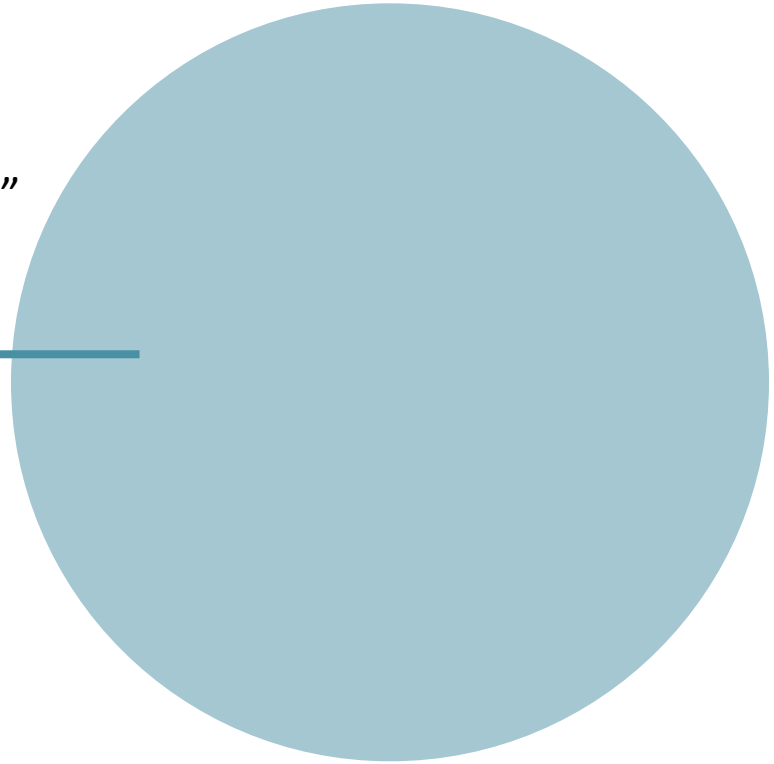
KOMPETENCECENTER FOR TRAUMER

Dissociation som psykiatrisk fænomen

Af Cæcilie Böck Buhmann, speciallæge i psykiatri

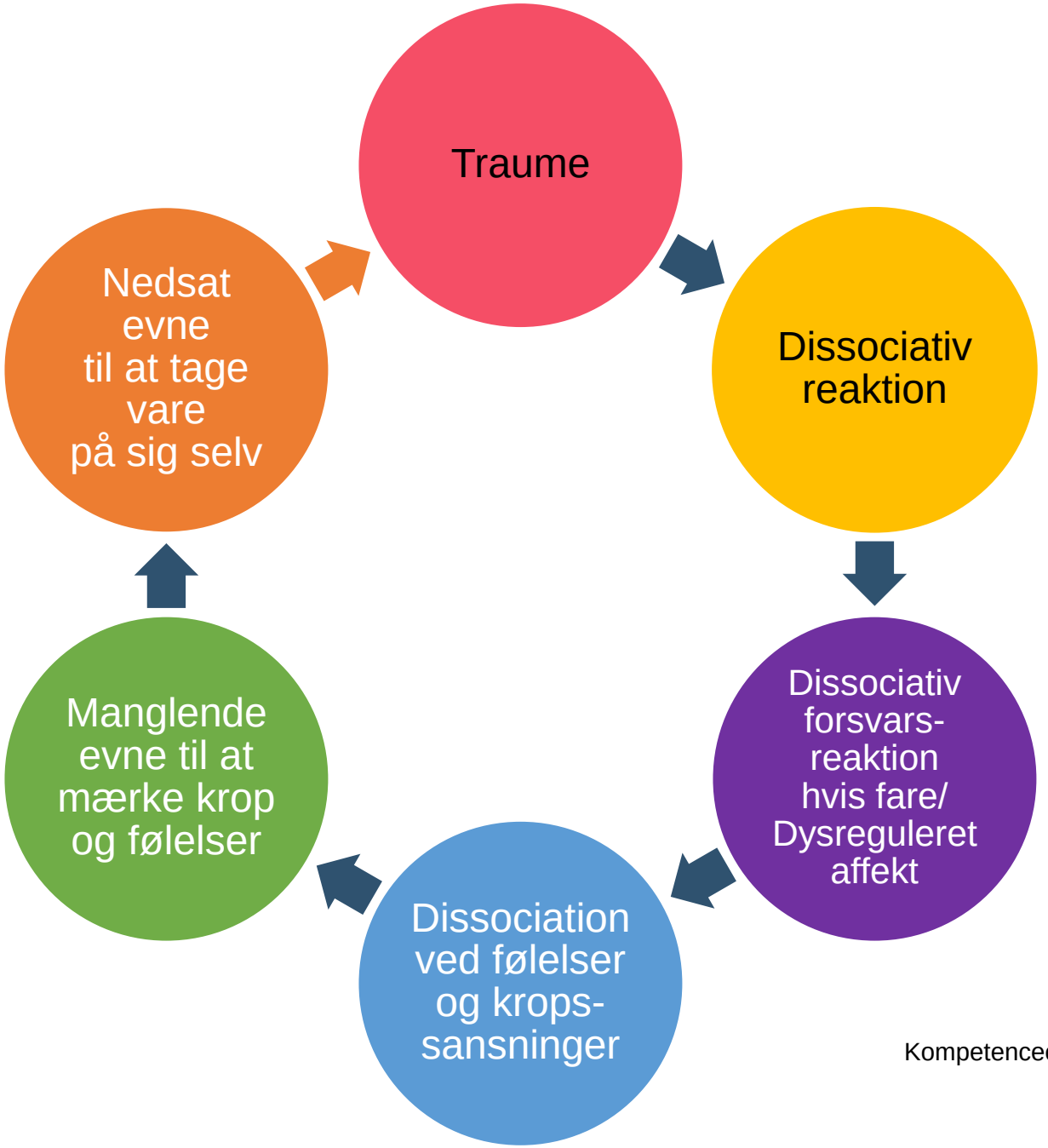
DEFINITION AF DISSOCIATION

”Delvist eller fuldstændigt tab af den normale integration mellem erindring, identitetsbevidsthed, umiddelbare sanseoplevelser og beherskelsen af den legemlige motorik”
ICD-10



