Post traumatiske stress reaksjoner og andre helseplager

Av Synne Øien Stensland

Cover: Stian Hole, NASA & Nature

Nasjonalt kunnskapssenter
om vold og traumatisk stress

Mangel på systematisk kunnskap om barn og unges somatiske helseplager etter traumatiske hendelser kan føre til at de ikke får den hjelpen de trenger.

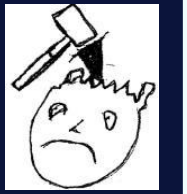
Vos et al 2015, the Lancet. Stensladn et al 2017. Heyer et al 2017.

Julia, 9 år. HODEPINE.

SYMPTOM:

SOMATISK: Pulserende smerter i hodet ukentlig eller oftere. Kommer brått. Intens smerte. Ledsagende lyd og lysskyhet. Kvalme/brekninger. Søvnvansker.

PSYKISK: Konsentrasjonsvansker. Trist.



FUNKSJON:

Sosialt tilbaketrukket.

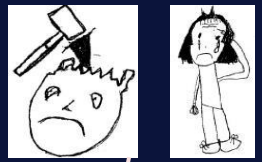
Dårlige skoleprestasjoner. Høyt skolefravær.

UTVIKLING:

Prepubertal. Forventet utvikling?

HEREDITET: Mor, Migrene.

Foto: Mary Ellen Mark.



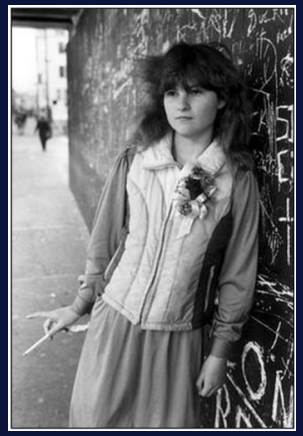
Bilde: The Guardian. Ref. Dyb & Stensland et al 2016.

Julia, 16 år. INTOX.

SYMPTOM:
PSYKISK: Selvskading. Depresjon. Spiseforstyrrelse, bulimi.
SOMATISK: Migrene. Smerter.
RISIKO: Fedme → hjerte/kar sykdom.

FUNKSJON:
Dårlige skoleprestasjoner. Høyt skolefravær. Frafall. Konflikt venner & familie.

UTVIKLING:
Postpubertal. Siste 1/3 av ungdomstid.



«Jeg hadde ingen steder å gjemme meg....jeg husker hvor forferdelig vondt det [den første voldtekten] *gjorde. Jeg følte skyld. Jeg følte skam. Jeg følte at noe var galt med meg. Jeg følte at jeg ikke var verdt noen ting. Og at det ikke var noen som brydde seg.»*

AP-politiker Libe Rieber-Mohns knuste barndom: Jeg hadde ingen steder å gjemme meg," 2014.

Foto: Mary Ellen Mark. The Guardian.

INNHOLD

REAKSJONER
- Posttraumatiske stress reaksjoner
- Somatiske reaksjoner
- Hvordan kan plagene henge sammen?

UTREDNING & BEHANDLING?
- Motivasjon & symptom

Vinall et al 2016.

Posttraumatiske stress reaksjoner

Smerte, utmattelse?

Gjenopplevelse

Unngåelse

Negative endringer tanker/følelser

Endret fysiologisk respons

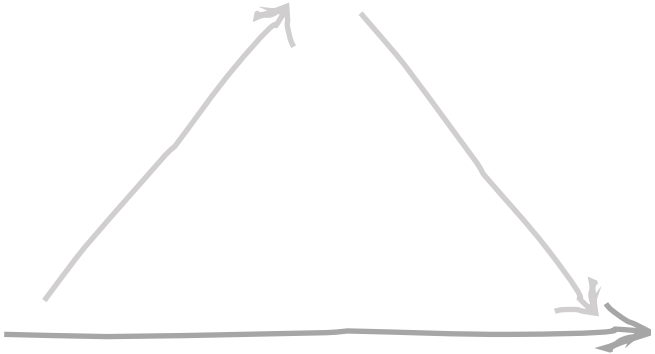
Traumespesifike

Generelle reaksjoner

Vold, seksuelle overgrep, mobbing & andre traumatiske hendelser

Somatiske helseplager

Posttraumatiske stress reaksjoner



Vold, seksuelle overgrep, mobbing & andre traumatiske hendelser

Sing et al 2005. Stensland et al 2017. Heyer et al 2017.

ARTICLE

The headache of terror

A matched cohort study of adolescents from the Utøya and the HUNT

Synne Øien Stensland, MD, PhD, John-Anker Zwart, MD, PhD, Tore Wentzel-Larsen, MSc, and Grete Dyb, MD, PhD

Corresponde
Dr. Stensland
synne.stenslar

Neurology® 2018;90:1-8. doi:10.1212/WNL.0000000000004805

RELATED ARTI

Editorial
The pain of

Abstract

Objective

EDITORIAL

The pain of terror

Geoffrey L. Heyer, MD, and Kenneth J. Mack, MD, PhD

Neurology® 2018;90:1-2. doi:10.1212/WNL.0000000000004813

Headaches and stress go hand in hand. Most individuals with a primary headache disorder perceive triggers for their attacks, and emotional stress is the trigger most commonly reported.[1] Daily perceived stress correlates temporally with daily migraine activity.[2] There may be a reciprocal relationship between migraines and stress whereby each influences the other over time.[2] An association also occurs between perceived stress intensity and primary headache frequency.[3] Even early childhood stressors such as trauma and neglect may predispose an individual to headaches later in life.[4] Most studies that have analyzed the stress-headache relationship have done so by measuring prior or current stressors as reported by the patient with headaches. Rarely can the onset or worsening of headaches be attributed to a single life event.

