Stress

Montag 12:15 – 13:45 Uhr

HZO 80

Prof. Oliver T. Wolf

FAKULTÄT FÜR PSYCHOLOGIE
Arbeitseinheit Kognitionspsychologie
www.cog.psy.ruhr-uni-bochum.de

Vorläufige Terminübersicht

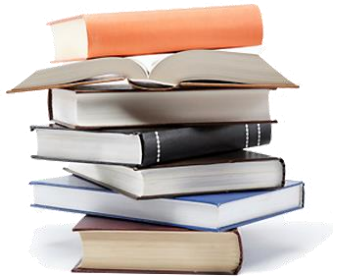
20.10.25	Übersicht und Einführung
27.10.25	Stress und das SNS: Walter Cannon (Dr. Katja Langer)
03.11.25	Stress und die HHNA: Hans Selye
10.11.25	Stress und die HHNA: Munck und Sapolsky Die kognitive Wende: Lazarus
17.11.25	Stress und Gesundheit: McEwen und die allostatische Belastung Stress im Arbeitsleben: Siegrist und die Effort Reward Imbalance
24.11.25	Burnout (Dipl. Psych. Natalie Freund)
01.12.25	Soziale Evaluation als bedeutsamer Stressor: Dickerson & Kemeny (Dr. Katja Langer)
08.12.25	Soziale Unterstützung als Stresspuffer/Oxytozin
15.12.25	Stress und Gehirn: akute und chronische Effekte
12.01.26	Pränataler Stress und seine Folgen
(Aufzeichnung)	Frühkindlicher Stress und seine Folgen (Prof. Robert Kumsta)
19.01.26	Posttraumatische Belastungsstörung

Literatur

- Wolf, O.T. (2009). Stress and memory in humans: Twelve years of progress? *Brain Research, 1293*, 142-154.

- Roozendaal, B., McEwen, B.S., Chattarji, S. (2009). Stress, memory and the amygdala. *Nature Reviews Neuroscience, 10(6)*, 423-433.

- Davidson, R.J., McEwen, B.S. (2012). Social influences on neuroplasticity: stress and interventions to promote well-being.
  Nature Neuroscience, 15, 689-695.



Gliederung

- zwei Forschungsansätze
- Cortisol & Gedächtnis
  - Stress & Gedächtniskonsolidierung
  - Stress & Gedächtnisabruf
- chronischer Stress

Stress & Gehirn: zwei Forschungsansätze

RUB

- **zwei Forschungsansätze**
- Cortisol & Gedächtnis
  - Stress & Gedächtniskonsolidierung
  - Stress & Gedächtnisabruf
- chronischer Stress

Stress & Gehirn

STRESS

CRH

↓

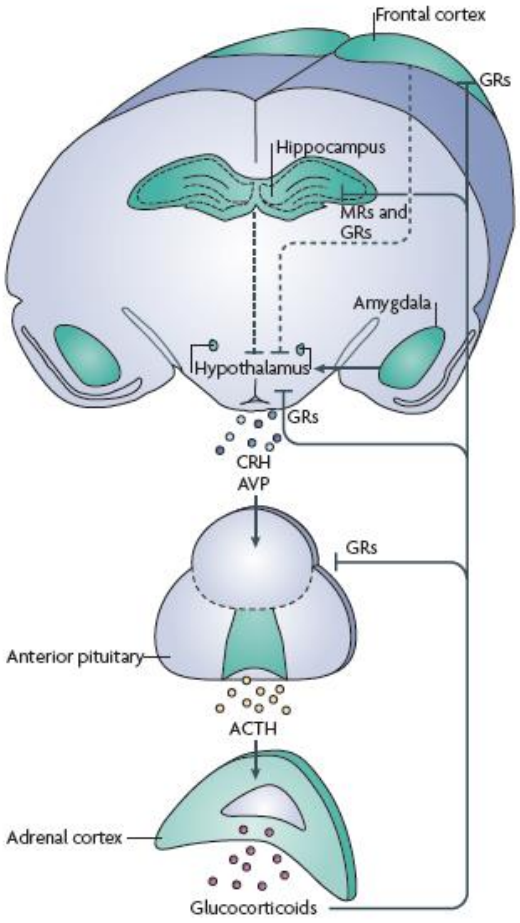
ACTH

↓

Cortisol

Gehirn | **Metabolismus** | **Immunsystem**

Cortisol & Gehirn (Lupien et al., 2009)



Stress & Gehirn: Review (Wolf, 2009)

BRAIN RESEARCH 1293 (2009) 142–154

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Review

Stress and memory in humans: Twelve years of progress?

*Oliver T. Wolf**

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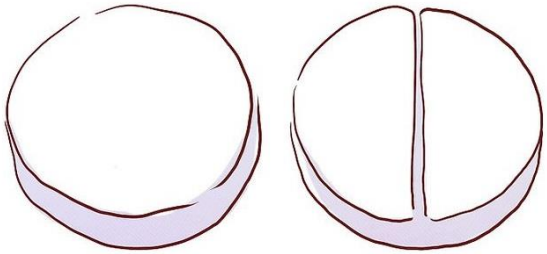
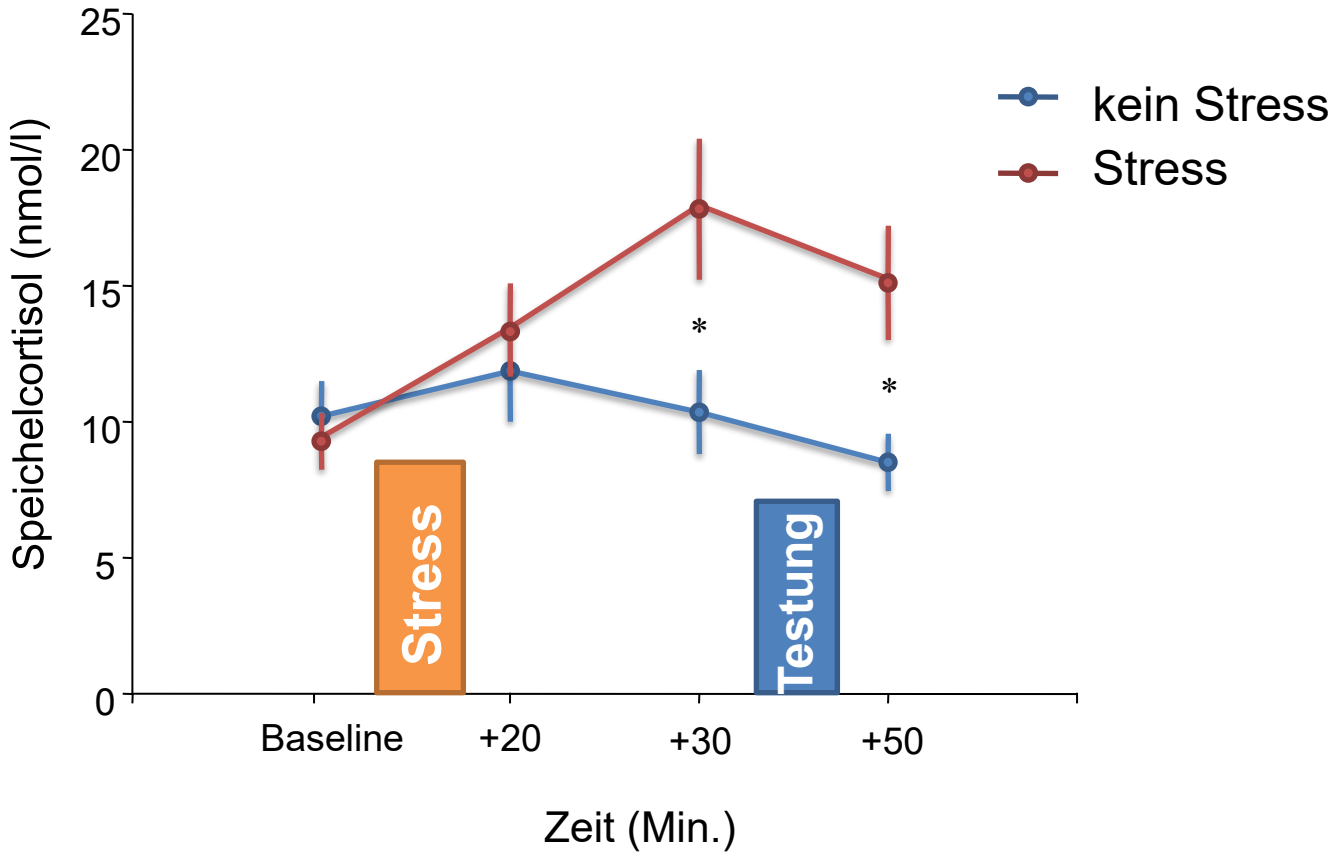
Stress & Gehirn: zwei Forschungsansätze

- Stressinduzierung mittels standardisierter Laborstressoren (TSST oder SECPT)

- experimentelle pharmakologische Manipulation der relevanten Stresshormone



Stress & Gehirn: psychosozialer Stress im Labor (Kuhlmann et al., 2005)

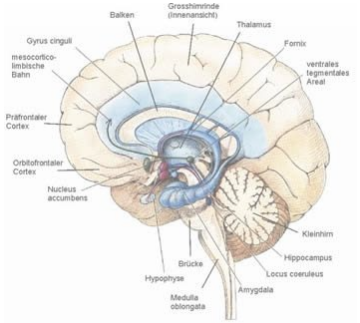


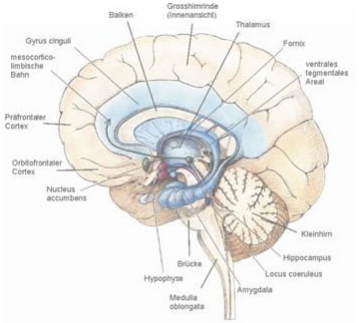
Stress & Gehirn: Cortisol & Gedächtnis

RUB

- zwei Forschungsansätze
- **Cortisol & Gedächtnis**
  - Stress & Gedächtniskonsolidierung
  - Stress & Gedächtnisabruf
- chronischer Stress

Stress & Gehirn: Multiple Gedächtnissysteme im Gehirn





Gedächtnis

deklaratives Gedächtnis

prozedurales Gedächtnis

episodisches Gedächtnis

semantisches Gedächtnis

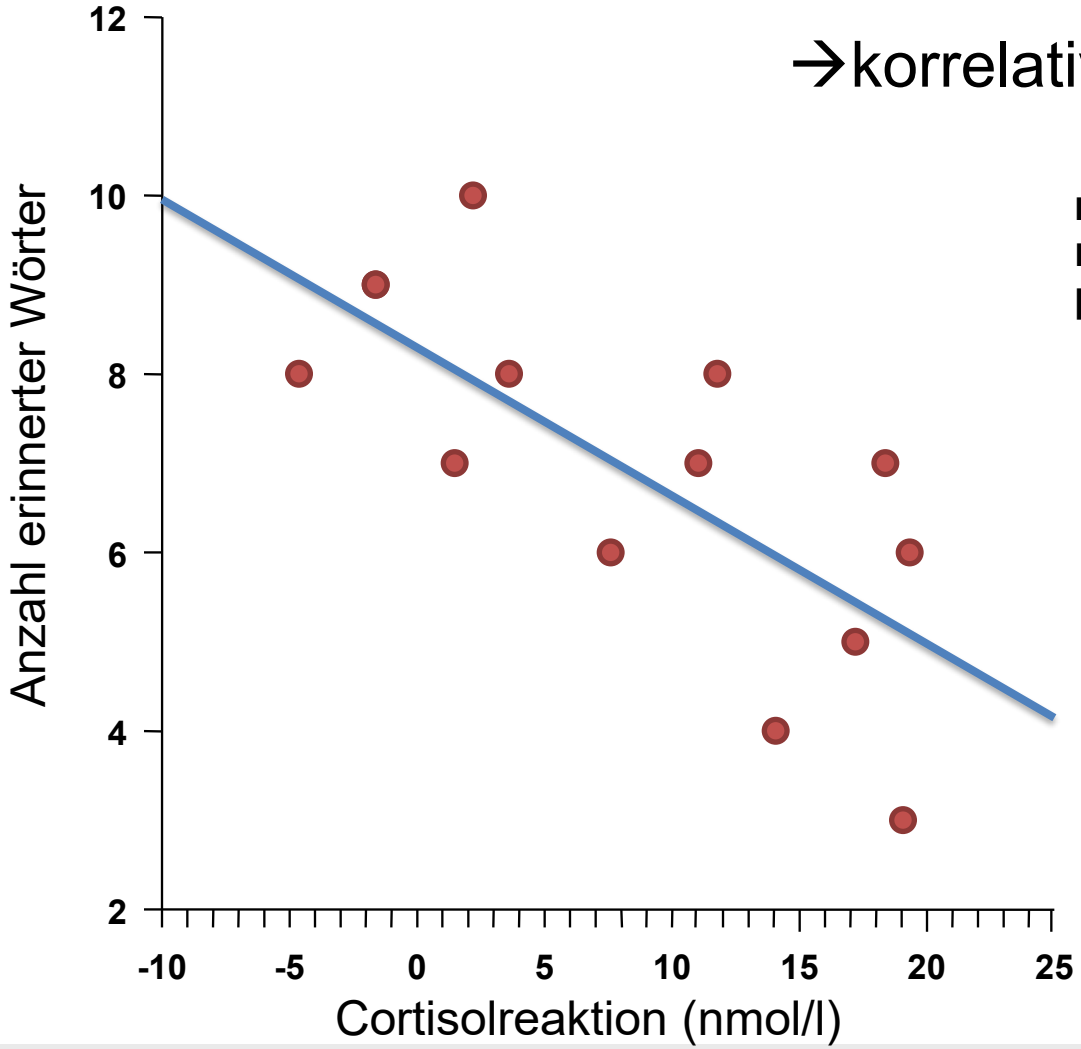
Priming

Fertigkeiten

klassische Konditionierung

Squire (1992)