- Et symptom
- En gruppe af lidelser
- En forsvarsmekanisme

DISSOCIATION
”En ond cirkel”



Oversigt af Buhmann,
Kompetencecenter for Traumer 2022

DISSOCIATION SOM BEGREB

- Diagnosegruppe (F44) – stilles yderst sjældent
- Ny Dissociativ PTSD diagnose i DSM-5 i 15-30% af tilfælde af PTSD (Wolf 2022)
- Indgår i forståelsen af genoplevelsessymptomer i PTSD
- Indgår i ny kompleks ICD-10 diagnose (affektregulering) i ICD-11
- Kan være associeret med ”uhensigtsmæssig mestring” som selvskade (se fx ICD-11)
- Kan som symptom indgå i andre diagnostiske kategorier (angst, psykose m.m.)
- Kan være diagnostisk svær at skelne fra psykose ved co-morbiditet

DISSOCIATIVE SYMPTOMER OG DIAGNOSER

Diagnose fx

- PNES (psykogene non-epileptiske kramper)
- DID (dissociative Identity Disorder)
- Dissociativ Fugue
- Dissociativ amnesi
- Dissociativ bevægelsesforstyrrelse
- Dissociativ stupor

- Kompleks PTSD

Symptom (se ratings som DSS og DES) fx

- ✓ Depersonalisation (ude af kroppen følelse, afkoblet fra sig selv)
- ✓ Derealisation (verden føles uvirkelig)
- ✓ Amnesi: Har glemt episoder eller perioder i ens liv
- ✓ Kan ikke mærke smerte – kan ikke mærke noget
- ✓ Ufrivillige bevægelser, kramper eller lammelser uden strukturel årsag
- ✓ Kan ikke huske, hvordan man er kommet fra A-B
- ✓ Frosset – ukontaktbar men ved bevidsthed

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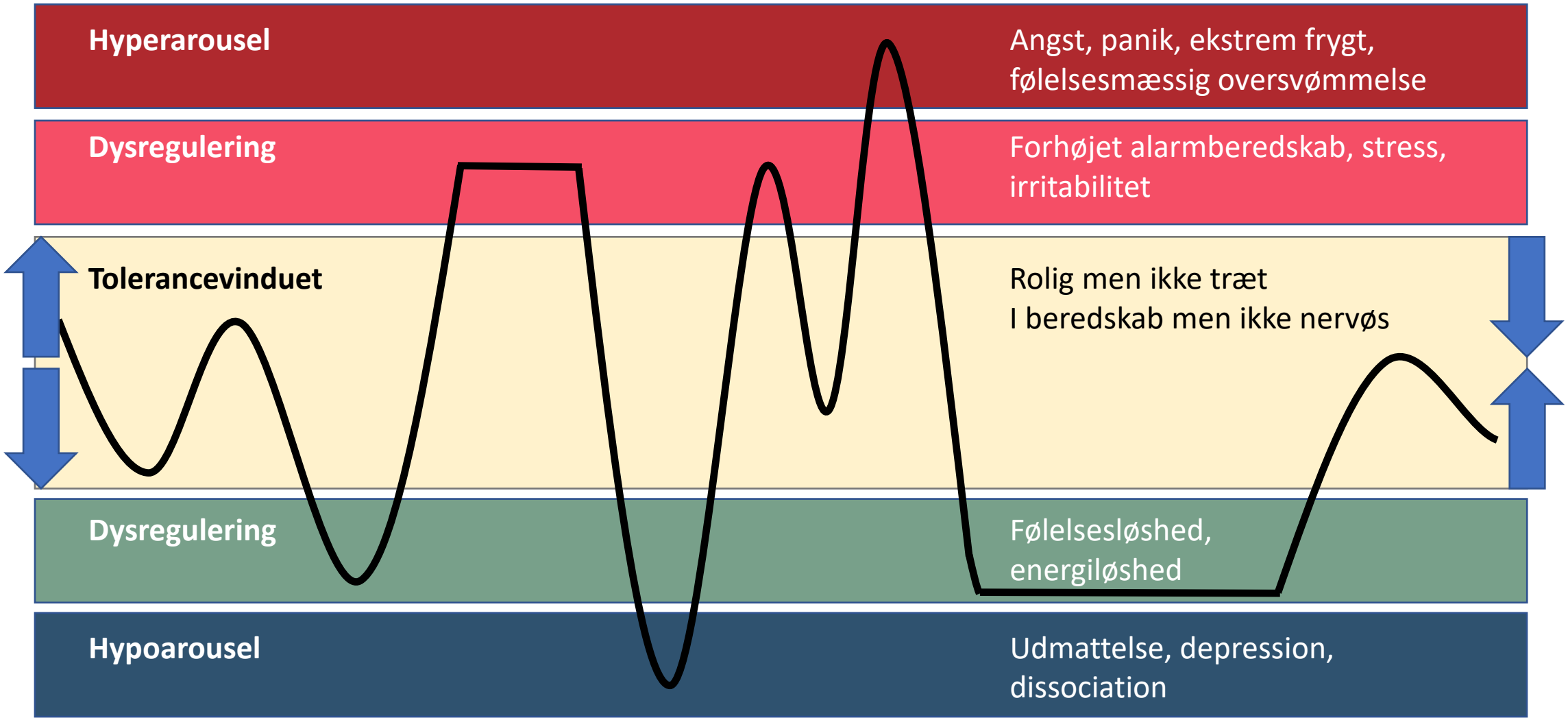
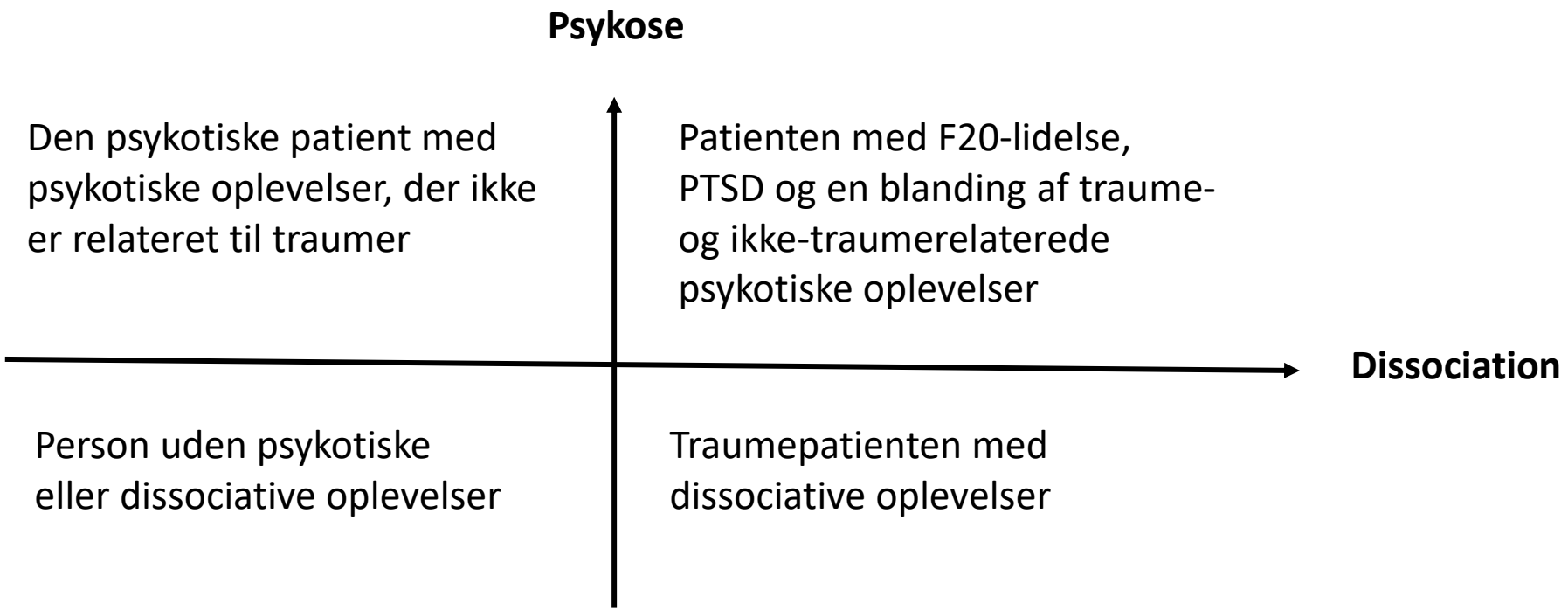
TOLERANCEVINDUET & DISSOCIATION



