

LITERATURA / *REFERENCES*

1. Wooley CF. Jacob Mendez DaCosta: medical teacher, clinician, and clinical investigator. Am J Cardiol 1982; 50: 1145-8.
2. World Health Organization. The ICD-11 Classification of Mental and Behavioural Disorders: Clinical Descriptions and Diagnostic Guidelines. Geneva: World Health Organization, 2018. https://icd.who.int/
3. American Psychiatric Association. Diagnostic and statistical manual of mental disorders (5th ed.). Washington, DC: Author, 2013.
4. Edmondson D. An Enduring Somatic Threat Model of Posttraumatic Stress Disorder Due to Acute Life-Threatening Medical Events. Soc Pers Psychol Comp 2014; 8: 118-34.
5. Wikman A, Bhattacharyya M, Perkins-Porras L, Steptoe A. Persistence of posttraumatic stress symptoms 12 and 36 months after acute coronary syndrome. Psychosom Med 2008; 70: 764-72.
6. Ginzburg K, Ein-Dor T. Posttraumatic stress syndromes and health-related quality of life following myocardial infarction: 8-year follow-up. Gen Hosp Psychol 2011K; 33: 565-71.
7. Matsuoka Y, Nakano T, Inagaki M, Sugawara Y, Akechi T, Imoto S i sur. Cancer-related intrusive thoughts as an indicator of poor psychological adjustment at 3 or more years after breast surgery: a preliminary study. Breast Canc Res Treat 2002; 76: 117-24.
8. Mehnert A, Berg P, Henrich G, Herschbach P. Fear of cancer progression and cancer-related intrusive cognitions in breast cancer survivors. Psycho-Oncol 2009; 18: 1273-80.
9. Mahmoodi Z, Dolatian M, Shaban Z, Shams J, Alavi-Majd H, Mirabzadeh A. Correlation between Kind of Delivery and Posttraumatic Stress Disorder. Ann Med Health Sci Res 2016; 6: 356-61. doi:10.4103/amhsr.amhsr_397_15.
10. Garthus-Niegel S, von Soest T, Vollrath ME, Eberhard-Gran M. The impact of subjective birth experiences on post-traumatic stress symptoms: A longitudinal study. Arch Womens Ment Health 2013; 16: 1-10.
11. Andersen LB, Melvaer LB, Videbech P, Lamont RF, Joergensen JS. Risk factors for developing post-traumatic stress disorder following childbirth: A systematic review. Acta Obstet Gynecol Scand 2012; 91: 1261-72.
12. Galatzer-Levy IR, Bryant RA. 636,120 ways to have posttraumatic stress disorder. Persp Psychol Sci 2013; 8: 651-62.
13. Blanchard, EB, Hickling EJ, Barton KA, Taylor AE, Loos WR, Jones-Alexander J. One-year prospective follow-up of motor vehicle accident victims. Behav Res Ther 1996; 34: 775-86.
14. Rothbaum BO, Foa EB, Riggs DS, Murdock TB, Walsh W. A prospective examination of posttraumatic stress disorder in rape victims. J Trauma Stress 1992; 5: 455-75.
15. Epstein RS. Avoidant symptoms cloaking the diagnosis of PTSD in patients with severe accidental injury. J Trauma Stress 1993; 6: 451-8.
16. Mayou R, Tyndel S, Bryant B. Long-term outcome of motor vehicle injury. Psychosom Med 1997; 59: 578-84.
17. Mayou RB, Bryant R. Duthrie, B. Psychiatric consequences of road traffic accidents. BMJ 1993; 307(6905): 647-51.
18. Wisco BE, Marx BP. Miller MW, Krystal EJ, Southwick JH, Pietrzak SM *et al*. A Comparison Of ICD-11 And DSM Criteria for Posttraumatic Stress Disorder In Two National Samples Of U.S. Military Veterans. J Affect Dis 2015; 223: 17-19.
19. Karatzias T, Cloitre M, Maercker A, Kauzlauskus E, Sherlin M, Hyland P *et al*. PTSD And Complex PTSD: ICD-11 Updates On Concept and Measurement in the UK, USA, Germany And Lithuania. Eur J Psychotraumatol 2017; 8(Sup7): 1418103. Doi :10.1080/20008198.2017.1418103.
20. Ehlers A, Mayou, Ra, Bryant B. Psychological Predictors Of Chronic Posttraumatic Stress Disorder After Motor Vehicle Accidents. J Abnorm Psychol 1998; 107: 508-19.
21. Solomon Z, Benbenishty R, Waysman M, Bleich A. Compensation And Psychic Trauma: A Study of Israeli Combat Veterans. Am J Ortho-Psychol 1994; 64: 91-102.
22. Frueh BC,Hamner MB,Cahill SP, Gold PB, Hamlin KL. Apparent symptom Overreporting in Combat Veterans Evaluated for PTSD. Clin Psychol Rev 2000; 20: 853-85.