Stress & Gehirn: Cortisol, Stress & Gedächtnis (Kirschbaum et al., 1996)

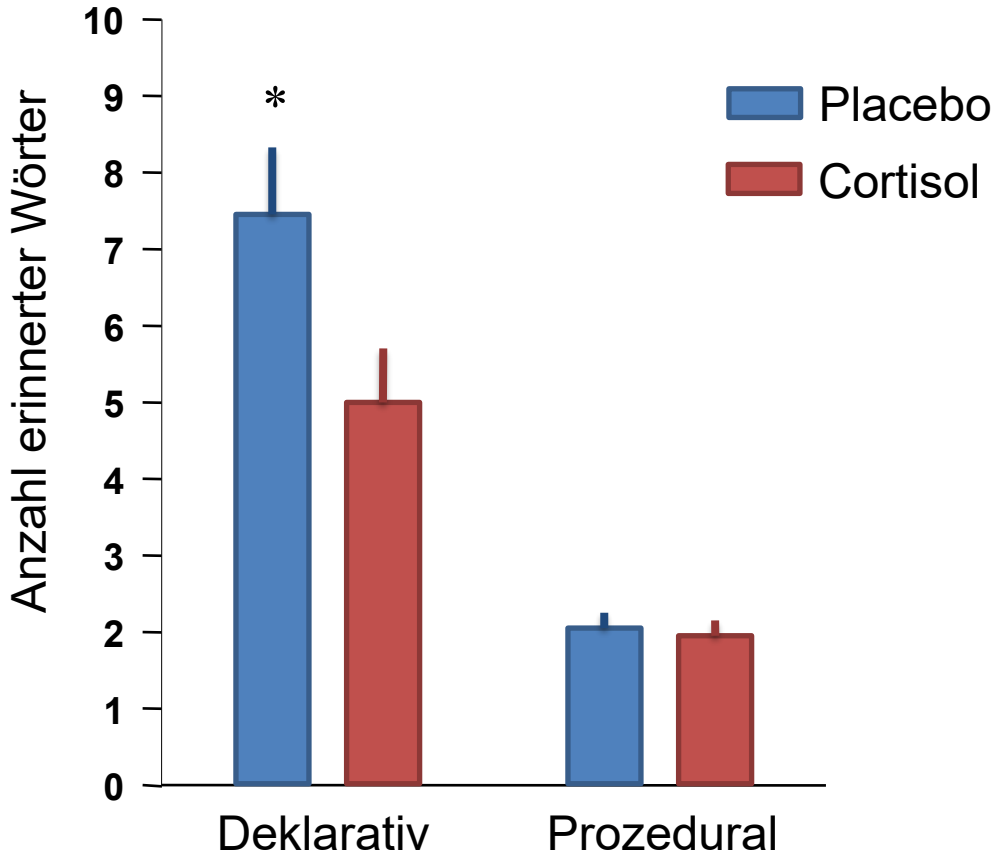


→korrelative Hinweise

r = -.70
n = 13
p = .007

Stress & Gehirn: Cortisol & Gedächtnis (Kirschbaum et al., 1996)

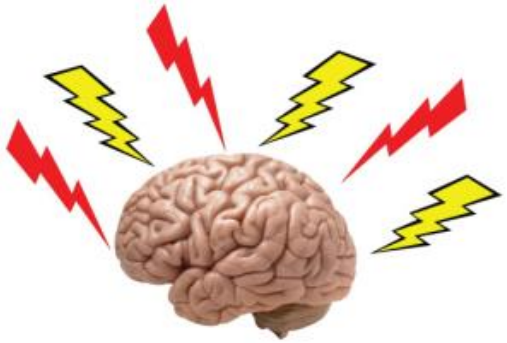
Hinweise aus einer ersten placebokontrollierten Studie:



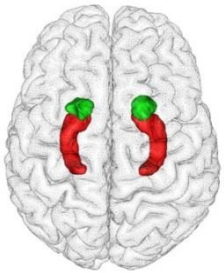
Stress & Gehirn: Cortisol, Stress & Gedächtnis

RUB

- sowohl **verbessernde** als auch **verschlechternde** Effekte von akutem Stress

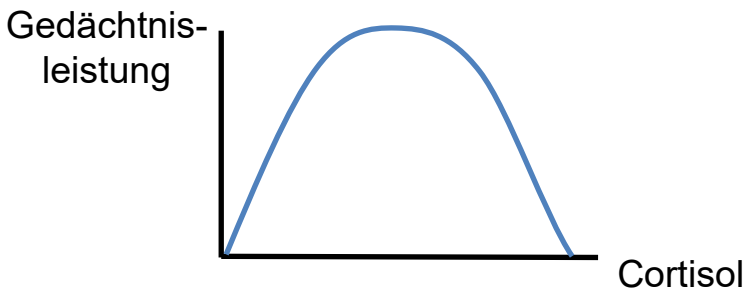


- mögliche Erklärungen:

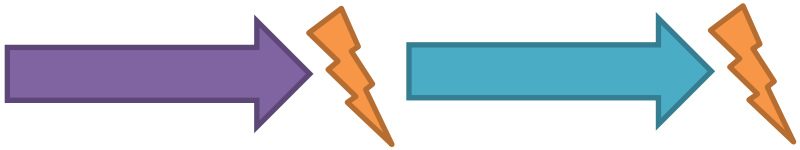


Hippocampus
↕
Amygdala

umgekehrte U-Funktion



phasenabhängige Effekte



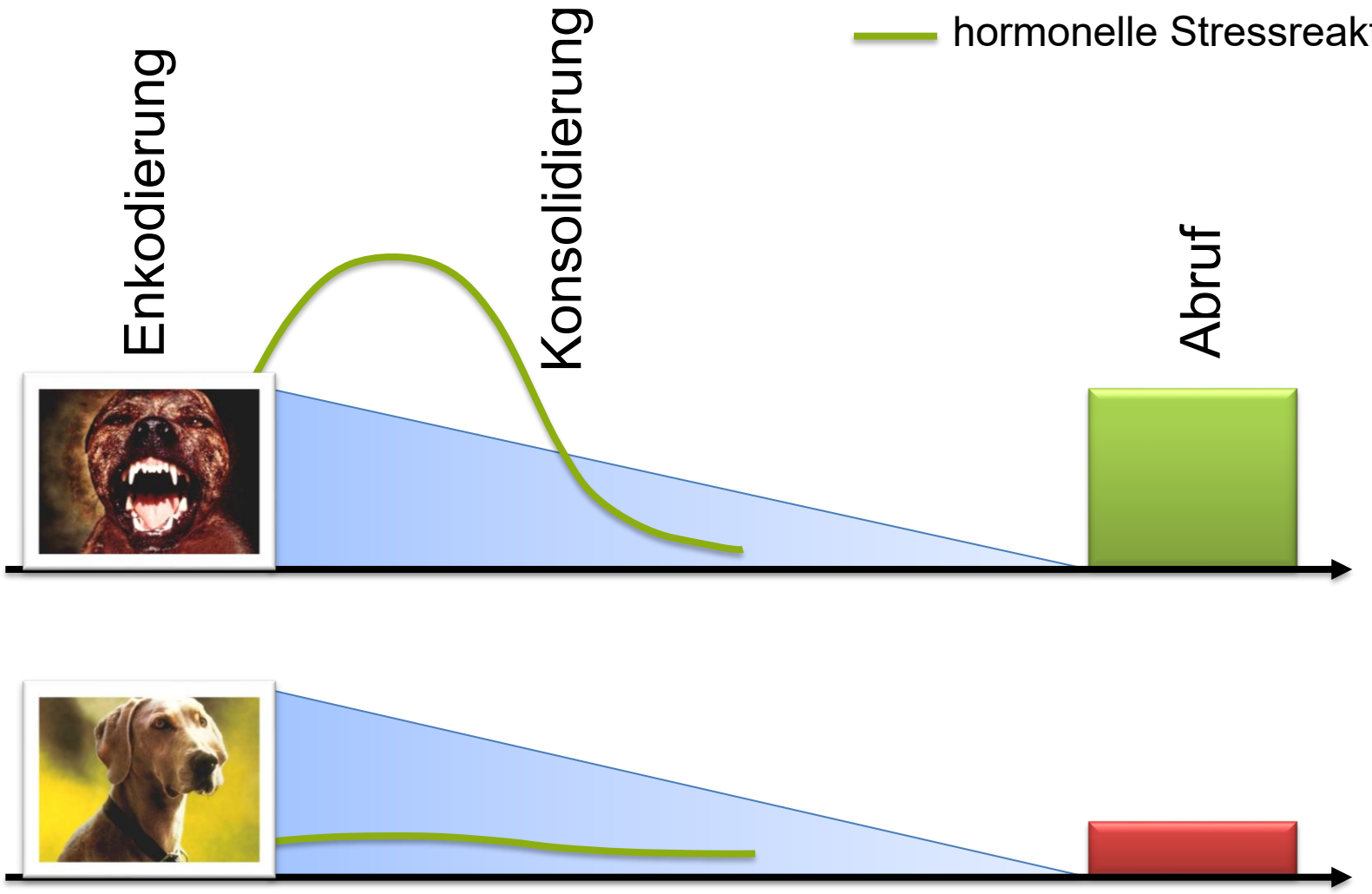
Stress, Gedächtnis & Amygdala: Review (Roozendaal et al., 2009)

Stress, memory and the amygdala

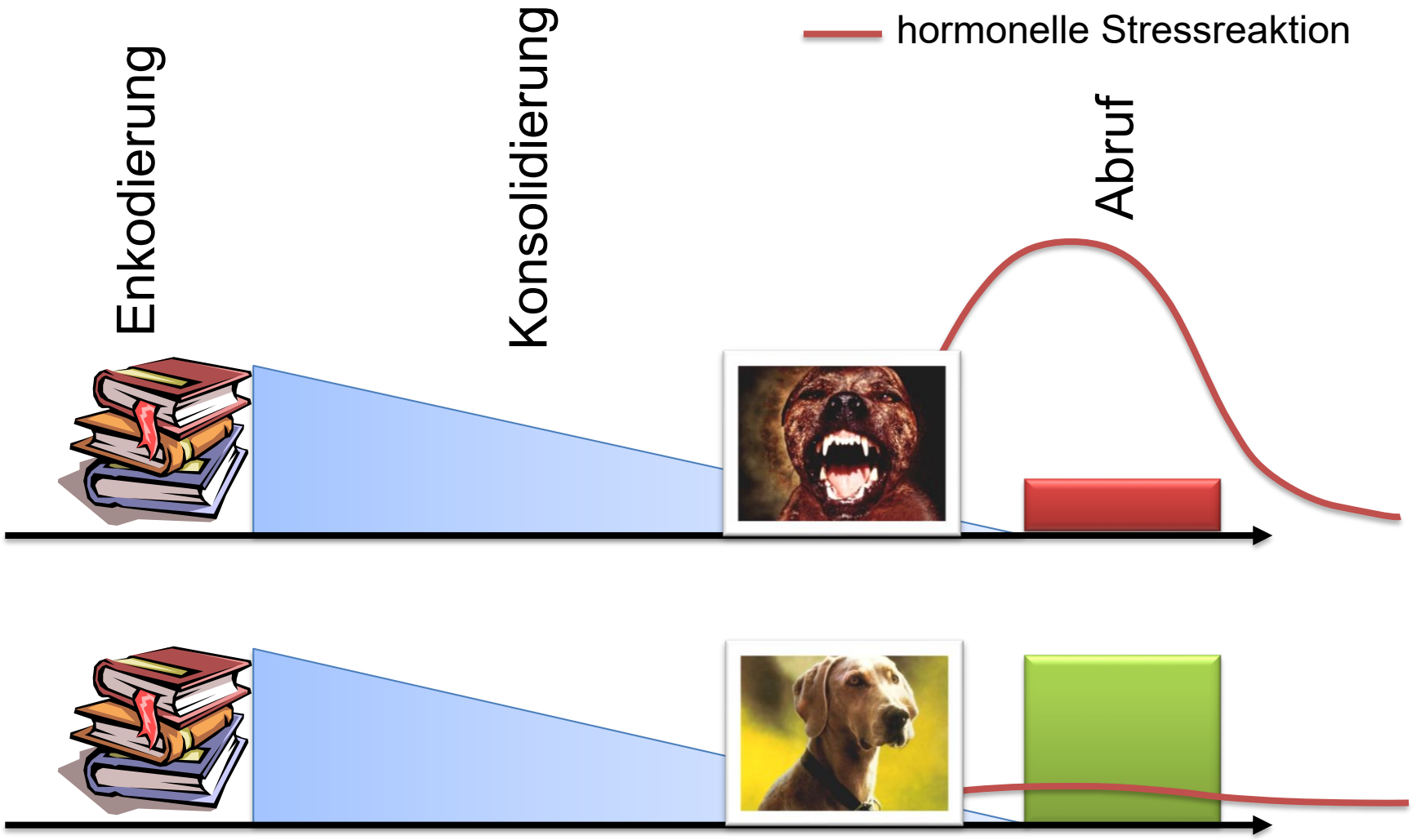
Benno Roozendaal, Bruce S. McEwen‡ and Sumantra Chattarji§*

Abstract | Emotionally significant experiences tend to be well remembered, and the amygdala has a pivotal role in this process. But the efficient encoding of emotional memories can become maladaptive — severe stress often turns them into a source of chronic anxiety. Here, we review studies that have identified neural correlates of stress-induced modulation of amygdala structure and function — from cellular mechanisms to their behavioural consequences. The unique features of stress-induced plasticity in the amygdala, in association with changes in other brain regions, could have long-term consequences for cognitive performance and pathological anxiety exhibited in people with affective disorders.