Dan Siegel 1999

PSYKOSE / DISSOCIATION



Et spektrum: Vagtsomhed → Mistillid → Mistroiskhed → Paranoia

Dissociation – Fænomenologisk perspektiv

JULIE NORDGAARD

Konceptuelt uklart på flere niveauer

01

Hvad dissocierer?

Personligheden, bevidstheden, tankerne?

02

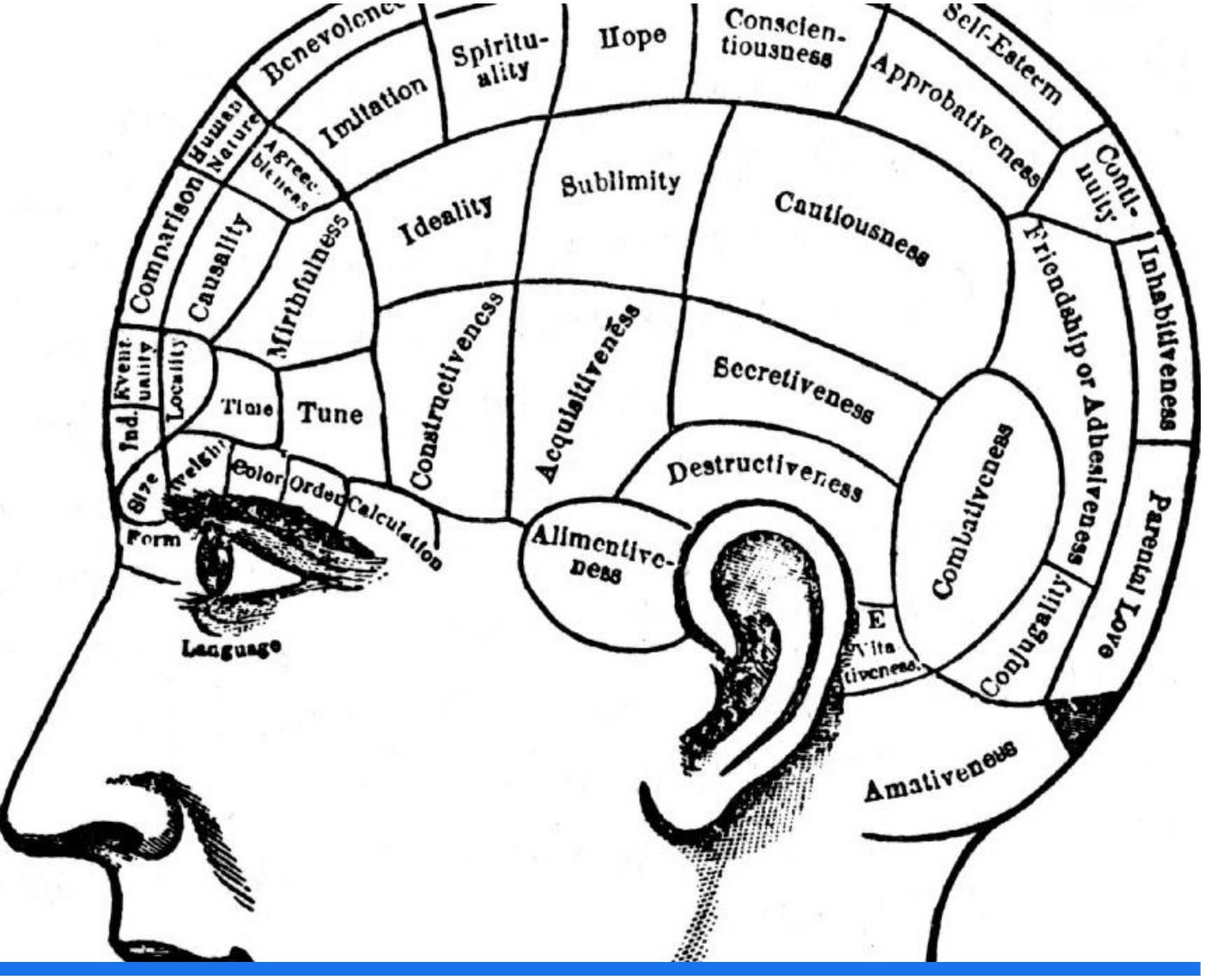
Niveau?