23. Sayer NA, Murdoch M, Carlson KF. Compensation and PTSD: Consequences for symptoms and treatment. PTSD Res Quart 2007; 18: 1-8.
24. Clark LA, Watson D, Mineska S. Temperament, personality, and the mood and anxiety disorders. J Abnorm Psychol 1994; 103: 103-16.
25. Miller MW, Resick PA. Internalizing and externalizing subtypes in female sexual assault survivors: implications for the understanding of complex PTSD. Behav Ther 2007; 38: 58-71.
26. Gil S. Pre-traumatic personality as a predictor of posttraumatic stress disorder among undergraduate students exposed to a terrorist attack: A prospective study in Israel. Pers Ind Diff 2005; 39: 819-27.
27. Breslau N, Davis GC, Andreski P. Risk factors for PTSD-related traumatic events: A prospective analysis. Am J Psychiatry 1995; 152: 529-35.
28. Talbert FS, Braswell LC, Albrecht JW, Hyer LA, Boudewyns PA. NEO-PI profiles in PTSD as a function of trauma level. J Clin Psychol 1993; 49: 663- 9.
29. Casella L, Motta, RW. Comparison of characteristics of Vietnam veterans with and without Posttraumatic Stress Disorder. Psychol Rep 1990; 67: 595-605.
30. Hyer L, Braswell L, Albrecht B, Boyd S, Boudewyns, P, Talbert S. Relationship of NEO-PI to personality styles and severity of trauma in chronic PTSD victims. J Clin Psychol 1994; 50: 699-707.
31. Sutker PB, Bugg F, Allain AN. Psychometric prediction of PTSD among POW survivors. Psychol Assess 1991; 3: 105-110.

32. Sutker, PB, Davis JM, Uddo M, Ditta SR. War zone stress, personal resources, and PTSD in Persian Gulf War returnees. J Abnorm Psychol 1995; 104: 444-452.
33. O'Toole BI, Marshall RP, Schureck RJ, Dobson M. Posttraumatic stress disorder and comorbidity in Australian Vietnam veterans: Risk factors, chronicity and combat. Austr N Z J Psychiatry 1998; 32: 32-42.
34. Lonigan CJ, Shannon MP, Taylor CM, Finch AJ, Sallee FR. Children exposed to disaster: II. Risk factors for the development of post-traumatic symptomatology. J Am Acad Child Adol Psych 1994; 33: 94-105.
35. Fauerbach JA, Lawrence JW, Schmidt CW, Munster AM, Costa PT. Personality predictors of injury-related posttraumatic stress disorder. J Nerv Ment Dis 2000; 188: 510-17.
36. McFarlane AC. An Australian disaster: The 1983 bushfires. Int J Ment Health 1990; 19: 36-47.
37. McFarlane AC. Risk factors for the acute biological and psychological response to trauma. In: Yehuda R (ed.). Risk factors for posttraumatic stress disorder. Washington, DC: American Psychiatric Press, 1999, 163-190.
38. DiGangi JA, Gomez D, Mendoza L, Jason LA, Keys CB. Pretrauma risk factors for posttraumatic stress disorder: A systematic review of the literature. Clin Psychol Rev 2013; 33: 728-44.
39. Domschke K. Patho-genetics of posttraumatic stress disorder. Psychiatria Danubina 2012; 24: 267-73.
40. Zakin G, Solomon Z, Neria Y. Hardiness, attachment style, and long-term psychological distress among Israeli POWs and combat veterans. Pers Ind Diff 2003; 34:819-829.
41. Thomassen AG, Sigurd W. Hystad SW, Johnsen BH, Johnsen GE, Bartone BT. The effect of hardiness on PTSD symptoms: A prospective mediational approach. Milit Psychol 2018; 30(2): 142-51.
42. Miller MW. Personality and the etiology and expression of PTSD: a three-factor model perspective. Clin Psychol Sci Practice 2003; 10: 373-93.
43. Fedoroff IC, Taylor S, Asmundson GJG, Koch WJ. Cognitive factors in traumatic stress reactions: Predicting PTSD symptoms from anxiety sensitivity and beliefs about harmful events. Behav Cogn Psychother 2000; 28: 5-15.
44. Marshall GN, Miles JNV, Stewart SH. Anxiety Sensitivity and PTSD Symptom Severity are Reciprocally Related: Evidence from a Longitudinal Study of Physical Trauma Survivors. J Abnorm Psychol 2010; 119: 143-50.
45. Stephenson KL, Valentiner DP, Kumpula MJ, Orcutt HK. Anxiety sensitivity and posttrauma stress symptoms in female undergraduates following a campus shooting. J Trauma Stress 2009; 22(6): 489-96.
46. Asmundson GJ, Stapleton JA. Associations between dimensions of anxiety sensitivity and PTSD symptom clusters in active-duty police officers. Cogn Behav Ther 2008; 37:66-75.