Stress & Gedächtnis: verbesserte Konsolidierung

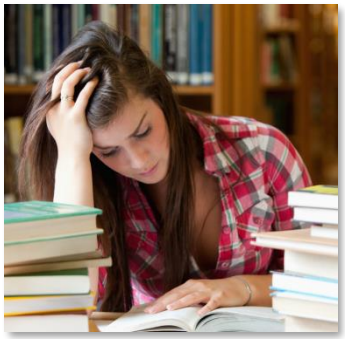


Stress & Gedächtnis: verschlechterter Abruf



Stress & Gedächtnis: phasenabhängige Effekte

unterschiedliche Effekte je nach Gedächtnisphase:



Enkodierung

Konsolidierung

Abruf

\+

Stress

\-

Stress

Stress & Gedächtnis: Amygdala

(Roozendaal et al., 2009)

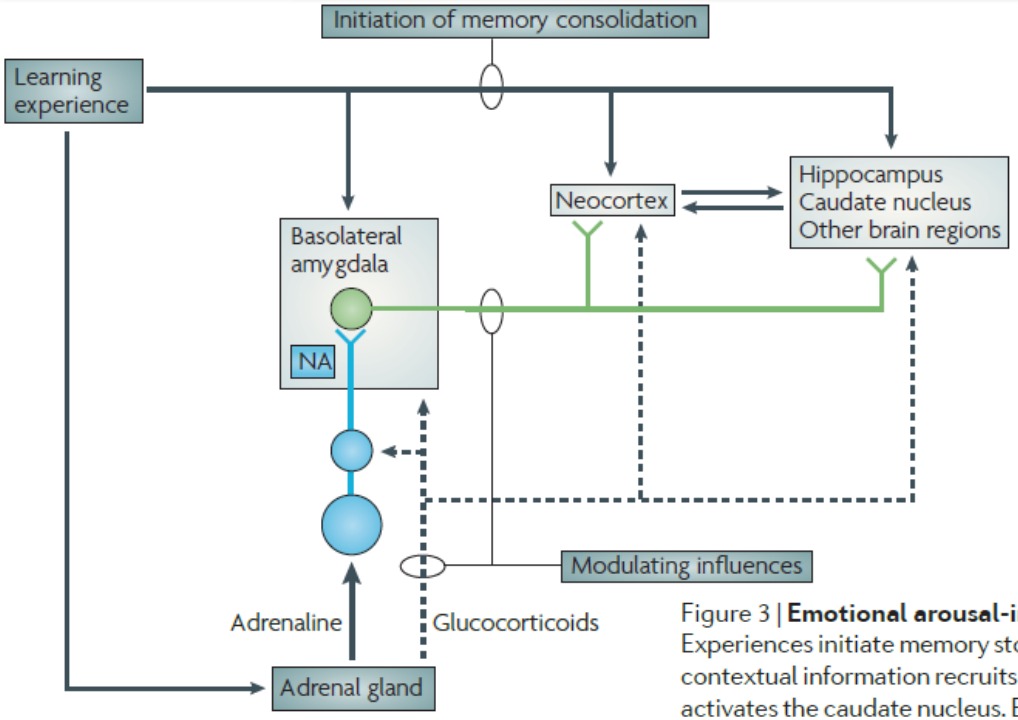


Figure 3 | **Emotional arousal-induced modulation of memory consolidation.** Experiences initiate memory storage in different brain regions. For example, spatial or contextual information recruits the hippocampus, whereas procedural information activates the caudate nucleus. Emotionally arousing experiences also release adrenaline and glucocorticoids from the adrenal gland and induce the release of noradrenaline (NA) in the basolateral complex of the amygdala (BLA). Adrenaline, which does not cross the blood–brain barrier, induces the release of noradrenaline in the BLA by activating vagal afferents to the nucleus of the solitary tract. Noradrenergic neurons in the nucleus of the solitary tract project both directly and indirectly to the BLA. Glucocorticoids freely enter the brain and can directly bind to glucocorticoid receptors in the BLA. Such stress-induced BLA activity modulates memory consolidation by influencing neuroplasticity in other brain regions. In addition, stress hormones directly activate other brain regions to enhance memory consolidation (dotted arrows); these effects also depend on intact BLA functioning[10]. Figure is modified, with permission, from REF. 10 © (2000) American Association for the Advancement of Science.

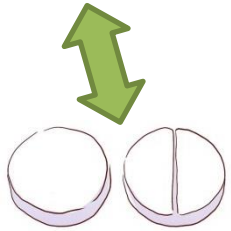
←Interaktion von Cortisol mit Noradrenalin (NA) in basolateraler Amygdala

Stress & Gehirn: Gedächtniskonsolidierung

- zwei Forschungsansätze
- Cortisol & Gedächtnis
  - **Stress & Gedächtniskonsolidierung**
  - Stress & Gedächtnisabruf
- chronischer Stress

Stress, Cortisol & Gedächtnis-konsolidierung: Humanstudien

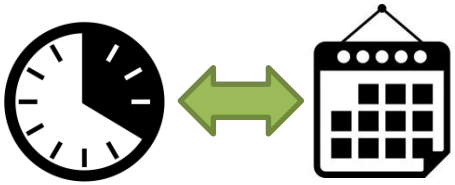
unterschiedliche Vorgehensweisen:



- Stressexposition vs. pharmakologische Cortisoladministration

- Treatment vor vs. direkt nach Lernphase

- Abruftestung Minuten vs. Tage später



Stress & Gedächtniskonsolidierung: Humanstudie (Cahill et al., 2003)

Enhanced Human Memory Consolidation With Post-Learning Stress: Interaction With the Degree of Arousal at Encoding

Larry Cahill,[1] Lukasz Gorski, and Kathryn Le

Department of Neurobiology and Behavior, and Center for the Neurobiology of Learning and Memory, University of California, Irvine, California 92697-3800, USA

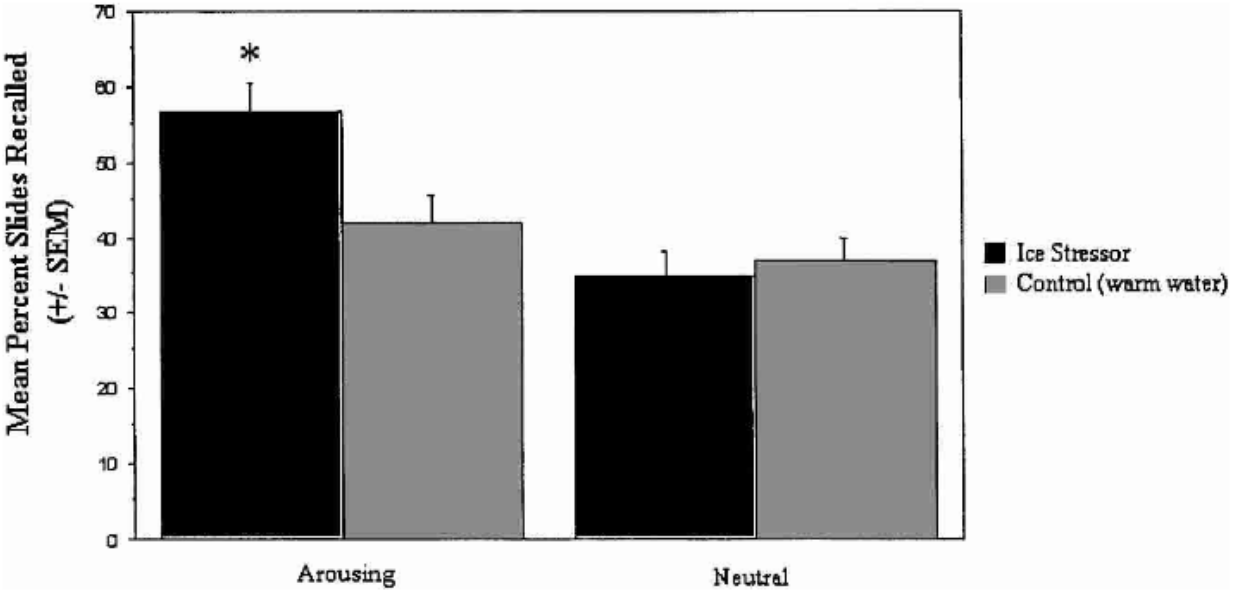
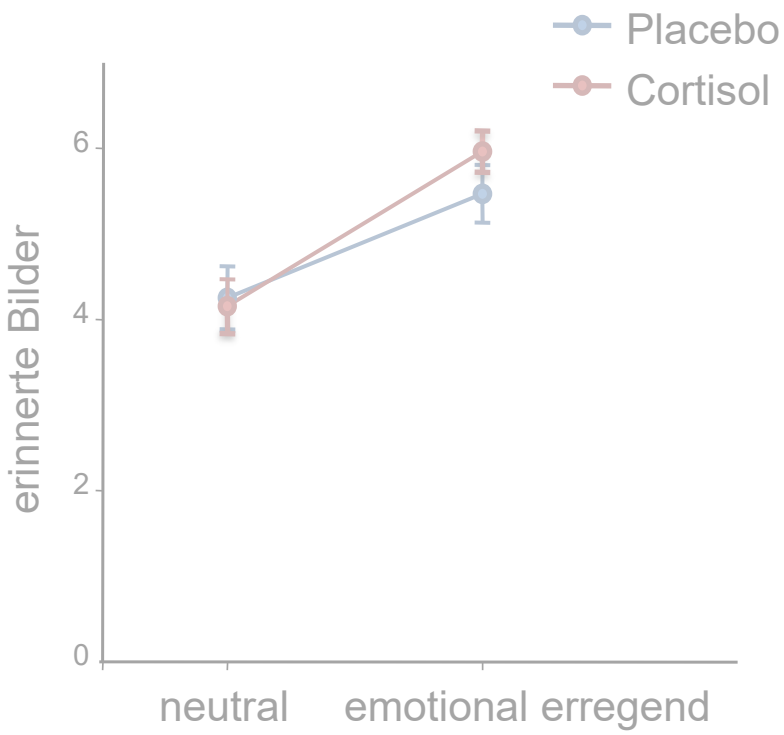


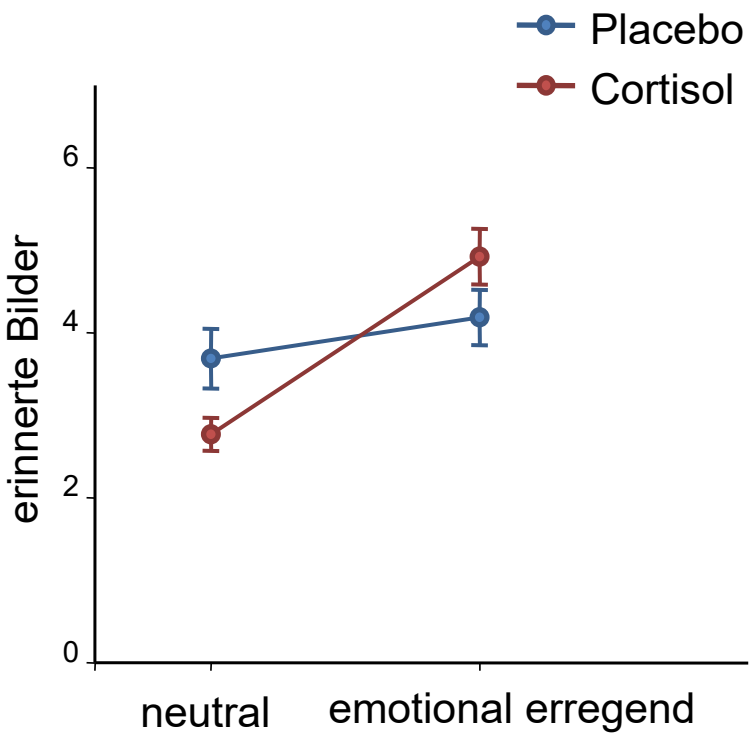
Figure 2 Average (± SEM) percent recall of slides defined as arousing and as neutral by the CPS and control groups. * = $P < .02$ from corresponding control value.