In this edition of *Neurology*, Stensland et al.[5] demonstrated that survivors of a horrific mass shooting developed an increased risk of migraine and tension-type headache. As described in their article, a lone gunman, disguised as a police officer, opened fire at a summer camp for adolescents, killing 69 people. Among the 362 survivors, 213 (nearly 60%) participated in the study. All survivors were exposed to terror; many lost friends; and many risked hypothermia or drowning trying to escape. Participants were matched to controls drawn from a population-based, general health study. A validated headache interview was conducted 4 to 5 months after the terror exposure to assess the outcomes of recurrent migraine and tension-type headaches over a 3-month period. Adolescent girls and boys exposed to terror experienced more recurrent primary headaches than their nonexposed peers. The odds ratios for migraine and tension-type headache were 4.27 and 3.39, respectively. A strong relationship between exposure to terror and higher headache frequency remained after adjustment for injury from gunfire, sex, age, previous traumatic experiences, psychological distress, and family economic status. The study is important for medical providers who care for survivors of mass violence and natural disasters because headache interventions, when indicated, can be initiated early and combined with therapies addressing the traumatic experience.

If a single episode of extreme psychological trauma can increase headache risk in susceptible individuals, what are the consequences of daily stressors for the patient with headache? The brain responds to potential and actual stressful events by modifying behaviors and by activating

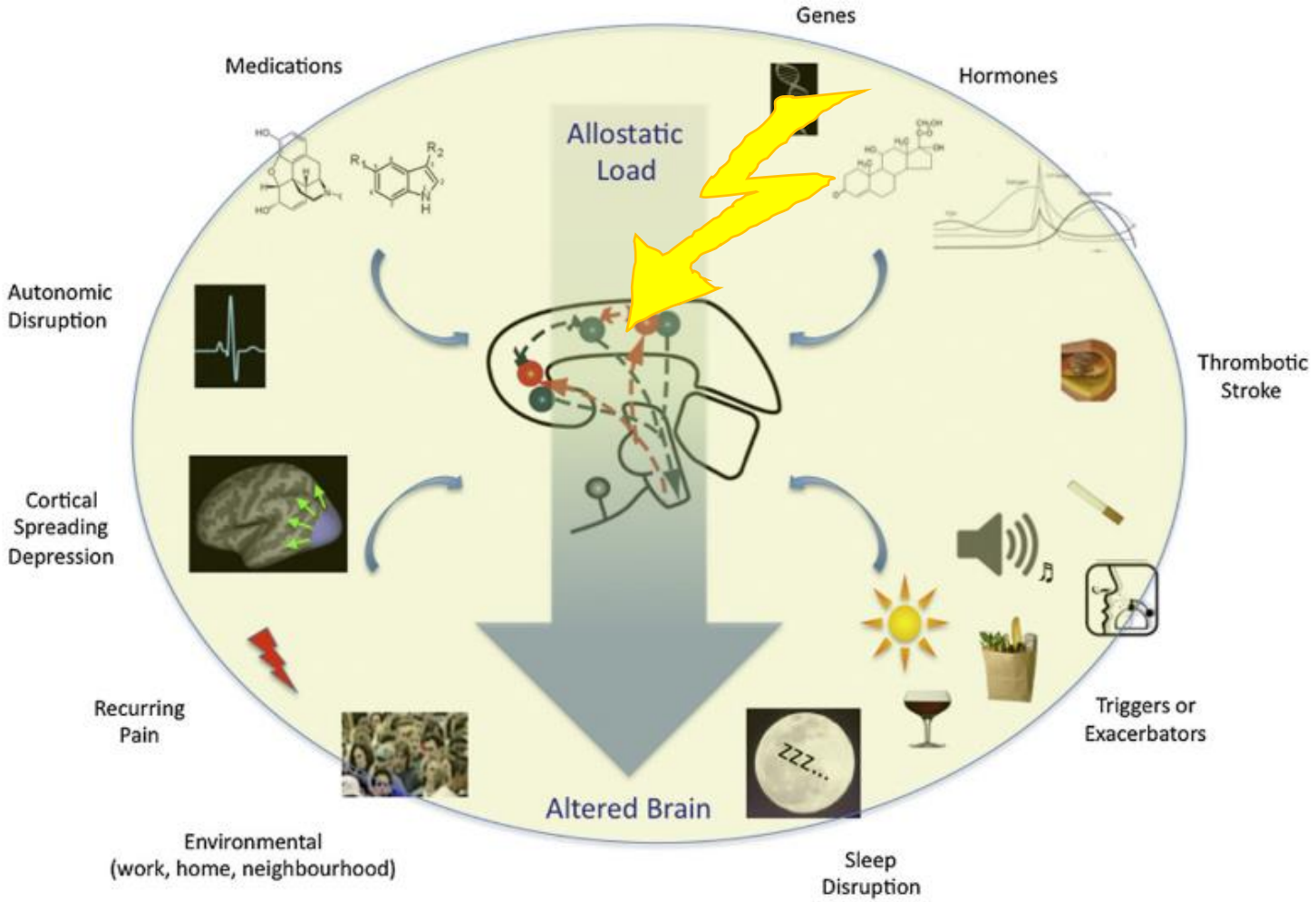


Figure 4. Migraine and Allostatic Effectors

Ref. Borsook et al 2012, May et al 2016, Vetvik et al 2017, Heyer et al 2018.