Deskriptivt eller forklarende?

03

Nosologisk?

Et symptom eller en gruppe af tilstande



01. Hvad er det som dissocieres?

- Personlighed eller bevidsthed?
- Antager at bevidstheden består af moduler som kan splittes
- Bevidsthed er ikke et object
- Hvem eller hvad laver afspaltningen?
- Kan det fraspaltede smeltes sammen med det øvrige igen?

02. Niveau?

Dissociation bruges både

- Deskriptivt: fx amnesi, søvngængeri, depersonalisation, derealisation, hallucinatoriske oplevelser
- Forklarende: den proces hvorved adfærd, tanker og følelse kan blive splittet fra hinanden – almindeligvis den psykodynamiske at noget er for svært og derfor spaltes fra

Anses for at være funktionelle, men er det korrekt? Dissociative fænomener også ses ved forskellige neurologiske tilstand fx partielle, komplekse kramper og så er der bevidsthedsindsnævring (ikke fuld bevidsthed)

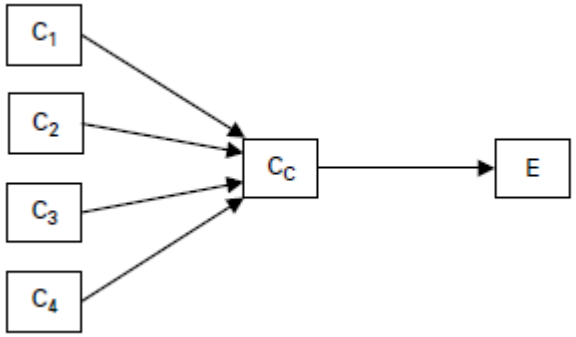
03. Nosologisk?

I de diagnostiske manualer er dissociation udover at være en forklarende term både et symptom og en gruppe af tilstande fx:

- ICD-10 under dissociative og somatoforme tilstande at termen "hysteri" ikke bruges, men at man har foretrukket at samle lidelserne som tidligere blev kaldt hysteri under "dissociation"

Sigurd Wiingaard Uldall
Dissociation fra et neurobiologisk perspektiv

Dissociation as nonconscious or nonintergrated mental modules or systems
Dissociation as an alteration in consciousness wherein disconnection/disengagement from the self or the environment is experienced
Dissociation af a defense mechanism



Psychological Medicine

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Original Article

A neurostructural biomarker of dissociative amnesia: a hippocampal study in dissociative identity disorder

Lora I. Dimitrova[1,2,*], Sophie L. Dean[3,*], Yolanda R. Schlumpf[4,5,†], Eline M. Vissia[6,†], Ellert R. S. Nijenhuis[5], Vasiliki Chatzi[7], Lutz Jäncke[4,8], Dick J. Veltman[2], Sima Chalavi[9] and Antje A. T. S. Reinders[1]

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*These authors contributed equally to this work

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Backgr nostic

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Correspondence

[‡]This article has been updated since its original publication. A notice detailing this change can be found here: https://doi.org/10.1017/S0033291722002306

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The elusive search for a biomarker of dissociative amnesia: a reaction to Dimitrova et al. (2021)[‡]

R. J. C. Huntjens[1], H. Otgaar[2,3], G. H. M. Pijnenborg[4] and I. Wessel[1]

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Dimitrova et al. (2021) claimed to have found a biomarker for dissociative amnesia in DID: the volume of the hippocampal subregion CA1. In this commentary, we argue that their claims are overstated.

First, the authors claimed to have investigated the neurobiology of dissociative *amnesia*. However, as an index of dissociative amnesia, they used the *subjective* amnesia scale scores of the DES, a self-report instrument of dissociative symptoms. An *objective* cognitive measure of dissociative amnesia was not included. Additionally, previous studies (i.e. around 15 publications) that did include such an objective measure were not discussed and these studies found (memory) *transfer* of neutral, self-referential/autobiographical, and trauma-related information between identities in DID rather than amnesia (e.g. Huntjens, Verschuere, & McNally, 2012; Marsh et al., 2018).

Second, are their results specific to DID? The authors concluded that 'the association of

ORIGINAL ARTICLES

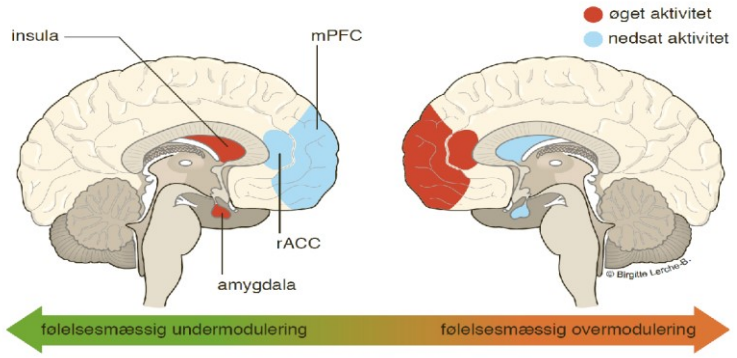
Brain Activation during Script-Driven Imagery Induced Dissociative Responses in PTSD: A Functional Magnetic Resonance Imaging Investigation

Ruth A. Lanius, Peter C. Williamson, Kristine Boksman, Maria Densmore, Madhulika Gupta, Richard W.J. Neufeld, Joseph S. Gati, and Ravi S. Menon

Background: *The goal of this study was to examine the neuronal circuitry underlying dissociative responses to traumatic script-driven imagery in sexual-abuse-related posttraumatic stress disorder (PTSD). Pilot studies in our laboratory have shown that PTSD patients had very different responses to traumatic script-driven imagery. Approximately 70% of patients relived their traumatic experience and showed an increase in heart rate while recalling the traumatic memory (Lanius et al 2001). The other 30% of patients had a dissociative response with no concomitant increase in*