47. Collimore KC, McCabe RE, Carleton RN, Asmundson GJ. Media exposure and dimensions of anxiety sensitivity: Differential associations with PTSD symptom clusters. J Anxiety Disord 2008; 22: 1021-8.
48. Bryant RA, Panasetis P. Panic symptoms during trauma and acute stress disorder. Behav Res Ther 2001; 39: 961-6.
49. Feldner MT, Zvolensky MJ, Schmidt NB, Smith RC. A prospective test of anxiety sensitivity as a moderator of the relation between gender and posttraumatic symptom maintenance among high anxiety sensitive young adults. Depress Anxiety 2008; 25: 190-9.
50. Boffa, JW, Norr AM, Raines AM, Albanese BJ, Short NA, Schmidt NB. Anxiety Sensitivity Prospectively Predicts Posttraumatic Stress Symptoms Following a Campus Shooting. Behav Ther 2016; 47: 367-76.
51. Brown KW, Ryan RM. The benefit of being present: mindfulness ans its role in psychological well-being. J Pers Soc Psychol 2003; 84: 822-48.
52. Schoorl M, Van Mil-Klinkenberg L, Van Der Does W. Mindfulness skills, anxiety sensitivity, and cognitive reactivity in patients with posttraumatic stress disorder. Mindfulness 2015; 6(5): 1004-11.
53. Kessler RC, Sonnega A, Bromet E, Hughes M, Nelson CB. Posttraumatic stress disorder in the National Comorbidity Survey. Arch Gen Psychiatry 1995; 52: 1048- 60.
54. Kulka RA, Schlenger WE, Fairbank JA, Hough RL, Jordan BK,Marmar CR *et al*. Trauma and the Vietnam War Generation:Report of Findings from the National Vietnam Veterans Readjustment Study. New York: Brunner/Mazel, 1990.
55. North CS, Nixon SJ, Shariat S, Mallonee S, McMillen JC, Spitznagel EL *et al*. Psychiatric disorders among survivors of the Oklahoma City bombing. J Am Med Assoc 1990; 282: 755-62.
56. King DW, King LA, Foy DW, Gudanowski DM. Prewar factors in combatrelated posttraumatic stress disorder: Structural equation modeling with a national sample of female and male Vietnam veterans. J Cons Clin Psychol 1996; 64: 520-31.
57. Koenen KC, Harley R, Lyons M, Wolfe, J, Simpson JC, Goldberg J *et al*. A twin registry study of familial and individual risk factors for trauma exposure and posttraumatic stress disorder. J Nerv Ment Dis 2002; 190: 209-18.
58. Green BL, Grace MC, Lindy JD, Gleser GC, Leonard A. Risk factors for PTSD and other diagnoses in a general sample of Vietnam veterans. Am J Psychiatry 1990; 147: 729-33.
59. Cederblad, M, Dahlin L. Intelligence and temperament as protective factors for mental health: A cross-sectional and prospective epidemiology study. Eur Arch Psychiatry Clin Neurosci 1995; 245: 11-19.
60. Fergusson DM, Lynskey MT. Adolescent Resiliency to Family Adversity. J Child Psychol Psychiatry 1996; 37: 281-92.
61. Silva RR, Alpert M, Munoz DM. Stress and vulnerability to posttraumatic stress disorder in children and adolescents. Am J Psychiatry 2000; 157: 1229- 1235.
62. Breh DC, Seidler GH. Is peritraumatic dissociation a risk factor for PTSD J Trauma Dissociation 2007; 8(1): 53-69.
63. Birmes P, Brunet A, Carreras D, Ducassé JL, Charlet JP, Lauque D *et al*. The predictive power of peritraumatic dissociation and acute stress symptoms for posttraumatic stress symptoms: a three-month prospective study. Am J Psychiatry 2003; 160: 1337-49.

64. Holeva V, Tarrier N. Personality and peritraumatic dissociation in the prediction of PTSD victims of road traffic accidents. J Psychosom Res 2001; (51): 687-92.
65. Ali T, Dunmore E, Clark D, Ehlers A. The role of negative beliefs in posttraumatic stress disorder: A comparison of assault victims and nonvictims. Behav Cogn Psychother 2002; 30: 249-57.
66. Frommberger U, Stieglitz RD, Straub S, Nyberg E, Schlickewei W, Kuner E *et al*. The concept of „sense of coherence" and the development of posttraumatic stress disorder in traffic accident victims. J Psychosom Res 1990; 46: 343-8.
67. Kaiser CF, Sattler DN, Bellack DR, Dersin J. A conservation of resources approach to a natural disaster: Sense of coherence and psychological distress. J Soc Behav Pers 1996; 11: 459-76.
68. Vickberg SMJ, Duhamel KN, Smith MY. Global meaning and psychological adjustment among survivors of bone marrow transplant. Psycho-Oncology 2001; 10: 29-39.