Overlappende nervebaner

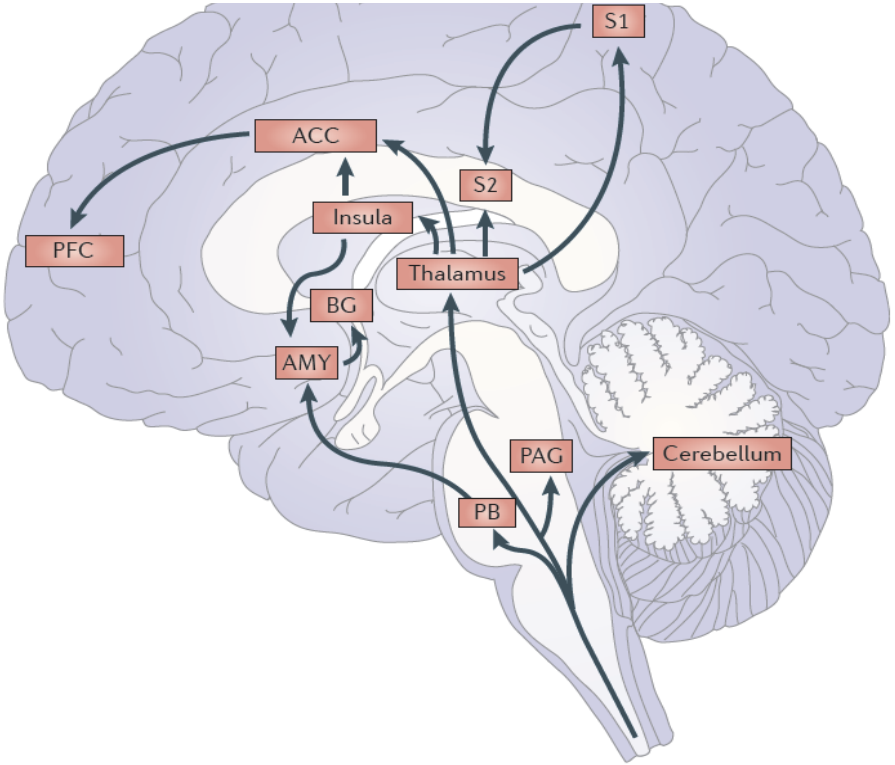


Figure 2 | **Afferent pain pathways include multiple brain regions.** Afferent nociceptive information enters the brain from the spinal cord. Afferent spinal pathways include the spinothalamic, spinoparabrachio–amygdaloid and spinoreticulo–thalamic pathways. Nociceptive information from the thalamus is projected to the insula, anterior cingulate cortex (ACC), primary somatosensory cortex (S1) and secondary somatosensory cortex (S2), whereas information from the amygdala (AMY) is projected