Cortisol & Gedächtnis-konsolidierung: Humanstudie

(Kuhlmann & Wolf, 2006)



direkter Abruf



verzögerter (24 h) Abruf

Cortisol & Gedächtnis-konsolidierung: Tierstudie (Roozendaal et al., 2009)

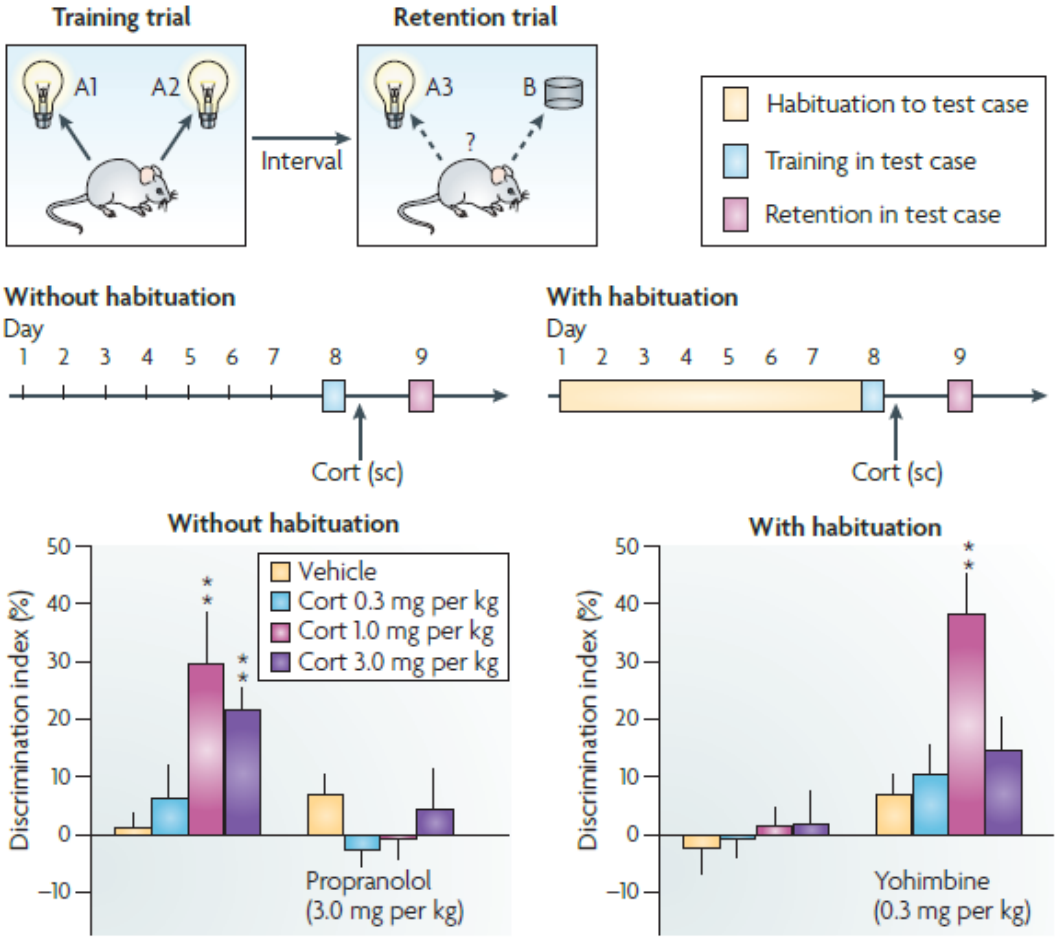
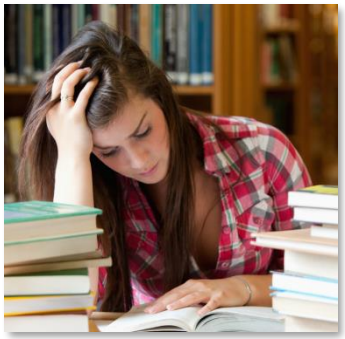


Figure 2 | **Glucocorticoid effects on memory consolidation for object-recognition training require arousal-induced noradrenergic activation.** Rats were either habituated to the training context for 7 days or not habituated. On day 8 they were given a 3 min training trial during which they could freely explore two identical objects, followed by systemic drug administration. Retention was tested 24 h later by placing the rats back into the apparatus for 3 min; now one object was similar to that explored during training whereas the other object was novel. Corticosterone (Cort) injections selectively enhanced memory consolidation in context-naive rats, which showed greater emotional arousal response during training. Systemic post-training administration of the β-adrenoceptor antagonist propranolol blocked this memory enhancement in naive (emotionally aroused) rats. Co-administration of the α2-adrenoceptor antagonist yohimbine with corticosterone enhanced object recognition memory in habituated (emotionally non-aroused) rats. The data represent the discrimination index (%) on a 24 h retention trial, expressed as the mean ± the standard error. The discrimination index was calculated as the difference in time spent exploring the novel and the familiar object, expressed as the ratio of the total time spent exploring both objects. ** p<0.0001 vs vehicle41. Figure is modified, with permission, from REF. 41 © (2006) National Academy of Sciences

Stress & Gedächtnis: phasenabhängige Effekte

unterschiedliche Effekte je nach Gedächtnisphase:



Enkodierung

Konsolidierung

Abruf

+/-

Stress

+

Stress

-

Stress

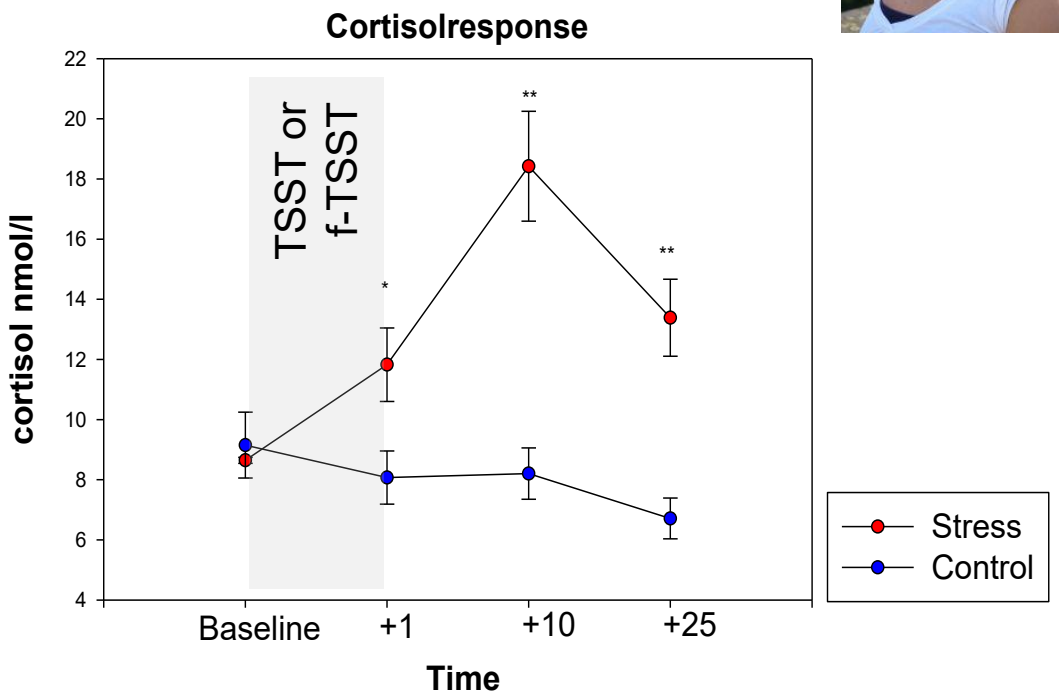
Psychosozialer Stress im Labor: Video – Quarks & Caspers

Stress & Gedächtnis: Erinnerungen an eine stressvolle Episode

(Wiemers, Schoofs & Wolf, 2013)

TSST

f-TSST

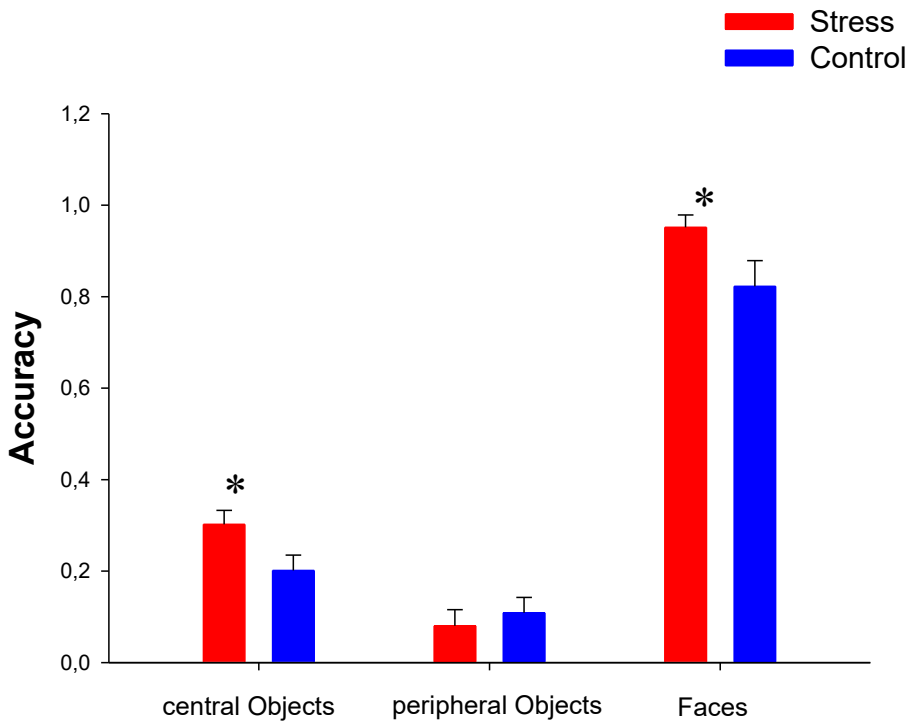


Stress & Gedächtnis: Erinnerungen an eine stressvolle Episode

(Wiemers, Sauvage, Schoofs, Hamacher-Dang & Wolf, 2013)

TSST

f-TSST



Memory trace of a stressful episode

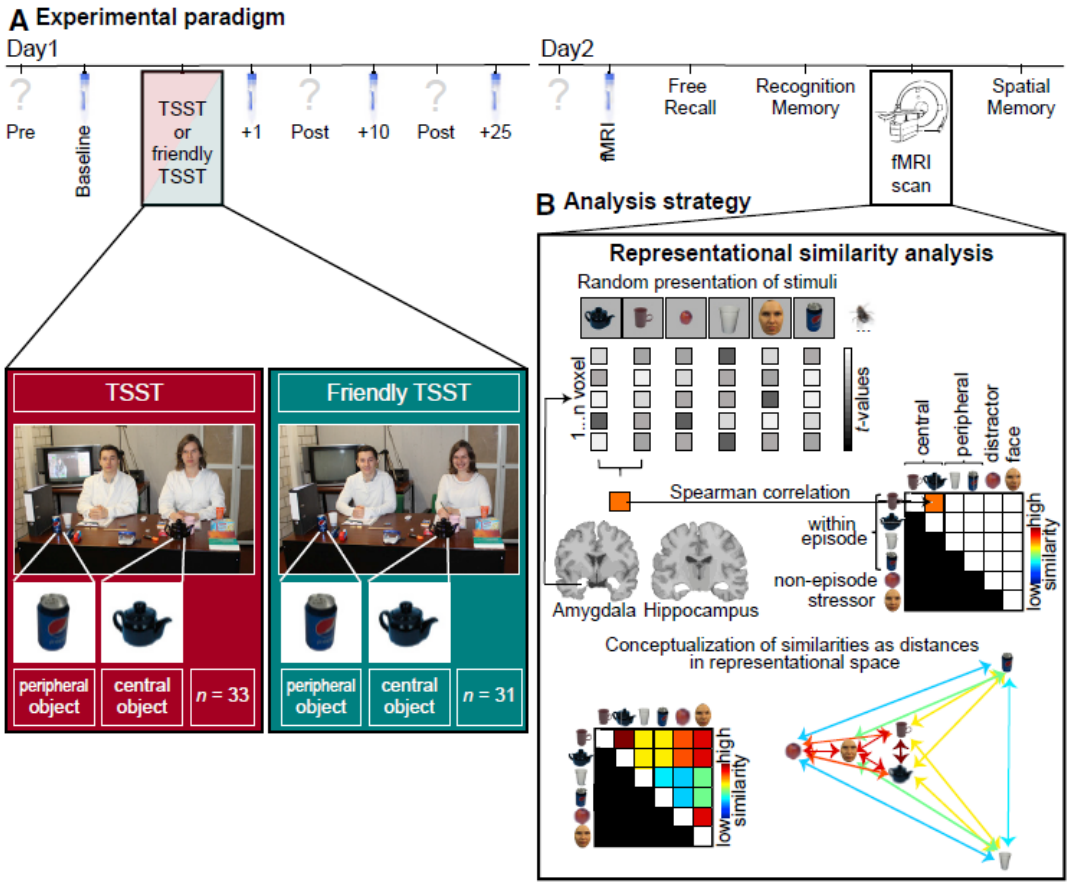


Figure 1. Experimental paradigm and analysis strategy

(A) The experiment was performed on two consecutive days. On day 1, participants conducted either the Trier social stress test (TSST) or a non-stressful control test ("friendly TSST," f-TSST). They provided saliva samples to assess cortisol levels and rated their affect and self-esteem before and after the (f-)TSST. On day 2, they performed a free recall and a recognition memory test of objects that occurred in the (f-) TSST and were then presented pictures of these objects and of the committee members in the MRI scanner.