Introduction

Dissociation is a common feature of posttraumatic stress disorder (PTSD). Dissociation often involves a disruption in the usually integrated function of consciousness, memory, identity, or perception of the environment. Acute dissociative responses to psychologic trauma have been found to predict the later development of chronic PTSD (Bremner et al 1992, 1997; Koopman et al 1994; Marmar et al 1994; Shalev et al 1996). Moreover, indi-



Key Words: PTSD, neuroimaging, psychophysiology, anterior cingulate, dissociation, temporal lobe

phenomena are not well understood, a recent study examined brain glucose metabolism in DSM-IV depersonalization disorder. Significantly lower metabolic activity was observed in the right superior and middle temporal gyri (areas 21, 22). Increased metabolic activity was seen in parietal areas (area 7B, 39) and left occipital cortex (area 19). Dissociation and depersonalization scores were significantly positively correlated with metabolic activity in area 7B. The authors suggested that depersonalization is associated with functional abnormalities in sequential hierarchical areas of the sensory cortex as well as in areas responsible for multimodal sensory integration (BA7B;

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PII S0006-3223(02)01367-7

reactivity between high and low dissociators.[185] These findings suggest that our current understanding of the role of dissociation in relation to arousal is very limited, and there is insufficient evidence at this point in time to confidently conclude that a dissociative subtype of PTSD is characterized by any particular form of emotional responding.

Friedman, M. J., Resick, P. A., Bryant, R. A., & Brewin, C. R. (2011). Considering PTSD for DSM-5.

Adding PTSD Subtypes

Two PTSD subtypes were introduced in *DSM-5*, both having met the high threshold for evidence supporting their inclusion. Work on the dissociative subtype was done by the Trauma Sub-Work Group, whereas that supporting the preschool subtype was done primarily by the Child and Adolescent Work Group. Regarding the dissociative subtype, findings with functional magnetic resonance imaging (fMRI) among individuals with PTSD and dissociative symptoms, showed a reversal of the usual fMRI pattern, marked by excessive prefrontal cortical activity associated with reduced activity in the amygdala (Lanius, Brand, Vermetten, Frewen, & Spiegel, 2012). A second line

Friedman, M. J. (2013). Finalizing PTSD in DSM-5: getting here from there and where to go next. *Journal of Traumatic Stress*, *26*(5), 548–556.

European Journal of Trauma & Dissociation 5 (2021) 100081

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Opinion Paper

Problems with the dissociative subtype of posttraumatic stress disorder in DSM-5

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Dissociative amnesia
Dissociative subtype of PTSD

ABSTRACT

The diagnostic criteria set for posttraumatic stress disorder (PTSD) has gradually evolved from DSM-III, to DSM-IV, to DSM-5. Besides a broadening of the event criterion for PTSD, the symptom domain now involves many different brain circuits and types of symptoms, including: fear and anxiety; anger and aggression; negative cognition and mood; hypo-arousal; and dissociative symptoms. The dissociative subtype of PTSD in DSM-5 is defined by the presence of depersonalization or derealization. However, the diagnostic criteria for PTSD also include dissociative flashbacks and dissociative amnesia. If these symptoms were included in the definition of the dissociative type of PTSD in future editions of the manual, then most cases of PTSD would be dissociative in nature, and non-dissociative cases would be a minority subtype. There does not appear to be any sound conceptual reason for excluding amnesia and flashbacks from the criteria for dissociative PTSD.

Posttraumatic Stress Disorder (PTSD) i
diagnosis in DSM-III (American Psychiatric
Most of the work leading up to the inclusio
came out of the United States Veterans Admi
work with Vietnam veterans. As a result, t
traumatizing event required for the diagnosis
of death or dismemberment. In DSM-III, a
(American Psychiatric Association, 1994), PI
one of the Anxiety Disorders. The emphas
criteria for PTSD, was on fear and anxiet
specialists in PTSD argued that PTSD should
Circuitry Disorder in DSM-5 (Andrews, Cl
2009). This was peculiar, since PTSD is clear
driven dysregulation of the autonomic n
therefore one would expect the fight respons
to be as prominent as the flight response (fea
there no proposal for PTSD to be classifie
Circuitry Disorder in DSM-5?

In any case, gradually, in DSM-IV and
traumatic event criterion for PTSD has beer
include exposure to sexual violence, witnessi
and learning that that a traumatic event occu
member or close friend. In conjunction wit

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https://doi.org/10.1016/j.ejtd.2018.08.005
2468-7499/

Molecular Psychiatry www.nature.com/mp

ARTICLE

Longitudinal volumetric evaluation of hippocampus and amygdala subregions in recent trauma survivors

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The hippocampus and the amygdala play a central role in post-traumatic stress disorder (PTSD) pathogenesis. While alternations in volumes of both regions have been consistently observed in individuals with PTSD, it remains unknown whether these reflect pre-trauma vulnerability traits or acquired post-trauma consequences of the disorder. Here, we conducted a longitudinal panel study of adult civilian trauma survivors admitted to a general hospital emergency department (ED). One hundred eligible participants (mean age = 32.97 ± 10.97, $n = 56$ females) completed both clinical interviews and structural MRI scans at 1-, 6-, and 14-months after ED admission (alias T1, T2, and T3). While all participants met PTSD diagnosis at T1, only $n = 29$ still met PTSD diagnosis at T3 (a "non-Remission" Group), while $n = 71$ did not (a "Remission" Group). Bayesian multilevel modeling analysis showed robust evidence for smaller right hippocampus volume ($P+$ of ~0.014) and moderate evidence for larger left amygdala volume ($P+$ of ~0.870) at T1 in the "non-Remission" group, compared to the "Remission" group. Subregion analysis further demonstrated robust evidence for smaller volume in the subiculum and right CA1 hippocampal subregions ($P+$ of ~0.021–0.046) in the "non-Remission" group. No time-dependent volumetric changes (T1 to T2 to T3) were observed across all participants or between groups. Results support the "vulnerability trait" hypothesis, suggesting that lower initial volumes of specific hippocampus subregions are associated with non-remitting PTSD. The stable volume of all hippocampal and amygdala subregions does not support the idea of consequential, progressive, stress-related atrophy during the first critical year following trauma exposure.