Bushnell, 2013, & Tracey & Mantyh, 2007

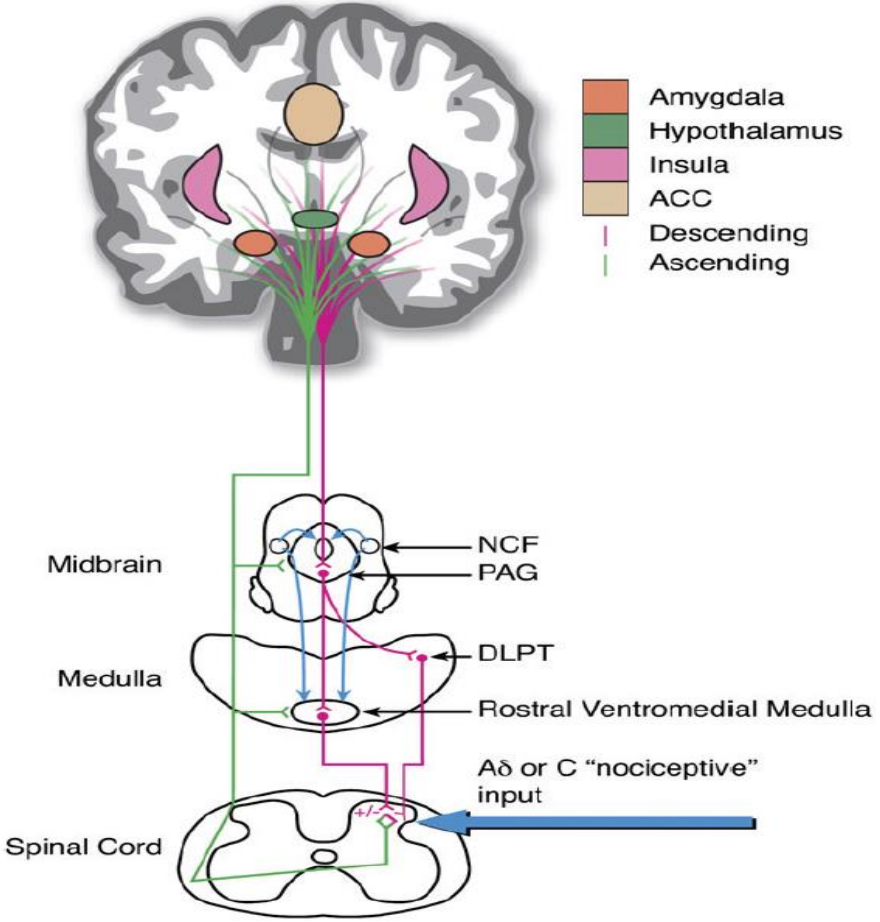
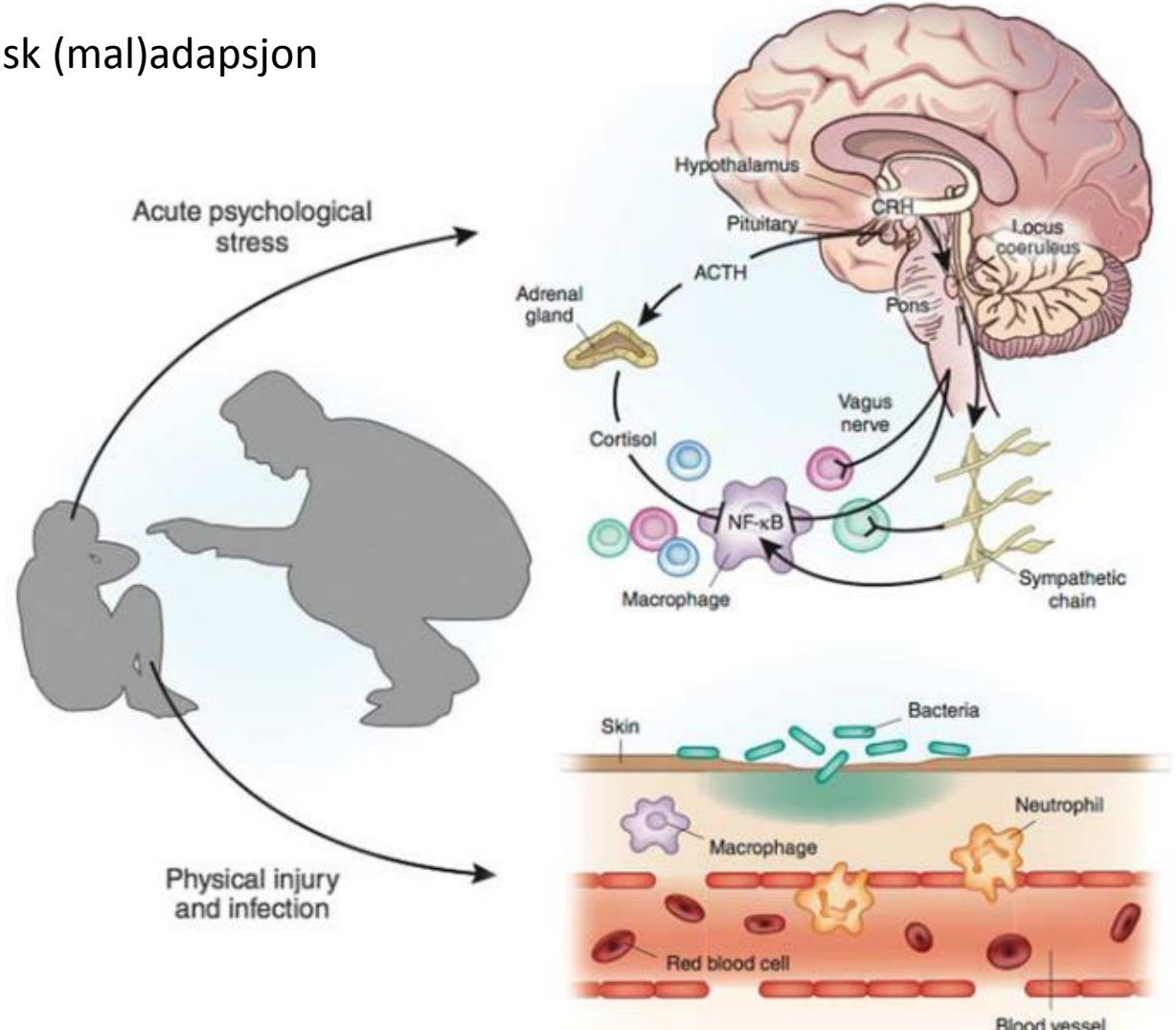