(B) Analysis strategy. Top: we applied representational similarity analysis on data from the amygdala and the hippocampus by correlating voxel patterns between pairs of pictures. More specifically, we extracted the similarity between voxel patterns as an indicator of how similar two objects, or an object and a face, were represented in the brain (e.g., LaRocque et al.[10]). Bottom: higher correlations indicate that voxel patterns are more similar to one another, i.e., that their distance in representational space is lower.

See also Figure S1 and Table S1.

Bierbrauer et al., (2021) Current Biology

The memory trace of a stressful episode

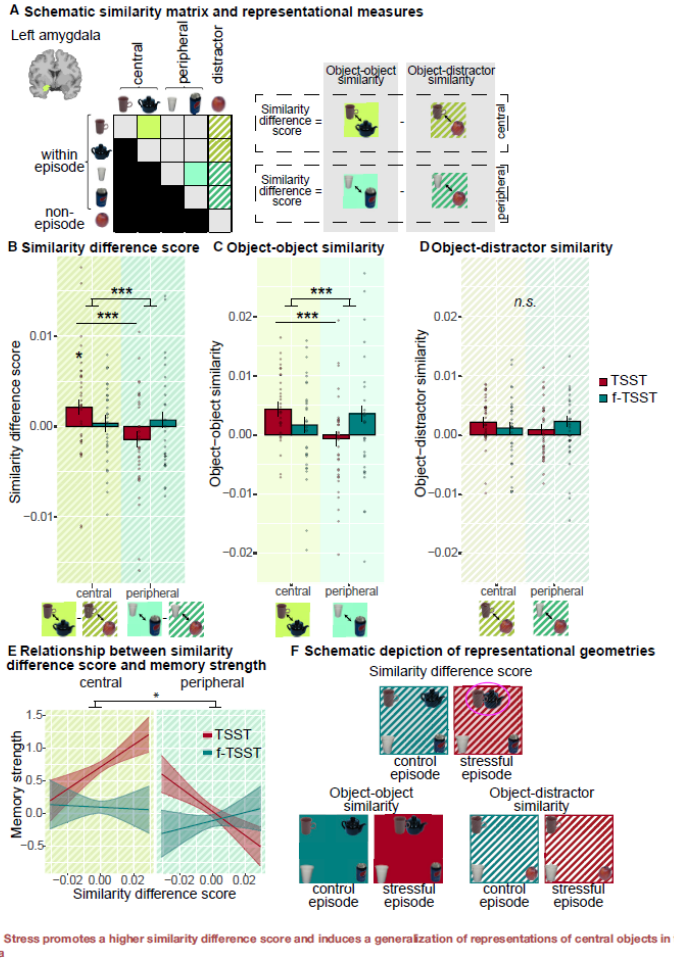


Figure 3. Stress promotes a higher similarity difference score and induces a generalization of representations of central objects in the left amygdala

In a stressful episode, central objects moved closer in representational space.

Bierbrauer et al., (2021) Current Biology

Memory trace of a stressful episode

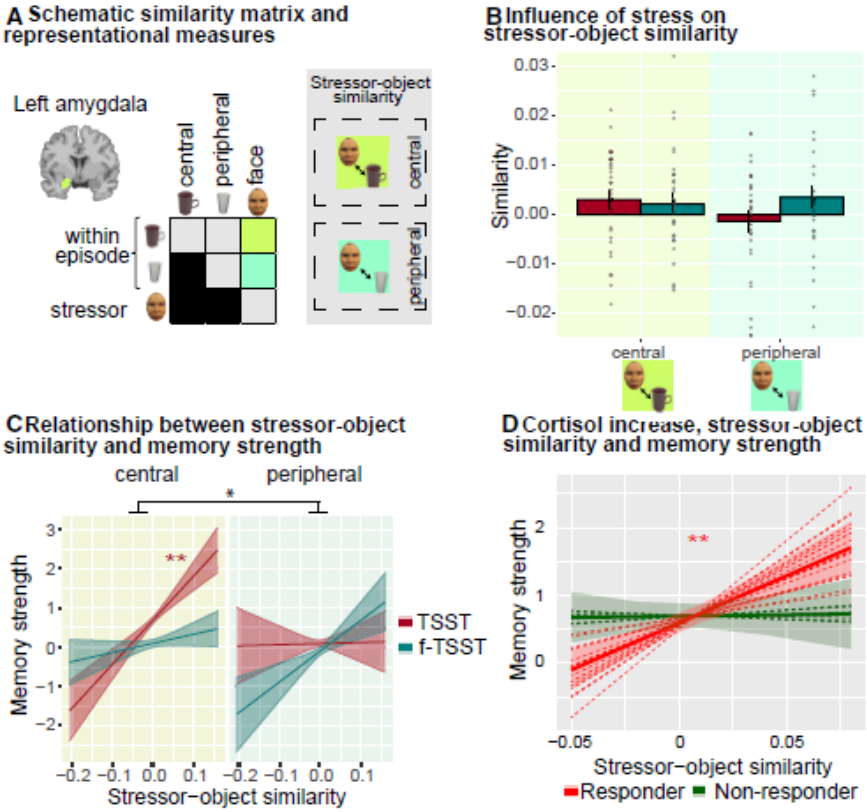


Figure 4. Stressor-object similarity accounts for memory benefits of stress

(A) Schematic depiction of representational similarity analysis in the left amygdala. Stressor-object similarity refers to the representational similarity of central or peripheral objects to the faces of the committee members.

(B) Stressor-object similarity does not differ between object types or between TSST (red bars) and f-TSST (cyan bars).

(C) Significant three-way interaction: stressor-object similarity predicts memory strength for central objects in the TSST (red lines), but not in the f-TSST (cyan lines).

(D) Selective effects of stressor-object similarity on memory strength for cortisol responders; depicted are model estimates for responders (red) and non-responders (green) and for single participants in the respective groups (dashed lines).

(E) Central objects that moved closer to the stressor in representational space were more likely to be remembered.

Dots show descriptive means for each participant; error bars show standard errors; $^{*}p_{corrected} < 0.05$; $^{**}p_{corrected} < 0.01$; see also Figures S3 and S4 and Tables S2 and S4.

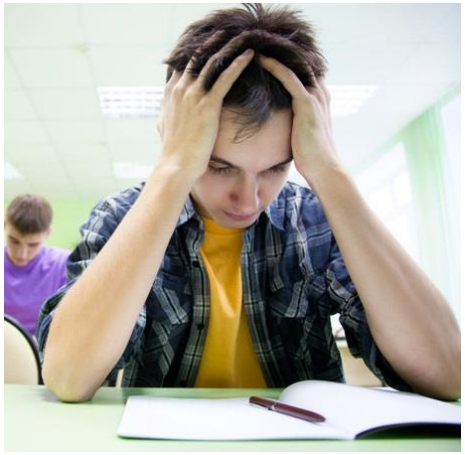
Bierbrauer et al., (2021) Current Biology

Stress & Gehirn: Gedächtnisabruf

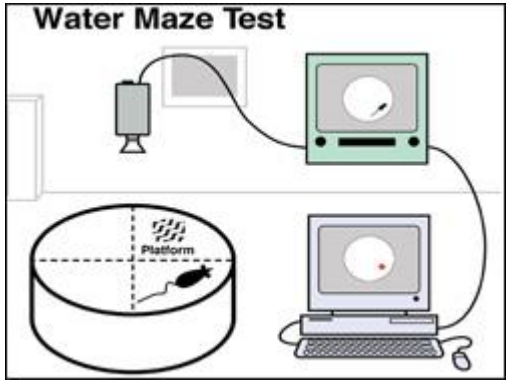
- zwei Forschungsansätze
- Cortisol & Gedächtnis
  - Stress & Gedächtniskonsolidierung
  - **Stress & Gedächtnisabruf**
- chronischer Stress

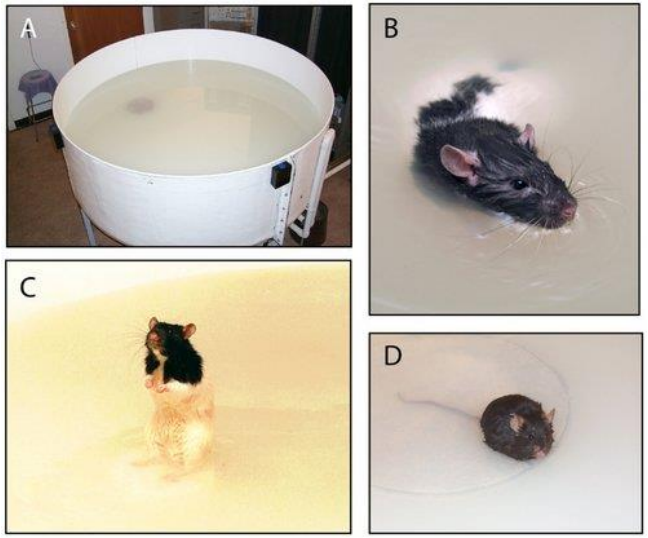
Stress & Gehirn: Gedächtnisabruf – die „negative“ Seite

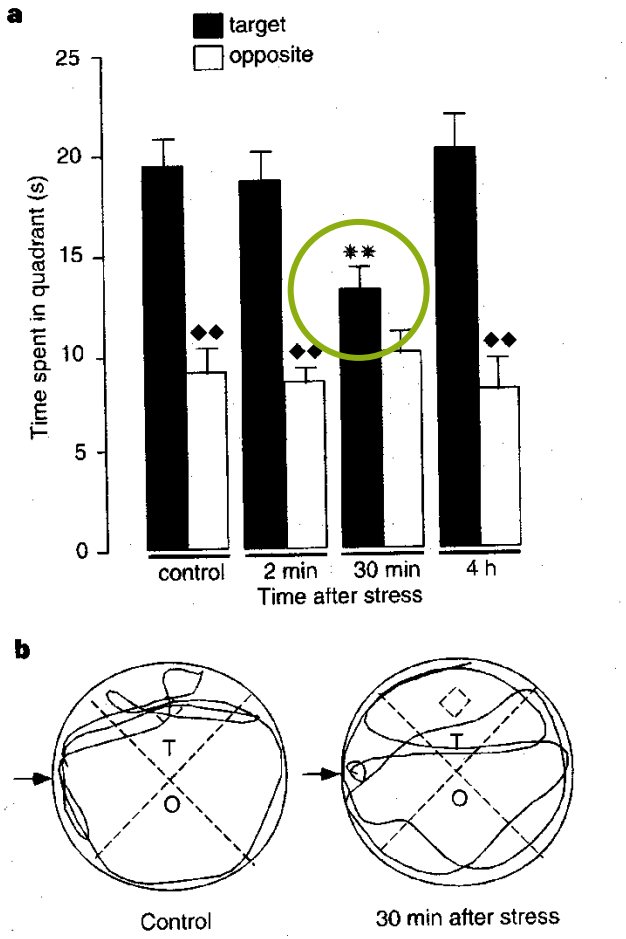
verschlechterter Gedächtnisabruf



Stress & Gedächtnisabruf: Nagetiere (De Quervain et al., 1998)

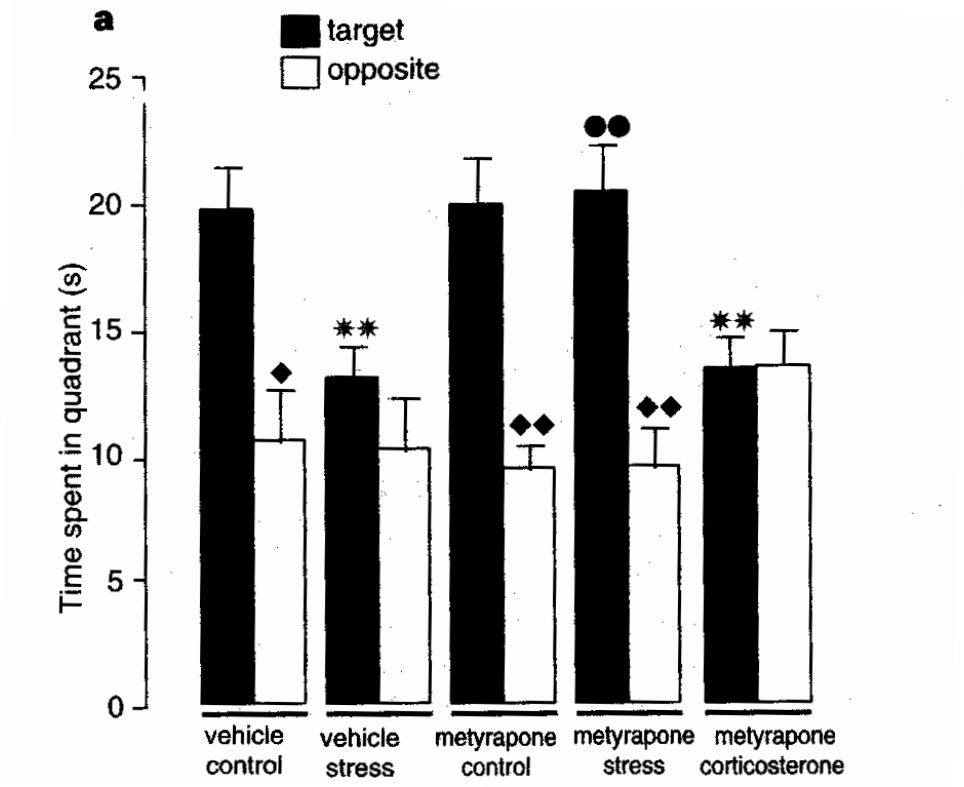






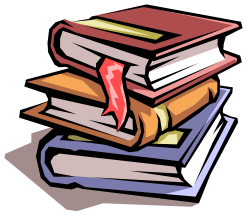
Stress & Gedächtnisabruf: Nagetiere (De Quervain et al., 1998)

- Abruf-verschlechternde Effekte von Stress durch Corticosterone vermittelt



Stress & Gedächtnisabruf: Cortisol vs. Placebo

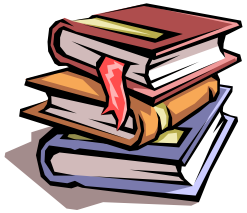
- Kuschelhormon = Oxytocin
- Stresshormon = Cortisol



Abruf
Kuschelhormon = ??