Molecular Psychiatry (2023) 28:657–667; https://doi.org/10.1038/s41380-022-01842-x

INTRODUCTION

Numerous studies have examined the relationship between post-traumatic stress disorder (PTSD) and the hippocampus and the amygdala, as both regions are implicated in the disorder's pathogenesis and pathophysiology [1]. The hippocampus is involved in providing contextual memory for emotion-related processes, whereas the amygdala mediates fear learning, extinction, and regulation [2–4]. Prior literature typically reported smaller hippocampus volume in PTSD patients [5–7], while evidence of altered amygdala volume is more equivocal, with findings of no difference, smaller or larger amygdala volume in PTSD patients compared to controls [8, 9]. A major unresolved question is whether these altered volumes reflect a pre-trauma vulnerability trait [10, 11], an outcome of the disorder's persistence associated distress [12, 13], or a combination of both [14].

Previous volumetric findings in PTSD populations did not provide a clear answer to the vulnerability vs. consequence debate for several reasons. First, most studies rely on cross-sectional designs and examine chronic PTSD samples [15], thus cannot disentangle predisposed from acquired volume abnormalities. The few longitudinal PTSD studies conducted to date [16–18] reported no change over time in either hippocampus or amygdala volumes, but those are limited by relatively low sample sizes ($27 \leq n \leq 44$) and follow-up durations (3- to 6-months post-trauma), that may not capture longer-term modifications of these regions [19]. Finally, studies typically examined only the whole hippocampus and/or amygdala, usually due to limited resolution and accuracy to segment their different subregions [20].

Yet, the hippocampus and amygdala consist of functionally and cytoarchitecturally distinct substructures that may play a different role in PTSD development and maintenance [21, 22]. Several animal studies described structural plasticity in specific subregions of these structures after stress exposure [23], but the translational value of these findings is limited by the lack of an adequate model system for PTSD. On the other hand, not many human studies investigated volumetric abnormalities of amygdala and hippocampus subregions in PTSD, and findings remain inconclusive [24–26]. To date, only two longitudinal studies examined subregion-specific

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In Press, Journal Pre-proof What's this?

Archival Report

Amygdala hyperactivity in PTSD: disentangling predisposing from consequential factors in a prospective longitudinal design

Lycia D. de Voogd [1 2 3 4 *], Mahur M. Hashemi [1 2 *], Wei Zhang [5], Reinoud Kaldewaij [6 7], Saskia B.J. Koch [1], Vanessa A. van Ast [8 9], Floris Klumpers [1 2 #], Karin Roelofs [1 2 #]

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https://doi.org/10.1016/j.biopsych.2025.02.894

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Abstract

Background

Substantial inter-individual differences exist in the vulnerability to develop post-traumatic stress disorder (PTSD) symptoms following trauma exposure. Identification of neurocognitive risk markers for PTSD-symptoms could aid early assessment and identification of preventive intervention-targets for PTSD, particularly in high-risk

Sigurd Wiingaard Uldall

nature mental health

Perspective

https://doi.org/10.1038/s44220-024-00324-z

Why reliving is not remembering and the unique neurobiological representation of traumatic memory

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Check for updates

Breanne E. Kearney & Ruth A. Lanius

Anecdotally, the difference between autobiographical and traumatic memory is clear; one is remembered, the other vividly relived. Further distinction can be drawn between past-centered traumatic recurrences and present-centered trauma-related intrusions. While some argue sensorimotor representations to be building blocks of all memory forms, traumatic memory seems to haunt in the form of unintegrated sensations and actions. This Perspective hypothesizes a neurobiological delineation between these memory forms, where traumatic memory is uniquely characterized by alterations to lower-level and primary sensorimotor processes. The proposed demarcation has implications for legal systems, which assume interrogations of autobiographical and traumatic memory can be methodologically identical. Clinically, first-line treatments for post-traumatic stress disorder have yet to explicitly address sensorimotor processes, where up to half of individuals are non-responsive and 20% drop out. We are left with urgent needs to consider sensorimotor fragmentation of traumatic memory and advance interventions that assimilate these fragments into a past-contextualized autobiography.

Traumatic memory has been anecdotally portrayed as relived sensations and re-enactments for well over a century[1]. These vivid yet fragmented perceptual and motoric traumatic re-experiences are devoid of temporal context or verbal narrative; they exist as the unintegrated vestiges of overwhelming events and re-emerge as a present-day experience[2]. Brewin and colleagues[3] hypothesized that these sensory and motoric traces are neurobiologically represented among lower-level perceptual systems cut off from higher-order, verbally accessible cognitive representations that typically provide context. Without access to a cognitively constructed perspective, it was proposed that these low-level perceptual systems remain siloed and impervious to deliberate reform, contributing to the unchanged quality of traumatic memory[3–5]. An important distinction should be made between this phenomenon of reliving a past traumatic event, often termed flashbacks or dissociative flashbacks, and trauma-related intrusive memories or intrusions[6]. While intrusions similarly emerge involuntarily, they spare one's sense of present-day reality. The former, herein termed traumatic memory for simplicity, are past-centered, immersive, temporally disjointed and profoundly disorienting. The trauma is re-embodied and the past becomes ever-present[7,8]. Table 1 distinguishes between these different, yet often conflated, memory forms.

We here provide several illustrative examples of traumatic memory. A veteran who witnessed their comrade killed in an improvised-explosive-device blast while deployed in Afghanistan has flashbacks to the event in its sensorial (smelling smoke and dust, seeing their comrade's grimacing face, sensing internal anguish and terror) and motoric (running to take cover) elements anytime they hear fireworks. An individual who endured severe childhood maltreatment re-experiences visceral anguish and rapid heartbeat when a critical glance from their supervisor at work suddenly turns into a visual depiction of their abusive parent's face. A survivor of childhood sexual abuse is haunted by flashbacks to their childhood

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Article

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Neural patterns differentiate traumatic from sad autobiographical memories in PTSD

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Ofer Perl[1,2,3], Or Duek[4,5,6], Kaustubh R. Kulkarni[2,3], Charles Gordon[2,3], John H. Krystal[4,5], Ifat Levy[7,8], Ilan Harpaz-Rotem[4,5,6] & Daniela Schiller[1,2,9]

For people with post-traumatic stress disorder (PTSD), recall of traumatic memories often displays as intrusions that differ profoundly from processing of 'regular' negative memories. These mnemonic features fueled theories speculating a unique cognitive state linked with traumatic memories. Yet, to date, little empirical evidence supports this view. Here we examined neural activity of patients with PTSD who were listening to narratives depicting their own memories. An intersubject representational similarity analysis of cross-subject semantic content and neural patterns revealed a differentiation in hippocampal representation by narrative type: semantically similar, sad autobiographical memories elicited similar neural representations across participants. By contrast, within the same individuals, semantically similar trauma memories were not represented similarly. Furthermore, we were able to decode memory type from hippocampal multivoxel patterns. Finally, individual symptom severity modulated semantic representation of the traumatic narratives in the posterior cingulate cortex. Taken together, these findings suggest that traumatic memories are an alternative cognitive entity that deviates from memory per se.