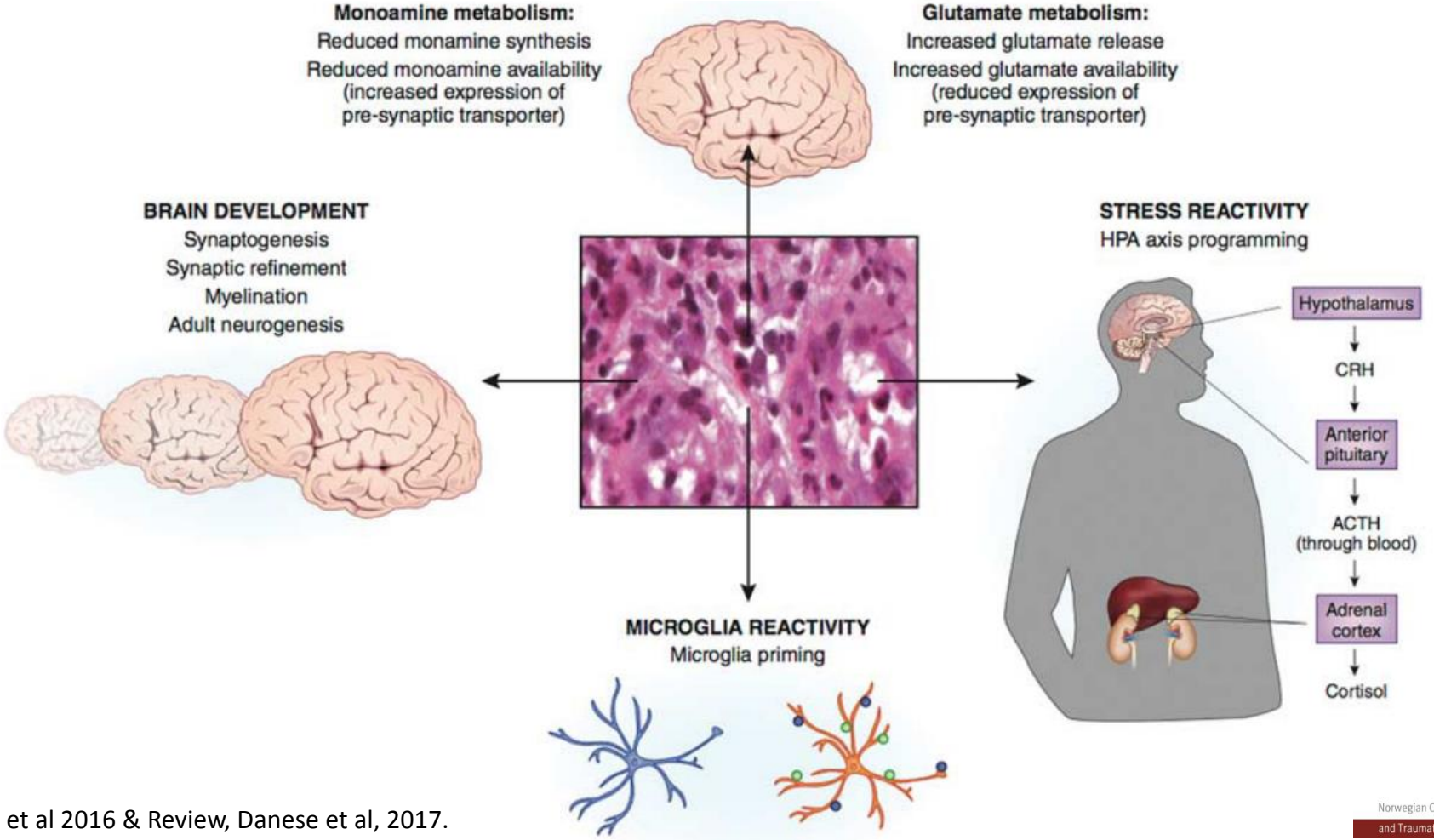
Figure 3. The Descending Pain Modulatory System
NCF (nucleus cuneiformis); PAG (periaqueductal gray); DLPT (dorso-