69. Parrot C, Howes JL. The application of cognitive therapy to posttraumatic stress disorder. In: Vallis TM, JL Howes, Miller PC (eds.). The challenge of cognitive therapy: Applications to nontraditional populations. New York: Plenum, 1991, 85-109.
70. Carlier IVE, Lambers RD, Gersons BPR. The dimensionality of trauma: A multidimensional scaling comparison of police officers with and without posttraumatic stress disorder. Psychiatry Res 2000; 97: 29-39.
71. Dutton MA, Burghard KJ, Perrin SG, Chrestman KR. Battered women's cognitive schemata. J Trauma Stress 1994; 7: 237-55.
72. Benight CC, Antoni, MH, Kilbourn K, Ironson G, Kumar MA, Fletcher MA *et al*. Coping self-efficacy buffers psychological and physiological disturbances in HIV-infected men following a natural disaster. Health Psychol 1997; 16: 248-55.
73. Ferren PM. Comparing perceived self-efficacy among adolescent Bosnian and Croatian refugees with and without posttraumatic stress disorder. J Trauma Stress 1999; 12: 405-20.
74. Alvarez-Conrad J, Zoellner LA, Foa EB. Linguistic predictors of trauma pathology and physical health. Appl Cogn Psychol 2001; 15:159-70.
75. Ehlers A, Mayou RA, Bryant B. Cognitive predictors of posttraumatic stress disorder in children: Results of a prospective longitudinal study. Behav Res Ther 2003; 41: 1-10.
76. Smith K, Bryant RA. The generality of cognitive bias in acute stress disorder. Behav Res Ther 2000; 3: 709-15.
77. Englehard IM, Macklin ML, McNally RJ. Emotion- and intrusion-based reasoning in Vietnam veterans with and without chronic posttraumatic stress disorder. Behav Res Ther 2001; 39: 1339-48.
78. Englehard IM, van den Hout MA, Arntz A, McNally RJ. A longitudinal study of „intrusion-based reasoning" and posttraumatic stress disorder after exposure to a train disaster. Behav Res Ther 2002; 40: 1415-34.
79. Mayou RA, Ehlers A, Bryant B. Posttraumatic stress disorder after motor vehicle accidents: 3-year follow-up of a prospective longitudinal study. Behav Res Ther 2002; 40: 665-75.
80. Solomon SD, Gerrity ET, Muff AM. Efficacy of treatments for posttraumatic stress disorder. J Am Med Assoc 1992; 268: 633-8.
81. Riskind JH. Looming vulnerability to threat: A cognitive paradigm for anxiety. Behav Res Ther 1997; 35: 386-404.
82. Reardon JM, Williams NL. The specificity of cognitive vulnerabilities to emotional disorders: Anxiety sensitivity, looming vulnerability and explanatory style. J Anxiety Disord 2007; 21: 625-43.
83. Williams NL, Shahar G, Riskind JH, Joiner TE. The looming maladaptive style predicts shared variance in anxiety disorder symptoms: Further support for a cognitive model of vulnerability to anxiety. J Anxiety Disord 2005; 19(2): 157-75.
84. Beevers CG, Lee H, Wells TT, Ellis AJ, Telch MJ. Association of predeployment gaze bias for emotion stimuli with later symptoms of PTSD and depression in soldiers deployed in Iraq. Am J Psychiatry 2011; 168(7): 735-41.
85. Elwood LS, Williams NL, Olatunji BO, Lohr JM. Interpretation biases in victims and non-victims of interpersonal trauma and their relation to symptom development. J Anxiety Disord 2007; 21(4): 554-67.
86. Amir N, Coles ME, Foa EB. Automatic and strategic activation and inhibition of threat-relevant information in posttraumatic stress disorder. Cognit Ther Res 2002; 26(5): 645-55.
87. Constans JI. Information-processing biases in PTSD. In: Vasterling JJ, Brewin C (eds). Neuropsychology of PTSD: Biological, Cognitive, and Clinical Perspectives. New York: Guilford Press; 2005, 105-129.
88. Yehuda R, Golier JA, Harvey PD, Stavitsky K, Kaufman S, Grossman RA *et al*. Relationship between cortisol and age-related memory impairments in Holocaust survivors with PTSD. Psychoneuroendocrinology 2005; 30(7): 678-87.
89. Bowman ML i Yehuda R. Risk Factors and the Adversity-Stress Model. In: Rosen, GM (ed.). Posttraumatic Stress Disorder: Issues and Controversies. West Sussex: John Wiley & Sons Ltd, 2008.
90. Bresla, N, Chilcoat HD, Kessler RC, Davis GC. Previous exposure to trauma and PTSD effects of subsequent trauma: Results from the Detroit area survey of trauma. Am J Psychiatry 1999; 156: 902-7.
91. Bremner JD, Southwick SM, Johnson DR, Yehuda R, Charney DS. Childhood physical abuse and combat-related posttraumatic stress disorder in Vietnam veterans. Am J Psychiatry 1993; 150: 235-9.