Cortisol

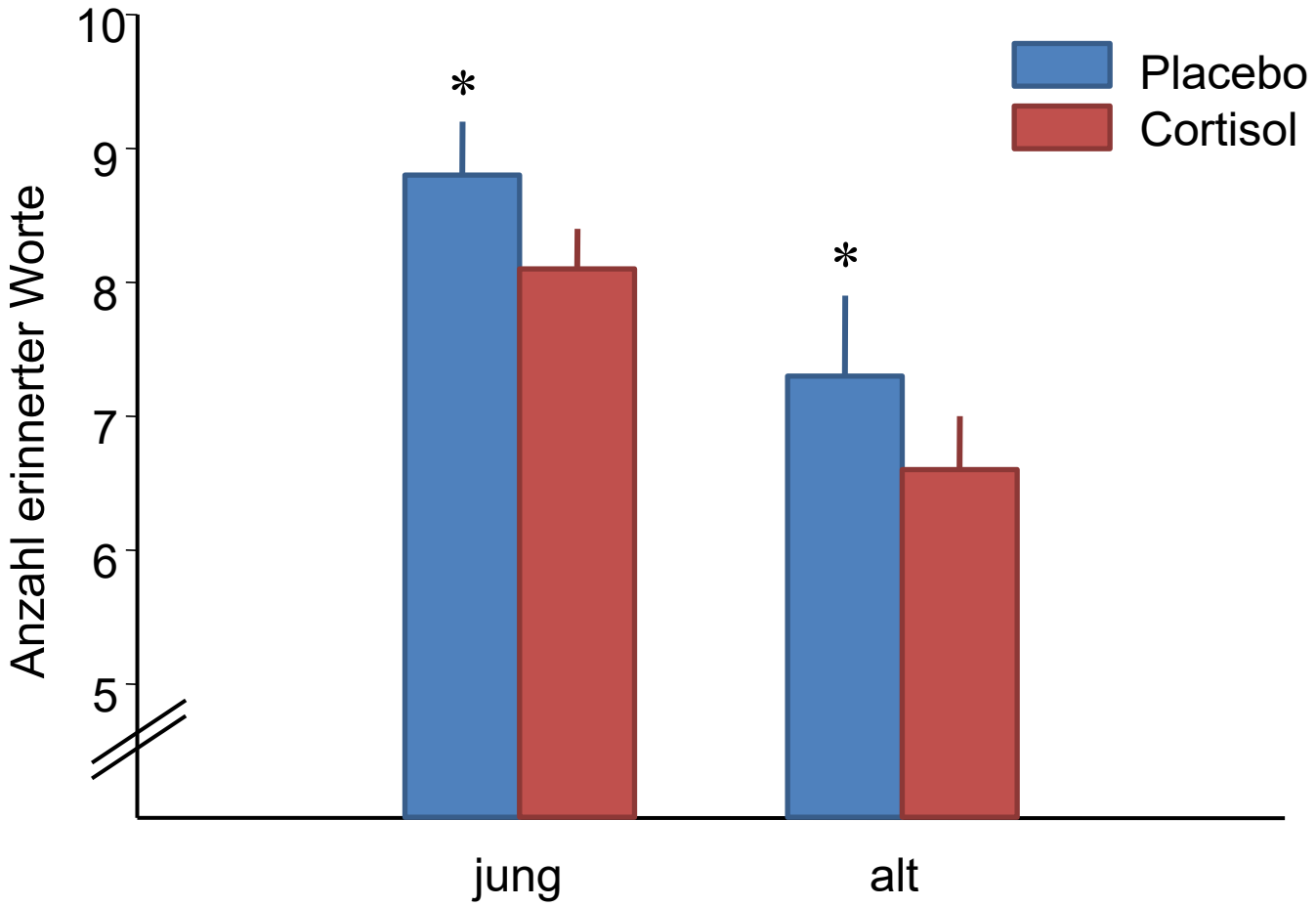
- Kuschelhormon = Oxytocin
- Stresshormon = Cortisol



Abruf
Kuschelhormon = Oxytocin

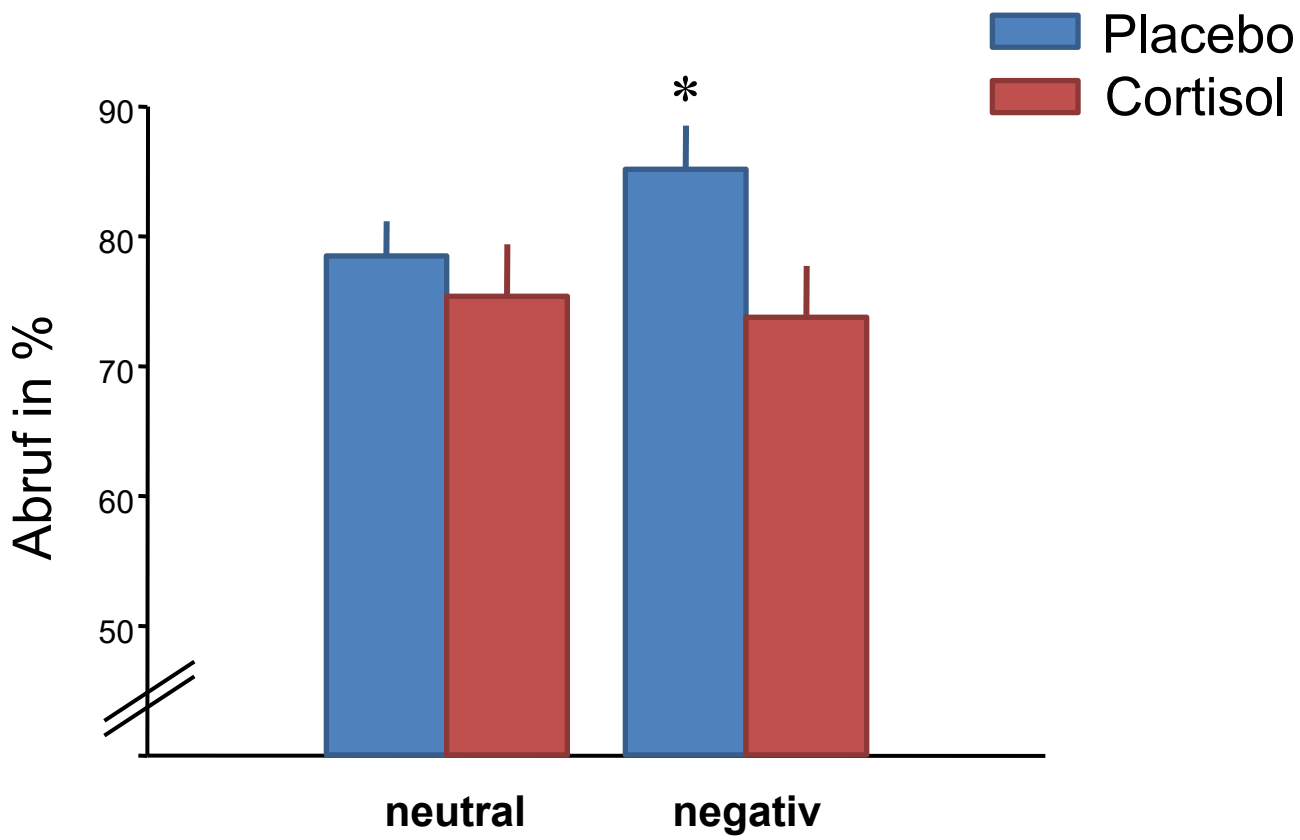
Placebo

Stress & Gedächtnisabruf: Cortisol vs. Placebo (Wolf et al., 2001)



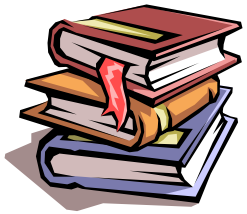
Cortisol & Gedächtnisabruf: Emotionalität des Lernmaterials

(Kuhlmann, Kirschbaum & Wolf, 2005)



Stress & Gedächtnisabruf: Stress vs. kein Stress (Kuhlmann, Piel & Wolf, 2005)

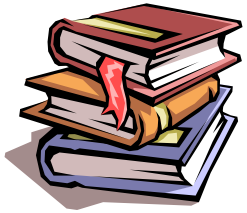
- Kuschelhormon = Oxytocin
- Stresshormon = Cortisol



Abruf
Kuschelhormon = ??

Stress

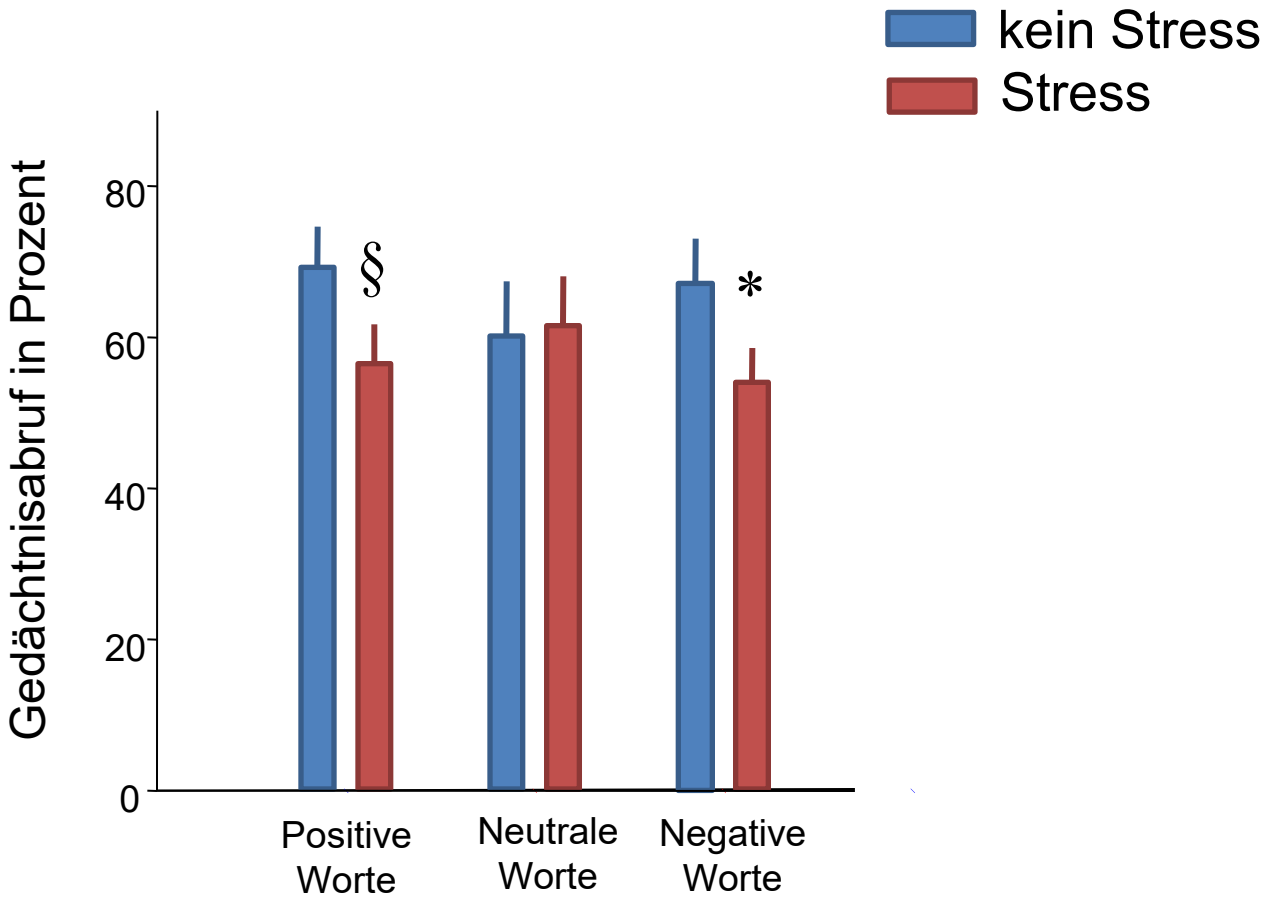
- Kuschelhormon = Oxytocin
- Stresshormon = Cortisol