Neuroimmunologisk (mal)adapsjon



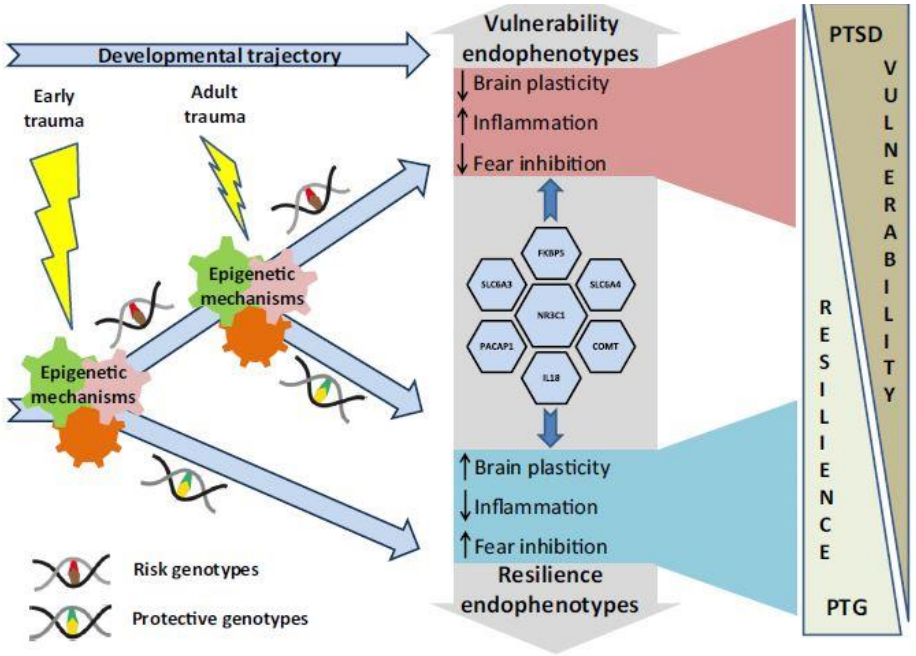
Danese et al 2017

Tilpasninger/endring i nevrologiske nettverk



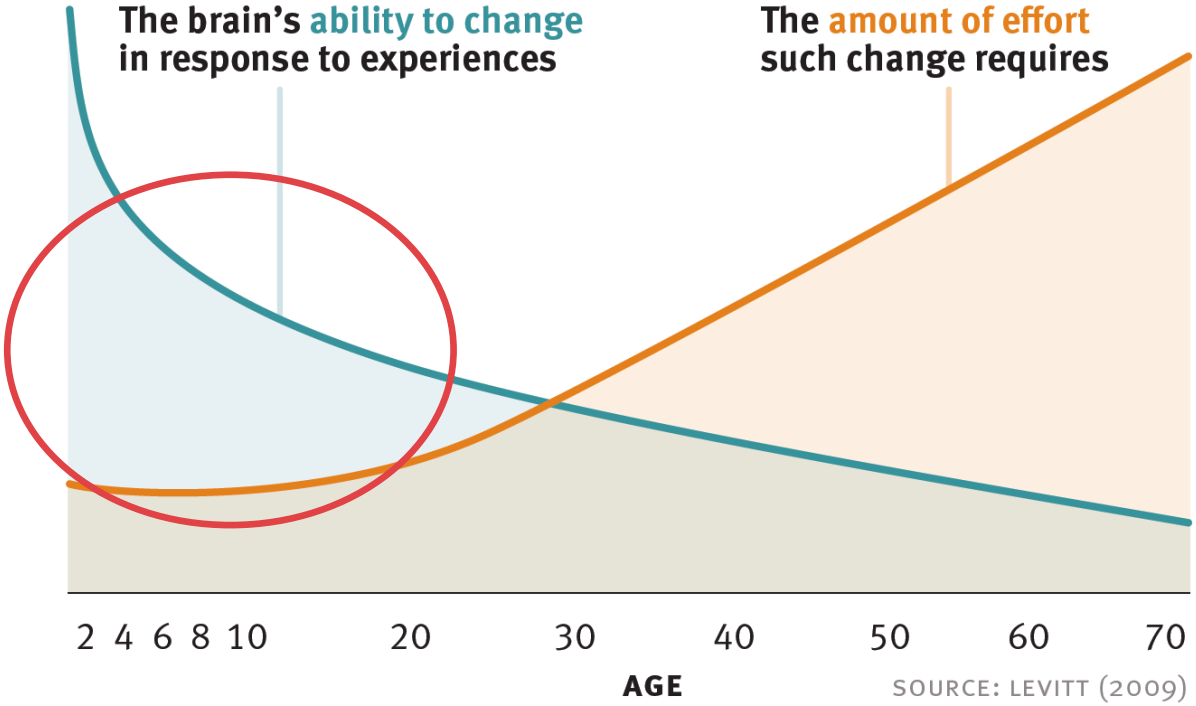
Maleki et al 2016 & Review, Danese et al, 2017.

Epigenetiske endring gir sårbarhet for en rekke psykiske & somatiske helseplager?



representation of gene-trauma-epigenetic interactions in posttraumatic stress disorder and related phenotypes. This model is supported, for example, for the methylation status of the *SLC6A4* promoter, shown to interact with the 5HTTLPR polymorphism of the gene promoter to predict psychological responses to trauma. In particular, individuals carrying the short (risk) allele were more prone to develop unresolved responses to trauma at lower methylation levels, but less prone to develop maladaptive responses at higher methylation levels (40). In line with these findings, the nine-repeat allele of the *SLC6A3* gene was shown to double the risk for posttraumatic stress disorder only in the presence of high methylation levels of the *SLC6A3* promoter (38). Genetic variation of the *COMT* gene was also shown to predict methylation levels of cytosine-phosphate-guanine (CpG) sites within the gene promoter, with higher methylation levels being associated with the Met/Met *COMT* genotype and predicting impaired fear inhibition (39). In addition to moderation of the associations of epigenetic factors with trauma responses, genetic variants may directly moderate trauma-induced epigenetic changes. A study conducted by our group showed that exposure to childhood abuse leads to demethylation of CpG sites in the functional glucocorticoid response element in intron 7 of the *FKBP5* gene only in rs1360780 T-allele (risk) carriers and not carriers of the opposite (protective) genotype (11). In this case, a genetically driven change in systemic glucocorticoid release likely alters DNA methylation changes after early trauma exposure. The risk *FKBP5* allele, which is located close to a functional glucocorticoid response element, was shown to increase binding of an enhancer region located in intron 2 to the transcription start site of the gene. This leads to enhanced induction of *FKBP5* expression after glucocorticoid receptor activation and subsequent glucocorticoid receptor resistance, impairing the negative feedback of the axis. In addition to such indirect effects, the effects of trauma on transcription and DNA methylation changes of sensitive loci may be directly moderated by genetic variants that affect transcription factor binding, remove or create CpG dinucleotides, or lead to altered expression of epigenetic readers and writers that command subsequent epigenetic changes. The term "epigenetic mechanisms" is used for simplification purposes and denotes the entirety of epigenetic processes, including DNA methylation, posttranslational histone modifications, noncoding RNAs, and three-dimensional changes in chromatin conformation, which act in concert to regulate gene transcription. PTG, posttraumatic growth; PTSD, posttraumatic stress disorder.