92. Yehuda R, Resnick HS, Schmeidler J, Yang RK, Pitman RK. Predictors of cortisol and 3-methoxy-4-hydroxy-phenylglycol responses in the acute aftermath of rape. Biol Psychiatry 1998; 43: 855-9.
93. Galea S, Ahern J, Resnick H. Psychological sequelae of the September 11 terrorist attacks in New York City. New Engl J Med 2002; 346: 982-7.
94. Falsetti SA, Resick PA. Causal attributions, depression, and post-traumatic stress disorder in victims of crime. J Appl Soc Psychol 1995; 25: 1027-42.
95. Corneil W, Beaton R, Murphy S, Johnson C, Pike K. Exposure to traumatic incidents and prevalence of posttraumatic stress symptomatology in urban firefighters in two countries. J Occup Health Psychol 1999; 4: 131-41.

96. Wykes T, Whittington R. Prevalence and predictors of early traumatic stress reactions in assaulted psychiatric nurses. J Forensic Psychiatry 1998; 9: 643-58.
97. Aldwin CM, Sutton KJ, Lachman M. The development of coping resources in adulthood. J Pers 1996; 64(4): 837-71.
98. Heinrichs M, Wagner D, Schoch W, Soravia LM, Helhammer DH, Ehlert, U. Predicting posttraumatic stress symptoms from pretraumatic risk factors: A 2-year prospective follow-up study in firefighters. Am J Psychiatry 2005; 162: 2276-86.
99. Southwick SM, Morgan CA, Nicolaou AL, Charney DS. Consistency of memory for combat-related traumatic events in veterans of Operation Desert Storm. Am J Psychiatry 1997; 154: 173-77.
100. Yehuda R, Boisoneau D, Lowy MT, Giller EL. Dose- response changes in plasma cortisol and lymphocyte glucocorticoid receptors following dexamethasone administration in combat veterans with and without posttraumatic stress disorder. Arch Gen Psychiatry 1995; 52: 583-93.
101. Yehuda R, Southwick SM, Krystal JM, Charney DS, Mason JW. Enhanced suppression of cortisol following dexamethasone administration in combat veterans with posttraumatic stress disorder and major depressive disorder. Am J Psychiatry 1993; 150: 83-6.
102. Milad MR, Pitman RK, Ellis CB, Gold AL, Shin LM *et al*. Neurobiological basis of failure to recall extinction memory in posttraumatic stress disorder. Biol Psychiatry 2009; 66(12): 1075-82.
103. Stein MB, Paulus MP. Imbalance of approach and avoidance: the yin and yang of anxiety disorders. Biol Psychiatry 2009; 66(12): 1072-4.
104. Bremner JD, Staib LH, Kaloupek D, Southwick SM, Soufer R, Charney DS *et al*. Neural correlates of exposure to traumatic pictures and sound in Vietnam combat veterans with and without posttraumatic stress disorder: a positron emissiontomography study. Biol Psychiatry 1999; 45: 806-16.
105. Rauch SL, van der Kolk BA, Fisler RE, Alpert NM, Orr SP, Savage CR *et al.* A symptom provocation study of posttraumatic stress disorder using positron emission tomography and script-driven imagery. Arch Gen Psychiatry 1996; 53: 380-7.
106. Shin LM, Kosslyn SM, McNally RJ, Alpert NM, Thompson WL *et al*. Visual imagery and perception in posttraumatic stress disorder: a positron emission tomographic investigation. Arch Gen Psychiatry 1997; 54: 233-41.
107. Bremner JD, Randall P, Scott TM, Bronen RA, Seibyl JP, Soutwick SM *et al*. MRI-based measurement of hippocampal volume in patients with combat-related posttraumatic stress disorder. Am J Psychiatry 1995; 152: 973-81.
108. Gilbertson MW, Shenton ME, Ciszewski A, Kasai K, Lasko NB, Orr SP *et al*. Smaller hippocampal volume predicts pathologic vulnerability to psychological trauma. Nature Neuroscience 2002; 5: 1242-7.
109. Shin LM, Lasko NB, Macklin ML, Karpf RD, Milad MR, Orr SP *et al*. Resting metabolic activity in the cingulate cortex and vulnerability to posttraumatic stress disorder. Arch Gen Psychiatry 2009; 66(10): 1099-107.
110. Shin LM, Bush G, Milad MR, Lasko NB, Brohawn KH, Hughes KC *et al*. Exaggerated activation of dorsal anterior cingulate cortex during cognitive interference: a monozygotic twin study of posttraumatic stress disorder. Am J Psychiatry 2011; 168(9): 979-85.
111. Admon R, Lubin G, Stern O, Rosenberg K, Sela L, Ben-Ami H *et al*. Human vulnerability to stress depends on amygdala's predisposition and hippocampal plasticity. Proc Natl Acad Sci USA 2009; 106(33): 14120-5.