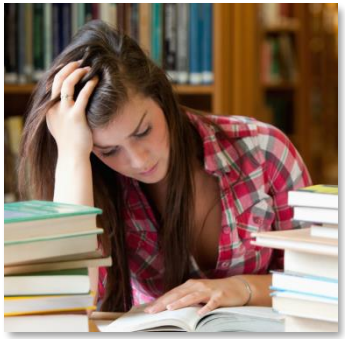
Abruf
Kuschelhormon = Oxytocin

kein Stress

Stress & Gedächtnisabruf: Stress vs. kein Stress (Kuhlmann, Piel & Wolf, 2005)



Cortisol & Gedächtnisabruf: PET-Studie (De Quervain et al., 2003)



Enkodierung

Konsolidierung

Abruf

Cortisol / Placebo

Cortisol & Gedächtnisabruf: PET-Studie (De Quervain et al., 2003)

European Journal of Neuroscience, Vol. 17, pp. 1296–1302, 2003

Glucocorticoid-induced impairment of declarative memory retrieval is associated with reduced blood flow in the medial temporal lobe

Dominique J.-F. de Quervain,[1] Katharina Henke,[1] Amanda Aerni,[1] Valerie Treyer,[2] James L. McGaugh,[3] Thomas Berthold,[2] Roger M. Nitsch,[1] Alfred Buck,[2] Benno Roozendaal[3] and Christoph Hock[1]

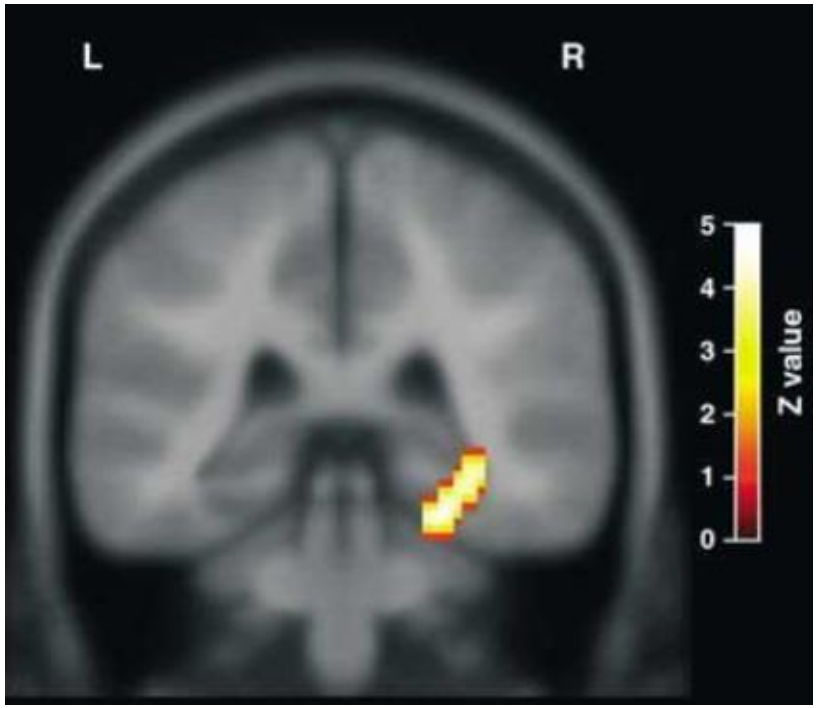


FIG. 2. Decrease in rCBF in the right posterior MTL as imaged by subtracting the cortisone from the placebo condition over all memory retrieval tasks. Activations are overlaid on canonical T1-weighted MR images of SPM99. For illustrative purposes, data are thresholded at $P < 0.0001$, uncorrected for multiple comparisons. The peak of cortisone-induced decrease in rCBF in the right MTL is located in the parahippocampal gyrus at coordinate position (26, −36, −20).

Cortisol & Gedächtnisabruf: Hippocampus – fMRT-Studie

(Oei, Elzinga, Wolf et al., 2007)

- reduzierte Aktivität im Hippocampus

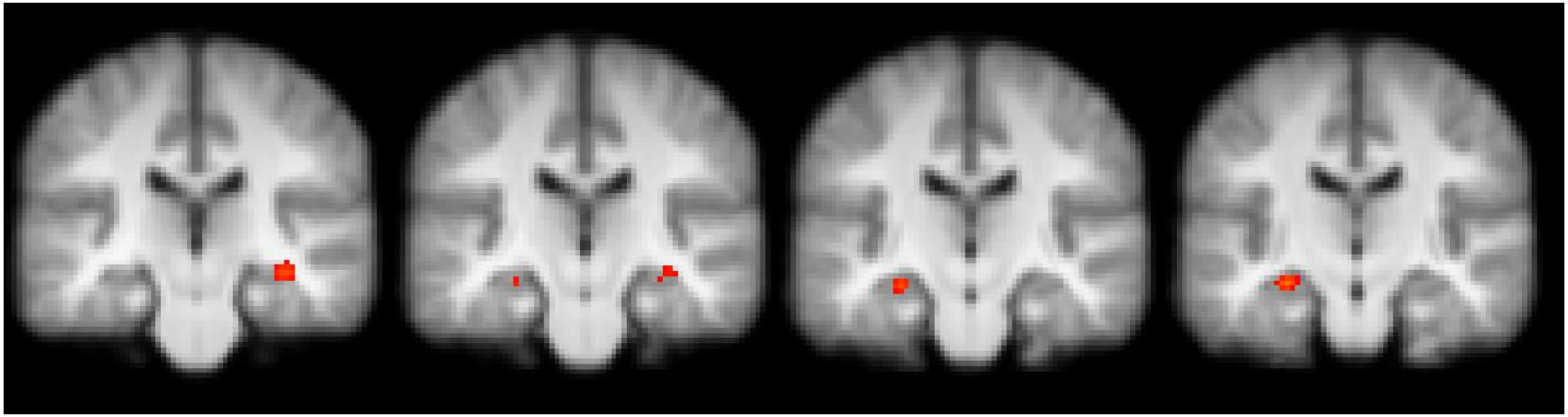
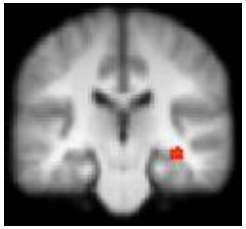


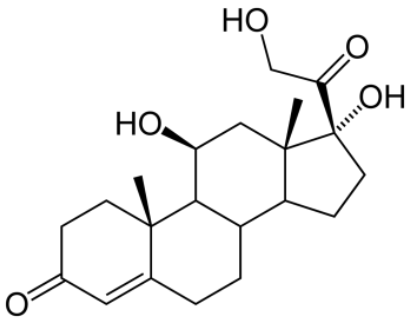
Fig. 2 Coronal slices showing significant hydrocortisone-induced decreases in activation as compared with placebo in left and right hippocampus ($z>2.3$, uncorrected p). *Left in the image* is left in the brain

Stress & Gedächtnisabruf: Fazit

Stress

reduzierte Hippocampus-Aktivität



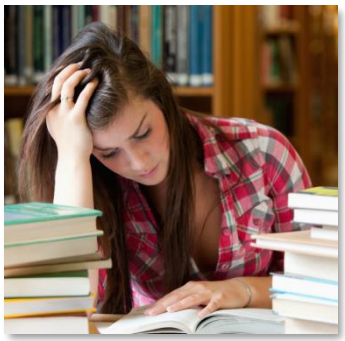


erhöhtes Cortisol

schlechter Abruf

Stress & Gedächtnis: phasenabhängige Effekte

entgegengesetzte Effekte auf Konsolidierung & Abruf:



Enkodierung

Konsolidierung

Abruf

Stress

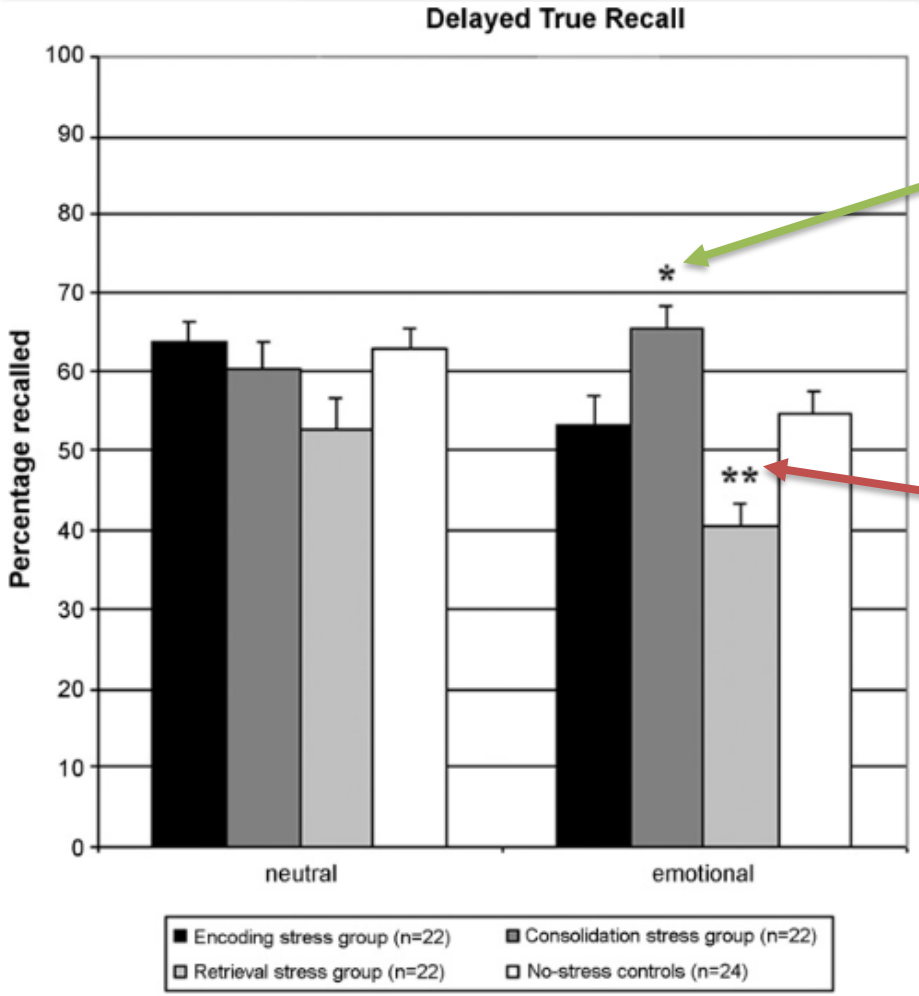
Stress

→ **verbesserte Konsolidierung:**
- Kuhlmann & Wolf (2006) *Behav Neurosci*
- Smeets et al. (2008) *PNEC*
- Preuss & Wolf (2009) *NLM*
- Wolf (2012) *Stress*

→ **beeinträchtigter Abruf:**
- Wolf et al (2001) *Behav Neurosci*
- Kuhlmann et al. (2005) *J Neurosci*
- Coluccia et al. (2008) *J Neurosci*
- Schwabe & Wolf (2009) *CABN*
- Merz et al. (2010) *Behav Neurosci*

Stress & Langzeitgedächtnis: phasenabhängige Effekte

(Smeets, Otgaar, Candel & Wolf, 2008)



* $p < 0.05$ compared to encoding stress group, retrieval stress group, and no-stress control group
** $p < 0.05$ compared to encoding stress group, consolidation stress group, and no-stress control group

Stress zu Beginn der Konsolidierungsphase: **Verbesserung** des emotionalen Gedächtnisses

Stress vor dem Abruf: **Verschlechterung** des emotionalen Gedächtnisses

Figure 2 Delayed true recall of neutral and emotional words. Error bars represent standard error of mean (S.E.).

Stress & Langzeitgedächtnis: phasenabhängige Effekte

(Meta-Analyse von Shields et al., 2017)

Psychological Bulletin

0033-2909/17/$12.00 http://dx.doi.org/10.1037/bul0000100

The Effects of Acute Stress on Episodic Memory: A Meta-Analysis and Integrative Review

Grant S. Shields, Matthew A. Sazma, Andrew M. McCullough, and Andrew P. Yonelinas
University of California, Davis

A growing body of research has indicated that acute stress can critically impact memory. However, there are a number of inconsistencies in the literature, and important questions remain regarding the conditions under which stress effects emerge as well as basic questions about how stress impacts different phases of memory. In this meta-analysis, we examined 113 independent studies in humans with 6,216 participants that explored effects of stress on encoding, postencoding, retrieval, or postreactivation phases of episodic memory. The results indicated that when stress occurred prior to or during encoding it impaired memory, unless both the delay between the stressor and encoding was very short and the study materials were directly related to the stressor, in which case stress improved encoding. In contrast, postencoding stress improved memory unless the stressor occurred in a different physical context than the study materials. When stress occurred just prior to or during retrieval, memory was impaired, and these effects were larger for emotionally valenced materials than neutral materials. Although stress consistently increased cortisol, the magnitude of the cortisol response was not related to the effects of stress on memory. Nonetheless, the effects of stress on memory were generally reduced in magnitude for women taking hormonal contraceptives. These analyses indicate that stress disrupts some episodic memory processes while enhancing others, and that the effects of stress are modulated by a number of critical factors. These results provide important constraints on current theories of stress and memory, and point to new questions for future research.