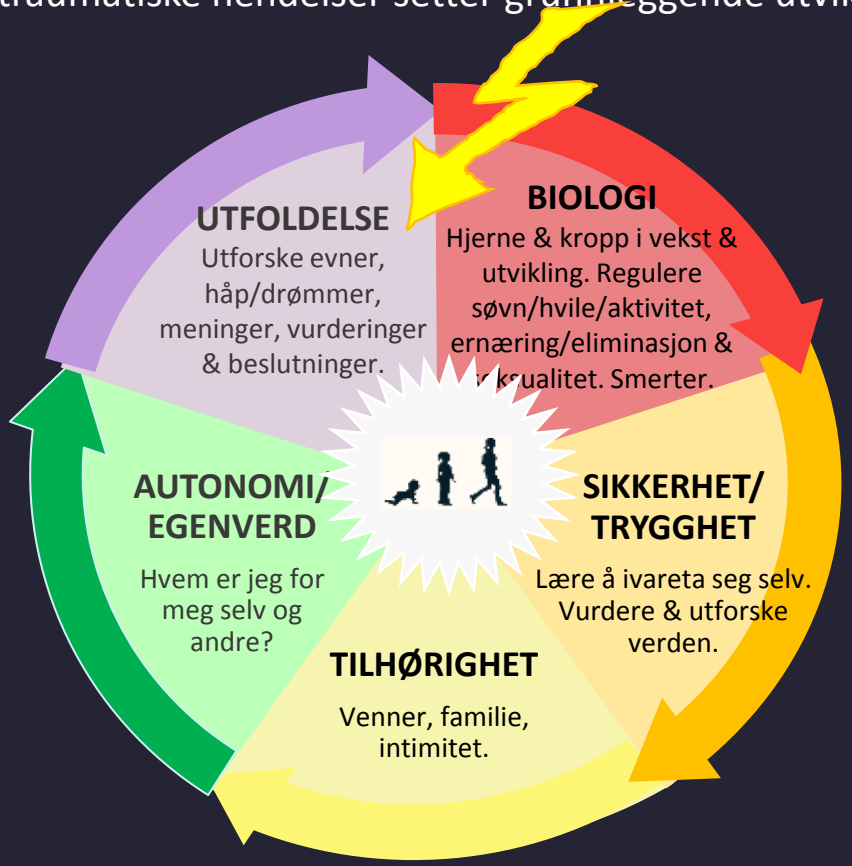
Binder et al 2016, Teicher et al 2016



Center on the Developing Child HARVARD UNIVERSITY

www.developingchild.harvard.edu

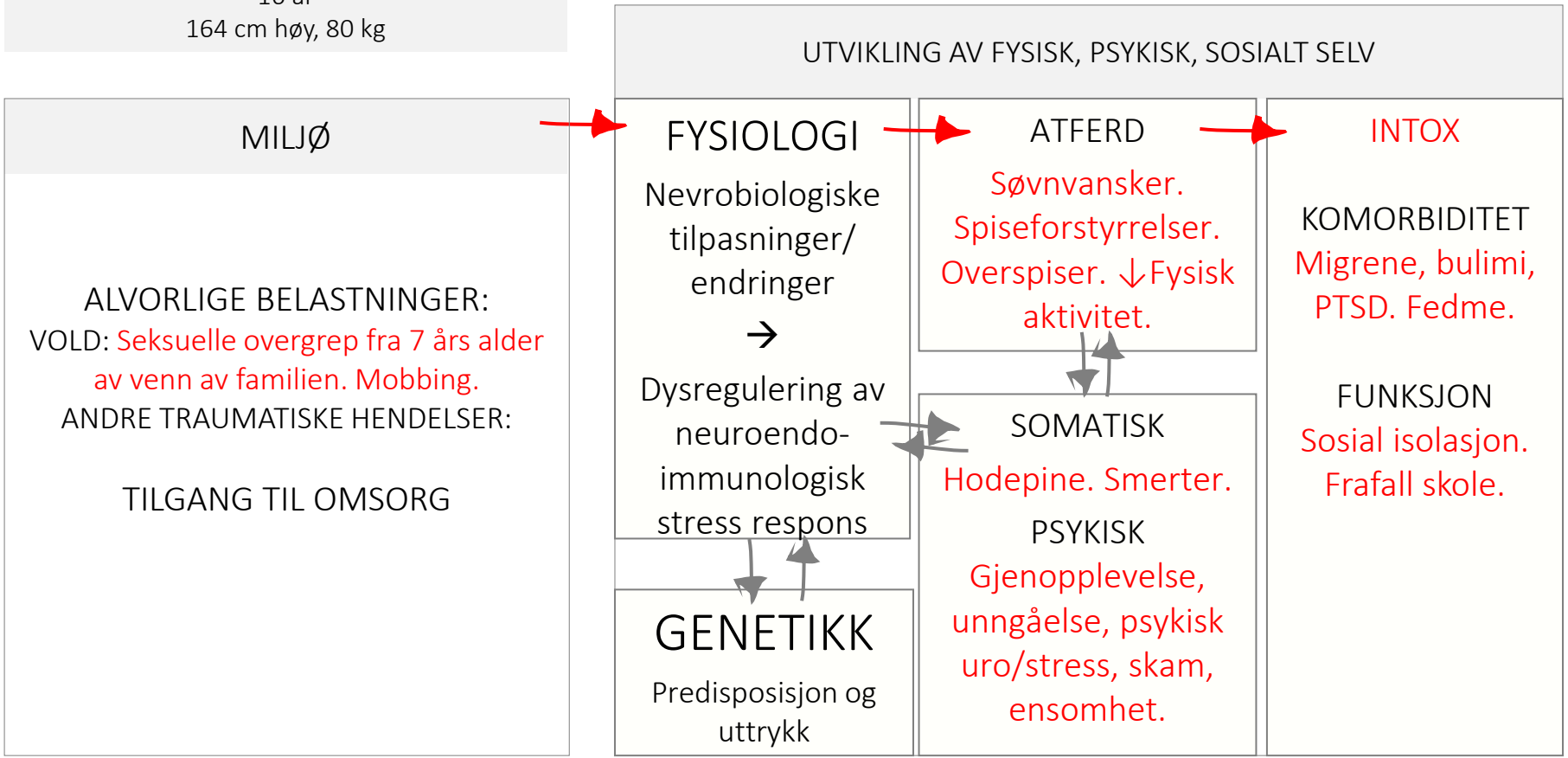
Oppsummert: Vold & andre traumatiske hendelser setter grunnleggende utviklingsoppgaver står på spill.