112. Admon R, Leykin D, Lubin G, Engert V, Andrews J, Prussner J *et al*. Stress-induced reduction in hippocampal volume and connectivity with the ventromedial prefrontal cortex are related to maladaptive responses to stressful military service. Human Brain Mapping 2013; 34(11): 2808-16.
113. Kim P, Evans GW, Angstadt M, Ho SS, Sripada CS *et al.* Effects of childhood poverty and chronic stress on emotion regulatory brain function in adulthood. Proc Natl Acad Sci USA 2013; 110: 18442-7.
114. Gee DG, Gabard-Durnam LJ, Flannery J, Goff B, Humphreys KL, Telzer EH *et al*. Early developmental emergence of human amygdala-prefrontal connectivity after maternal deprivation. Proc Natl Acad Sci USA 2013; 110: 15638-43.
115. Hendler T, Admon R. Predisposing Risk Factors for PTSD: Brain Biomarkers. In: Martin C *et al*. (eds). Comprehensive Guide to Post-Traumatic Stress Disorders. Cham: Springer International Publishing AG, 2016.
116. True WR, Lyons MJ. Genetic risk factors for PTSD: A twin study. In: Yehuda R (ed). Risk factors for posttraumatic stress disorder. Washington, DC: American Psychiatric Press, 1990, 61-78.
117. True WJ, Rice J, Eisen SA, Heath AC, Goldberg J, Lyons MJ *et al*. A twin study of genetic and environmental contributions to liability for posttraumatic stress symptoms. Arch Gen Psychiatry 1993; 50: 257-64.
118. Chantarujikapong SI, Scherrer JF, Xian H, Eisen SA, Lyons MJ, Goldberg J *et al.* A twin study of generalized anxiety disorder symptoms, panic disorder symptoms and posttraumatic stress disorder in men. Psychiatry Res 2001; 103: 133-46.
119. Delahanty DL, Raimonde AJ, Spoonster E. Initial posttraumatic urinary cortisol levels predict subsequent PTSD symptoms in motor vehicle accident victims. Biol Psychiatry 2000; 48: 940-7.
120. McFarlane AC, Atchison M, Yehuda R. The acute stress response following motor vehicle accidents and its relation to PTSD. Ann New York Acad Sci 1997; 821: 482-5.
121. Yehuda R. Are glucocortoids responsible for putative hippocampal damage in PTSD? How and when to decide. Hippocampus 2001; 11: 85-9.
122. Boscarino JA. Diseases among men 20 years after exposure to severe stress: Implications for clinical research and medical care. Psychosom Med 1997; 59: 605-14.
123. McNally RJ. Remembering trauma. Cambridge, MA: Harvard University Press, 2003.
124. Pole N. The psychophysiology of posttraumatic stress disorder: A meta-analysis. Psychol Bull 2007; 5: 725-46.

125. Paige SR, Reid GM, Allen MG, Newton JEO. Psychophysiological correlates of PTSD in Vietnam veterans. J Biol Psychiatry 1990; 27(4): 419-30.
126. Orr SP, Metzger LJ, Lasko NB, Macklin ML, Hu FB, Shalev AY *et al.* Physiologic responses to sudden, loud tones in monozygotic twins discordant for combat exposure: Association with posttraumatic stress disorder. Arch Gen Psychiatry 2003; 60: 283-8.
127. Keane TM, Kaloupek DG, Blanchard EB, Hsieh FY, Kolb LC, Orr SP *et al*. Utility of psychophysiological measurement in the diagnosis of posttraumatic stress disorder: Results from a Department of Veterans Affairs' cooperative study. J Consult Clin Psychol 1998; 66: 914-23.
128. Orr SP, Metzger LJ, Pitman RK. Psychophysiology of post-traumatic stress disorder. Psychiatry Clin North Am 2002; 25(2): 271-93.
129. Perkonigg A, Pfister H, Stein MB, Hofler M, Lieb R, Maercher A *et al*. Longitudinal course of posttraumatic stress disorder and posttraumatic stress disorder symptoms in a community sample of adolescents and young adults. Am J Psychiatry 2005; 162(7): 1320-7.
130. Blanchard EB, Hickling EJ, Taylor AE, Loos W. Psychiatric morbidity associated with motor vehicle accidents. J Nerv Ment Dis 1995; 183: 495-504.
131. Koren D, Arnon I, Klein E. Acute stress response and posttraumatic stress disorder in traffic accident victims: a one-year prospective, follow-up study. Am J Psychiatry 1999; 156: 367-73.
132. Davidson JRT, Hughes D, Blazer DG, George LK. Post-traumatic stress disorder in the community: an epidemiological study. Psychol Med 1991; 21: 731-21.
133. Breslau N, Davis GC. Posttraumatic stress disorder in an urban population of young adults: risk factors for chronicity. Am J Psychiatry 1992; 149: 671-5.