Personal memory is at the core of post-traumatic stress disorder (PTSD)[1]. Research on the neural mechanism of PTSD has largely focused on interpersonal basic learning and memory paradigms[2]. It is still unclear how traumatic memories differ from negative non-traumatic autobiographical memories. Is a traumatic memory an exceptionally strong manifestation of autobiographical memory or a different neural representation altogether?

To examine this, we needed to factor in differences across individual traumatic narratives and the idiosyncratic experiences that they evoked, and extract from them the common markers operating in a trauma-driven state. With this in mind, we designed a study that examines the neural responses of patients with PTSD to their own personal traumatic memory in the form of a structured, fully annotated, audio narrative. We compared traumatic memory, within each participant, to a negatively valenced, nontraumatic, sad memory and a calm positive memory.

Previous research has established the role of the hippocampus in the construction of relational cognitive maps, on to which events are bound across space and time to form episodic memories[3–5]. It is through this tracking of sequences of events that the hippocampus is necessary

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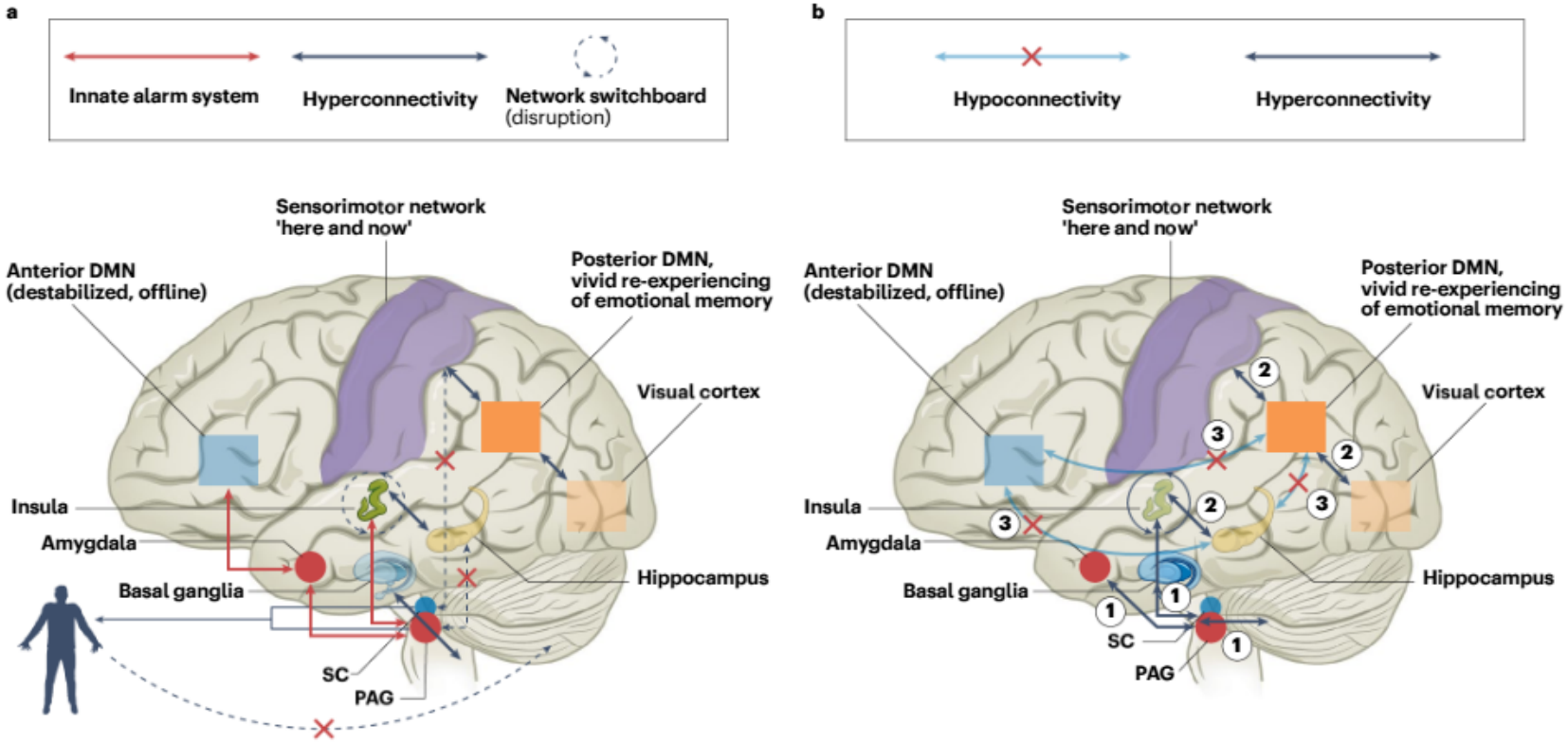

Fig. 3 | Traumatic memory. a, Under extreme stress from imminent threat, incoming sensory input alerts innate alarm regions and initiates low-level sensorimotor defensive responses. Ineffective, incomplete or lack of action (as depicted by static, overwhelmed silhouette) disrupts sensorimotor feedback, leading to massive prediction error. Sensory and motor circuits become stuck in hyperconnectivity patterns instead of transferring information to association cortices. **b**, Traumatic memory is involuntarily relived as sensory and motoric fragments. Traumatic memory traces are represented in (1) hyperactive/hyperconnected innate alarm circuits; (2) primary sensory and motor hyperconnectivity with the posterior DMN, which is involved in reliving of memory; and (3) hypoconnectivity among hubs of the DMN. These patterns are associated with vivid reliving and re-enactment of traumatic experience. The numbers correspond to the numbered descriptions in the caption. DMN, default mode network; PAG, periaqueductal gray; SC, superior colliculus. Figure created with BioRender.com.