Utviklingspsykologi for dummies. Ref Bronfenbrenner 1979, Engel1977, Rutter (child psychiatry), Nelson, Kliegman & Behrman, Repetti 2002, von Tetzchner , Grøholt & Sommerschild. Stensland 2012. Sing et al 2005.

Hva skjer med Julia ?

16 år
164 cm høy, 80 kg



Ref. McEwen et al 1998, Brewerton et al. 2007, Stensland et al 2013 & 2014, Ippolotti 2012, Danese et al 2014, Borsook et al 2012

Norwegian Centre for Violence and Traumatic Stress Studies

INNHOLD

REAKSJONER

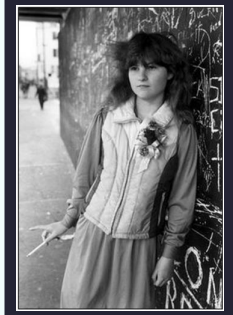
- Posttraumatiske stress reaksjoner
- Somatiske reaksjoner
- Mulige mekanismer

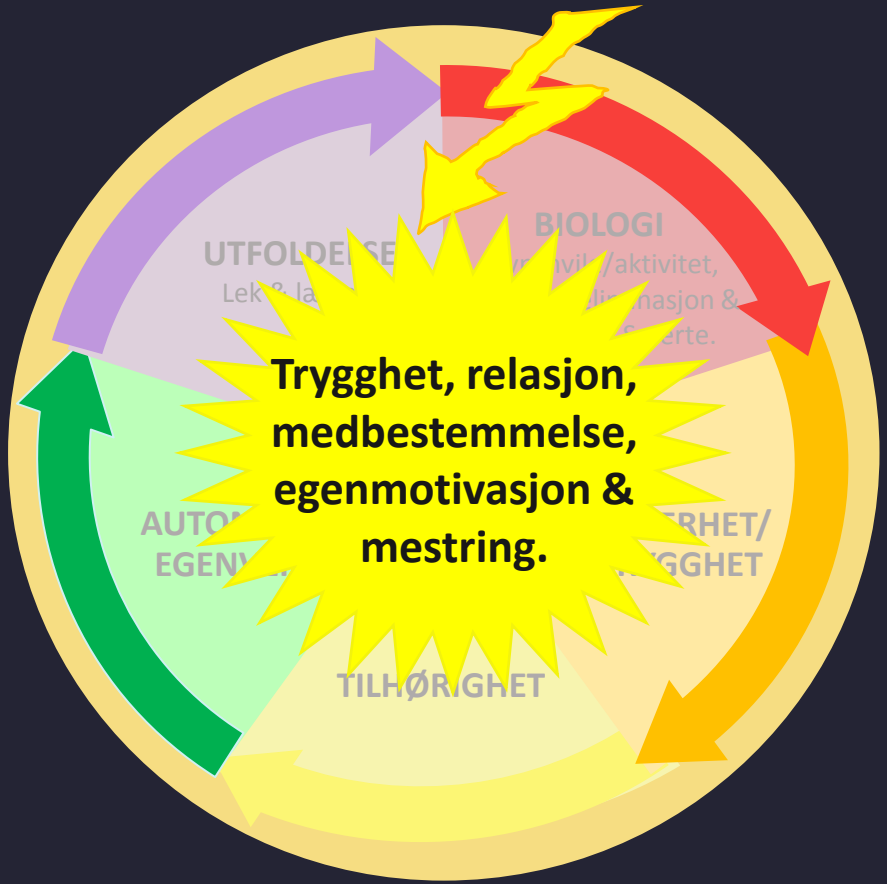
UTREDNING & BEHANDLING?

- Motivasjon & symptom

Motivasjon & Symptom

Motivasjon: Maks ‘placebo’ effekten eller den terapeutiske alliansen: Ivareta grunnleggende behov i møtet.





JA, vi klarer det

Med ferdigheter, øvelse, veiledning og kollegastøtte.

- Respekter SYMPTOMET de presenterer.
- MOTIVASJON: Vis omsorg. Maks 'placebo'effekten.
- KARTLEGG. Igjen. Og igjen.
  - psykiske og somatiske plager og atferdsvansker bredt.
  - Vold, seksuelle overgrep, mobbing og andre traumatiske hendelser.
  - Omsorgsforhold
  - Andre risikofaktorer.
- Tenk TIDLIG, MÅLRETTET INTERVENSJON: Gi samordnet psykisk OG somatisk behandling etter behov.
- Barn & unge trenger OMSORG. Ikke glem familien. Varsle barnevern/politi ved behov.
- Monitorer tiltak.

Takk for ferden & lykke til videre …

Kollegastøtte: August 1997: De russiske kosmonautene Vasily Tsibliyev (til venstre) og Alexander Lazutkin omfavner hverandre, etter å ha landet på de øde steppene i Kazakhstan. Foto: Sergei Karpukhin/EPA. Artikkel: Sandal et al 2005.