134. Green BL, Lindy JD, Grace MC, Glesser GC, Leonard AC, Korol M *et al.* Buffalo Creek survivors in the second decade: stabilityof stress symptoms. Am J Orthopsychiatry 1990; 60: 43-54.
135. Koren D, Arnon I, Klein E. Long term course of chronic posttraumatic stress disorder in traffic accident victims: a three-year prospective follow-up study. Behav Res Ther 2001; 39(12): 1449-58.
136. Harvey AG, Bryant RA. Two-year prospective evaluation of the relationship between acute stress disorder and posttraumatic stress disorder following mild traumatic brain injury. Am J Psychiatry 2000; 157: 626-8.
137. Zlotnick C, Warshaw M, Allsworth J, Shea MT, Keller MB. Chronicity in PTSD and predictors of course of comorbid PTSD in patients with anxiety disorders. J Trauma Stress Disord 1999; 12: 89-100.
138. Rachman S. Emotional processing, with special reference to post-traumatic stress disorder. Int Rev Psychol 2001; 13: 164-71.
139. Horowitz MJ. Stress response syndromes. Northvale, NJ: Jason Aronson, 1986.
140. Shalev AY. Posttraumatic stress disorder among injured survivors of a terrorist attack: predictive value of early intrusion and avoidance symptoms. J Nerv Ment Dis 1992; 180: 505-9.
141. Creamer M, Burgess P, Pattison P. Reaction to trauma: a cognitive processing model. J Abnorm Psychol 1992; 101: 452-9.
142. McFarlane AC. Avoidance and intrusion in posttraumatic stress disorder. J Nerv Ment Dise 1992; 180: 439-45.
143. Ehlers A, Clark DM. A cognitive model of posttraumatic stress disorder. Behav Res Ther 2000; 38: 319-45.
144. Difede J, Barocas D. Acute intrusive and avoidant PTSD symptoms as predictors of chronic PTSD following burn injury. J Trauma Stress 1999; 12: 363-9.
145. Roberts KA. Trauma recovery in military veterans with chronic PTSD: An emphasis on the problem of guilt. A treatment outcomes study. Dissertation Abstracts International 2000; 60(8-B), 4249.
146. Delahanty DL, Herberman HB, Craig KJ, Hayward MC, Fullerton CS, Ursano RJ *et al*. Acute and chronic distress and posttraumatic stress disorder as a function of responsibility for serious motor vehicle accidents. J Consult Clin Psychol 1997; 65: 560-7.
147. Hickling EJ, Blanchard EB, Buckley TC. Effects of attribution of responsibility for motor vehicle accidents on severity of PTSD symptoms, ways of coping, and recovery over six months. J Trauma Stress 1999; 12: 345-53.
148. Huw WW, Evans JJ, Needham P, Wilson BA. Neurological, cognitive andattributional predictors of posttraumatic stress symptoms after traumatic brain injury. J Trauma Stress 2002; 15: 397-400.
149. Janoff-Bulman R, Brickman P. Expectations and what people learn from failure. In : Feather NT (ed.). Expectations and actions. Hillsdale, NJ: Erlbaum, 1982, 207-237.
150. Frazier PA, Mortenson H, Steward J. Coping strategies as mediators of the relations among perceived control and distress in sexual assault survivors. J Couns Psychol 2005; 52: 267-78.
151. Koss MP, Figueredo AJ, Prince RJ. Cognitive mediation of rape's mental, physical, and social health impact: Test of four models in cross-sectional data. J Cons Clin Psychol 2002; 70: 926-41.
152. Peterson C, Seligman ME. Causal explanations as a risk factor for depression: Theory and evidence. Psychol Rev 1984; 91: 347-74.
153. McCormick RA, Taber JI, Kruedelback N. The relationship between attributional style and post-traumatic stress disorder in addicted patients. J Trauma Stress 1989; 2: 477-87.
154. Joseph SA, Brewin CR, Yule W, Williams R. Causal attributions and posttraumatic stress in adolescents. J Child Psychol Psychiatry 1993; 34: 247-53.

155. Foa EB, Ehlers A, Clark DM. The Posttraumatic Cognitions Inventory (PTCI): Development and validation. Psychol Assess 1999; 11: 303-14.
156. Tolin DF, Foa EB. Gender and PTSD: A cognitive model. In: Kimerling R, Ouimette P (ed). Gender and PTSD. New York: Guilford Press, 2002, 76-97.
157. Janoff-Bulman R. Shattered assumptions: Towards a new psychology of trauma. New York: Free Press, 1992.
158. Cascardi, M, Riggs DS, Hearst-Ikeda D, Foa EB. Objective ratings of assault safety as predictors of PTSD. J Interpers Viol 1996; 11: 65-78.