Sigurd Wiingaard Uldall

Dissociation

ChatGPT:	**Dissociation er et psykologisk fænomen, hvor en person oplever en adskillelse mellem deres tanker, minder, følelser eller identitetsfølelse. Det kan variere fra milde, hverdagslige oplevelser til alvorlige forstyrrelser, der ofte er forbundet med traumer.**
Lærebog:	**Dissociative symptomer er et udtryk for tab eller fraspaltning af normal integration mellem erindring, identitet, sanseoplevelser og motorik**
MBJ:	**Udfordrer illusionen om den frie vilje og bevidsthedens enhed**

Traditionel hysteri, conversion ICD10:

F44 Dissociative and conversion disorders
- F44.0 Dissociative amnesia
- F44.1 Dissociative fugue
- F44.2 Dissociative stupor
- F44.4 Conversion disorder with motor symptom or deficit
- F44.5 Conversion disorder with seizures or convulsions
- F44.6 Conversion disorder with sensory symptom or deficit
- F44.7 Conversion disorder with mixed symptom presentation
- F44.8 Other dissociative and conversion disorders
- F44.81 Dissociative identity disorder

F45 Somatoform disorders ? Ondt i livet belastning - udtrykt i smerte?

Oplevelse af en adskillelse (gør noget ! jeg kan ikke lade være):

Bulimia/Bingeating: A disorder characterized by recurrent episodes of binge-eating over which the individual feels a lack of control;

Alkoholism/ Drug dependency (sp morfica)

Cutting (borderline, cPTSD,)

Depersonalisationsoplevelser

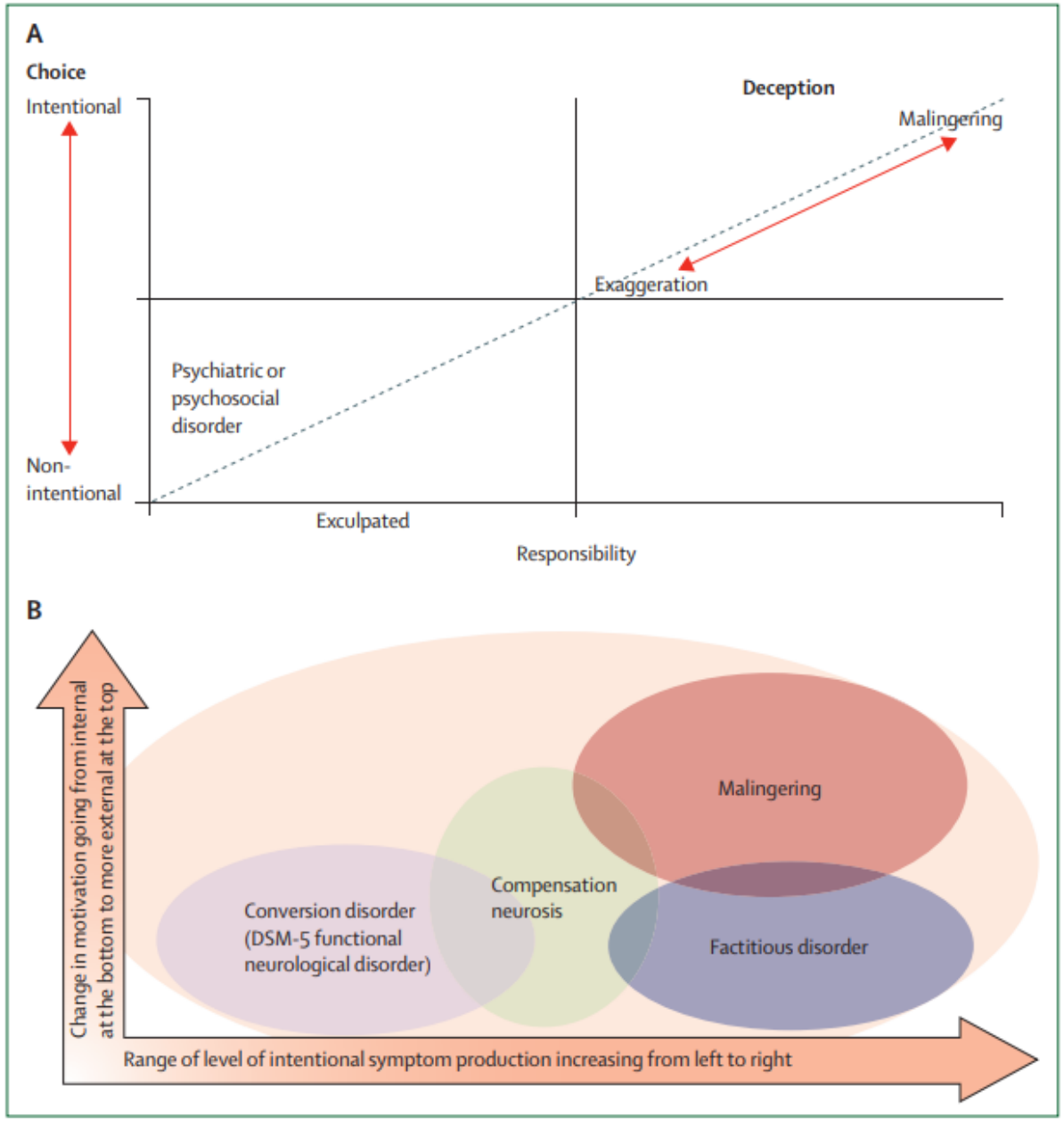

***Figure*: Two models of illness deception (A)[8] and compensation neurosis (B)[83]**
Reproduced by permission of Sage Publications (A) and American Psychiatric Press (B). Diagrams show the potential roles of patient choice, intentions, and motivation in symptom production and, ultimately, diagnosis. DSM-5=Diagnostic and Statistical Manual of Mental Disorders, fifth edition.

Bass og Halligan Lancet 2014