159. Ginzburg K, Solomon Z, Dekel R, Juvan N. Battlefield functioning and chronic PTSD: associations with percieved self efficacy and causal attribution. Pers Ind Diff 2003; 34: 463-76.
160. Orr SP, Claiborn JM, Altman B, Forgue DF,de Jong JB, Pitman RK, & Herz LR. Psychometric profile of posttraumatic stress disorder, anxious, and healthy Vietnam veterans: correlations with psychophysiologic responses. J Consult Clin Psychol 1990; 58: 329-35.
161. Regehr C, Cadell S, Jansen K. Perceptions of control and long-term recovery from rape. Am J Orthopsychiatry 1999; 69: 110-15.
162. Solomon Z, Benbenishty R, Neria Y, Abramowitz M, Ginzburg K, Ohry A. Assessment of PTSD: validation of the revised PTSD Inventory. Isr J Psychiatry Rel Sci 1993; 30: 110-15.
163. Antonovsky A. Unraveling the mystery of health. San Francisco: Jossy-Bass health series, 1987.
164. Taylor S, Fedoroff IC, Koch WJ, Thordarson DS, Fecteau G, Nicki RM. Posttraumatic stress disorder arising after road traffic collisions: Patterns of response to cognitive-behavior therapy. J Consult Clin Psychol 2001; 69: 541-51.
165. Bryant RA, Moulds ML, Guthrie RM, Dang ST, Nixon RDV. Imaginal exposure alone and imaginal exposure with cognitive restructuring in treatment of posttraumatic stress disorder. J Consult Clin Psychol 2003; 71: 706-12.
166. McDonagh A, Friedman M, McHugo G, Ford J, Sengupta A, Mueser K *et al*. Randomized trial of cognitive-behavioral therapy for posttraumatic stress disorder in adult female survivors of childhood sexual abuse. J Consult Clin Psychol 2005; 73: 515-24.
167. Stein NR, Dickstein BD, Schuster J, Litz BT, Resick PA. Trajectories of response to treatment for posttraumatic stress disorder. Behav Ther 2012; 43: 790-800.
168. Ehlers A, Mayou RA, Bryant B. Psychological predictors of chronic posttraumatic stress disorder after motor vehicle accidents. J Abnorm Psychol 1998; 107: 508-19.
169. Aderka IM, Foa EB, Applebaum E, Shafran N, Gilboa-Schechman E. Direction of influence between posttraumatic and depressive symptoms during prolonged exposure therapy among children and adolescents. J Consult Clin Psychol 2011; 79: 421-5.
170. Aderka IM, Gillihan SJ, McLean CP, Foa EB. The relationship between posttraumatic and depressive symptoms during prolonged exposure with and without cognitive restructuring for the treatment of posttraumatic stress disorder. J Consult Clin Psychol 2013; 81: 375-82.
171. Gillespie K, Duffy M, Hackmann A, Clark DM. Community-based cognitive therapy in the treatment of post-traumatic stress disorder following the Omagh bomb. Behav Res Ther 2002; 40: 345-57.
172. Klarić M, Frančišković T, Klarić B, Krešić M, Grković J, Diminić Lisica I *et al*. Social support and PTSD symptoms in war-traumatized women in Bosnia and Herzegovina. Psychiatria Danubina 2008; 20(4): 466-73.
173. Andrews B, Brewin CR, Rose S. Gender, social support, and PTSD in victims of violent crime. J Trauma Stress 2003; 16(4): 421-7.
174. Brewin CR, Andrews B, Valentine JD. Meta-analysis of risk factors for posttraumatic stress disorder in trauma exposed adults. J Consult Clin Psychol 2000; 68 (5): 748-58.
175. Ozer EJ, Best SR, Lipsey TL, Weiss DS. Predictors of posttraumatic stress disorder and symptoms in adults: a meta analysis. Psychol Bull 2003; 129 (1): 52-73.
176. Clapp JD, Gayle Beck J. Understanding the relationship between PTSD and social support: the role of negative network orientation. Behav Res Ther 2009;47(3): 237-44.
177. Blanchard EB, Hickling EJ, Taylor AE, Loos WR, Gerardi J. Psychological morbidity associated with motor vehicle accidents. Behav Res Ther 1994; 32: 283-90.
178. Bryant RA, Harvey AG. Avoidant coping style and post-traumatic stress following motor vehicle accidents. Behav Res Ther 1995; 33: 631-5.
179. Bryant B, Mayou RA, Lloyd-Bolstock S. Compensation claim following road accidents: A six year follow-up study. Med Sci Law 1997; 37: 326-36.
180. Sayer, NA, Spoont M, Nelson DB, Nugent S. Development and Psychometric Properties of the Disability Application Appraisal Inventory. Psychol Assess 2004; 16: 192-6.
181. Sayer NA, Spoont M, Nelson D. Veterans seeking disability benefits for post-traumatic stress disorder: Who applies and the self-reported meaning of disability compensation. Soc Sci Med 2004; 58: 2133-43.