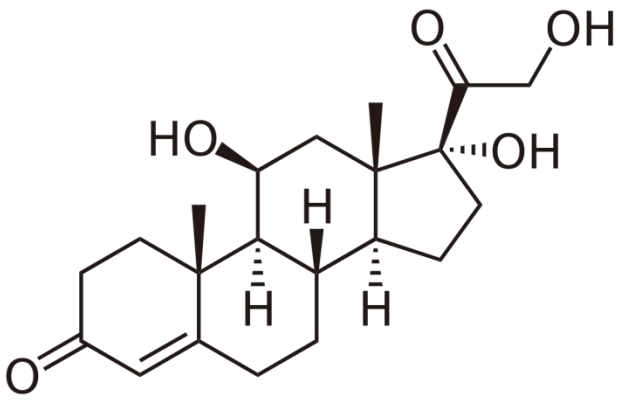
Keywords: acute stress, memory, meta-analysis

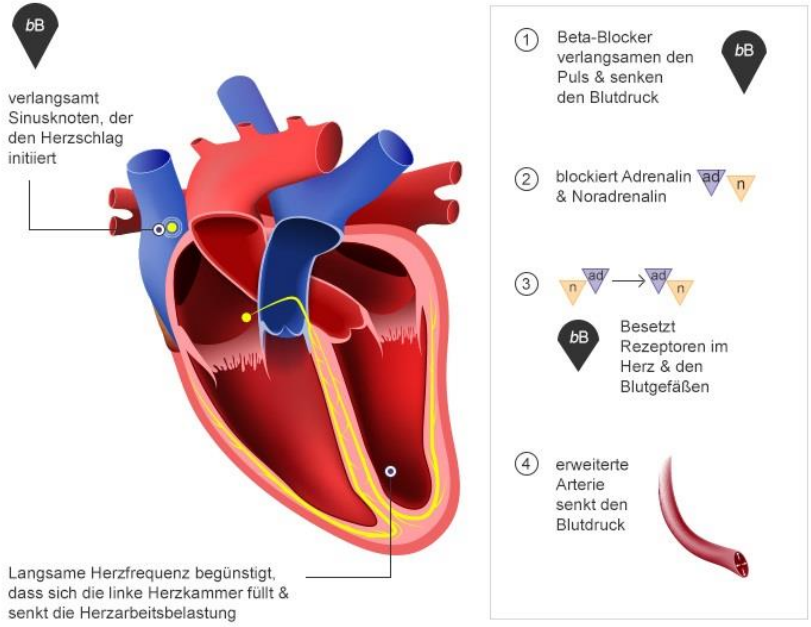
Supplemental materials: http://dx.doi.org/10.1037/bul0000100.supp

Stress & Gedächtnisabruf: negative Effekte – Was tun?

Mögliche pharmakologische Ansatzpunkte:

- Blockade der Cortisolsynthese
- Blockade der noradrenergen Aktivierung der Amygdala (Beta-Blocker)





Cortison & Gedächtnisabruf: Einfluss von Beta-Blockern

(De Quervain et al., 2007)

Preventive Effect of β-Adrenoceptor Blockade on Glucocorticoid-Induced Memory Retrieval Deficits

Dominique J.-F. de Quervain, M.D.

Amanda Aerni, B.S.

Benno Roozendaal, Ph.D.

Objective: Elevated glucocorticoid levels impair retrieval of emotional information, and animal studies indicate that this effect depends on concurrent emotional arousal-induced increases in noradrenergic transmission within the brain. The authors investigated whether the β-adrenoceptor antagonist propranolol blocks glucocorticoid-induced memory retrieval impairments in human subjects.

Method: In a double-blind, placebo-controlled study, 42 healthy volunteers were presented a set of words with variable emotionality and asked to learn them for recall. A day later, cortisone (25 mg), propranolol (40 mg), or both drugs were administered orally 1 hour before a free-recall test.

Results: Cortisone selectively impaired the recall of emotionally arousing words by 42%. This impairment was blocked by the concurrent administration of propranolol. Propranolol alone did not affect recall of either emotional or neutral words.

Conclusions: A pharmacological blockade of β-adrenoceptors prevents glucocorticoid-induced memory retrieval deficits in human subjects. This finding may have important implications for the treatment of memory deficits in hypercortisolemic states, such as stress and depression.

(Am J Psychiatry 2007; 164:967–969)

Cortison & Gedächtnisabruf: Einfluss von Beta-Blockern

(De Quervain et al., 2007)

Methode:

- **3 Gruppen** (jeweils doppelblind Cross-over):
  - Cortison vs. Placebo (n = 14)
  - Cortison plus Propanolol vs. Placebo (n = 14)
  - Propanolol vs. Placebo (n = 14)

- **Versuchsablauf**:
  - Tag 1: Lernphase (60 Wörter, post-hoc Einteilung in Arousal-Quartile)
  - Tag 2: Abrufphase (1 Std nach Substanzgabe)

Cortison & Gedächtnisabruf: Einfluss von Beta-Blockern

(De Quervain et al., 2007)

Ergebnisse: Cortisolspiegel

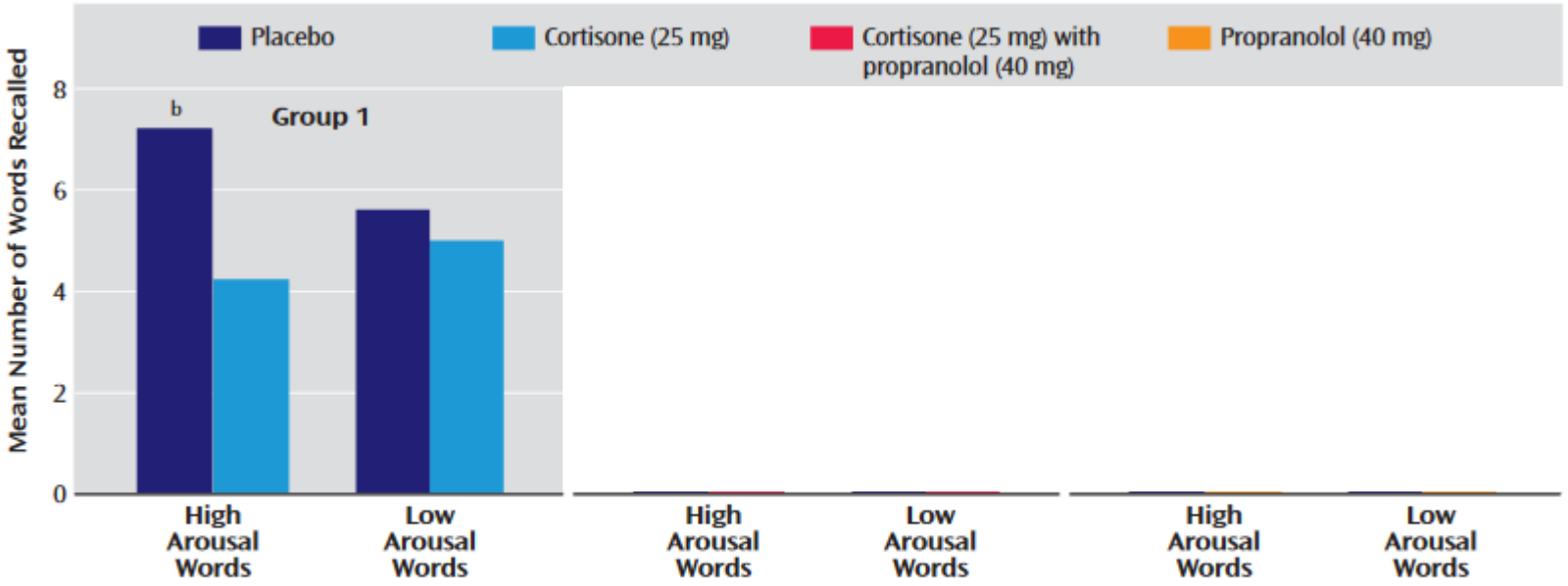
Gruppe	Placebo	aktives Treatment
Gruppe 1 Cortisone	6.4 nmol/l	27.6 nmol/l
Gruppe 2 Cortisone & Propranolol	3.2 nmol/l	35.2 nmol/l
Gruppe 3 Propranolol	3.9 nmol/l	4.1 nmol/l

Cortison & Gedächtnisabruf: Einfluss von Beta-Blockern

(De Quervain et al., 2007)

Ergebnisse: Abrufleistung

FIGURE 1. Effects of Cortisone, Propranolol, and Both Drugs Together on Memory Retrieval of Low- and High-Arousal Words[a]

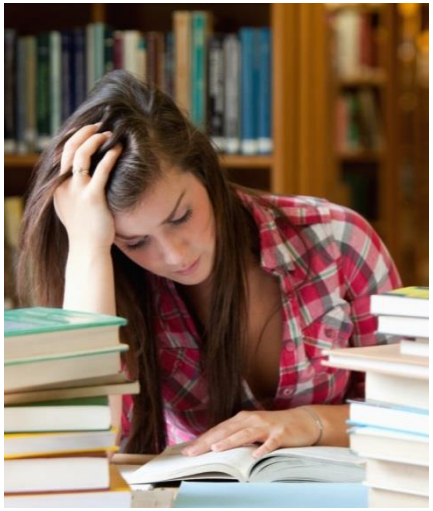


[a] Left panel: Cortisone administered 1 hour before recall testing impaired retrieval of high-arousal words, but not of low-arousal words. Center panel: Concurrent administration of propranolol prevented the impairing effect of cortisone on retrieval of high-arousal words. Right panel: Propranolol given alone 1 hour before recall testing did not affect retrieval of low- or high-arousal words.
[b] $p=0.0001$ compared with the corresponding cortisone condition (two-sided paired t test); N=14 per group.

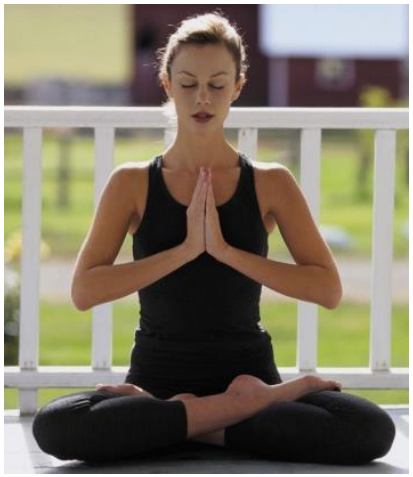
Stress & Gedächtnisabruf: negative Effekte – Was tun?

mögliche Ansatzpunkte:

Lernintensität



Stressmanagement



soziale Unterstützung

Stress & Gehirn: chronischer Stress

RUB

- zwei Forschungsansätze
- Cortisol & Gedächtnis
  - Stress & Gedächtniskonsolidierung
  - Stress & Gedächtnisabruf
- **chronischer Stress**

Nachhaltige Effekte einer akuten Stressexposition (Roozendaal et al., 2009)

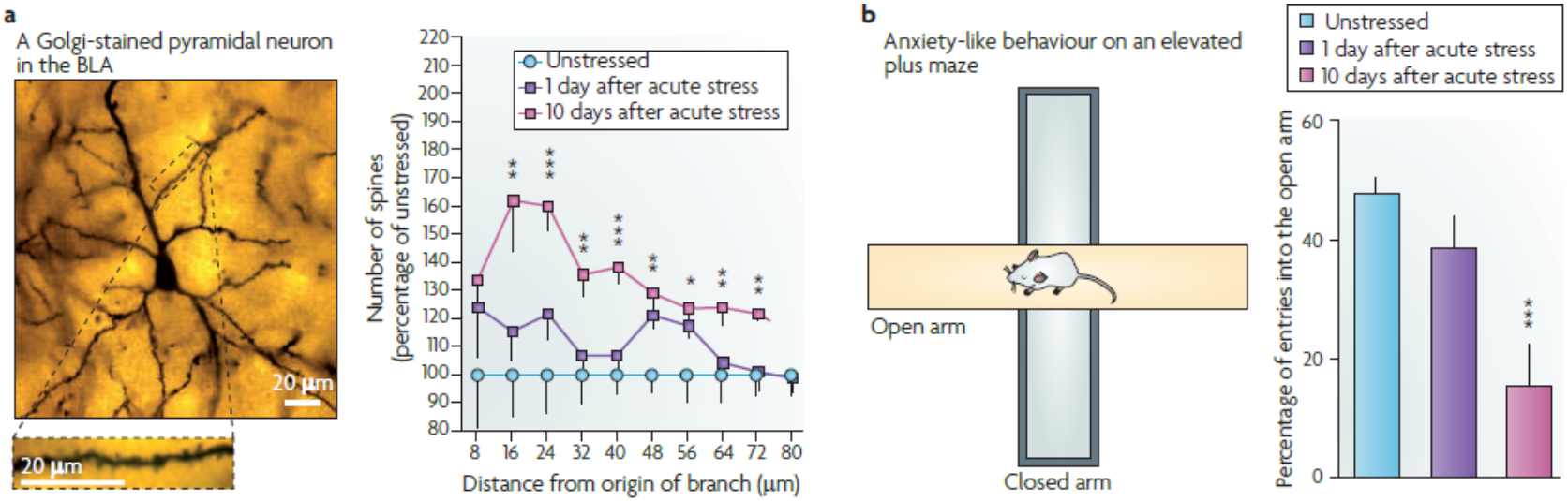
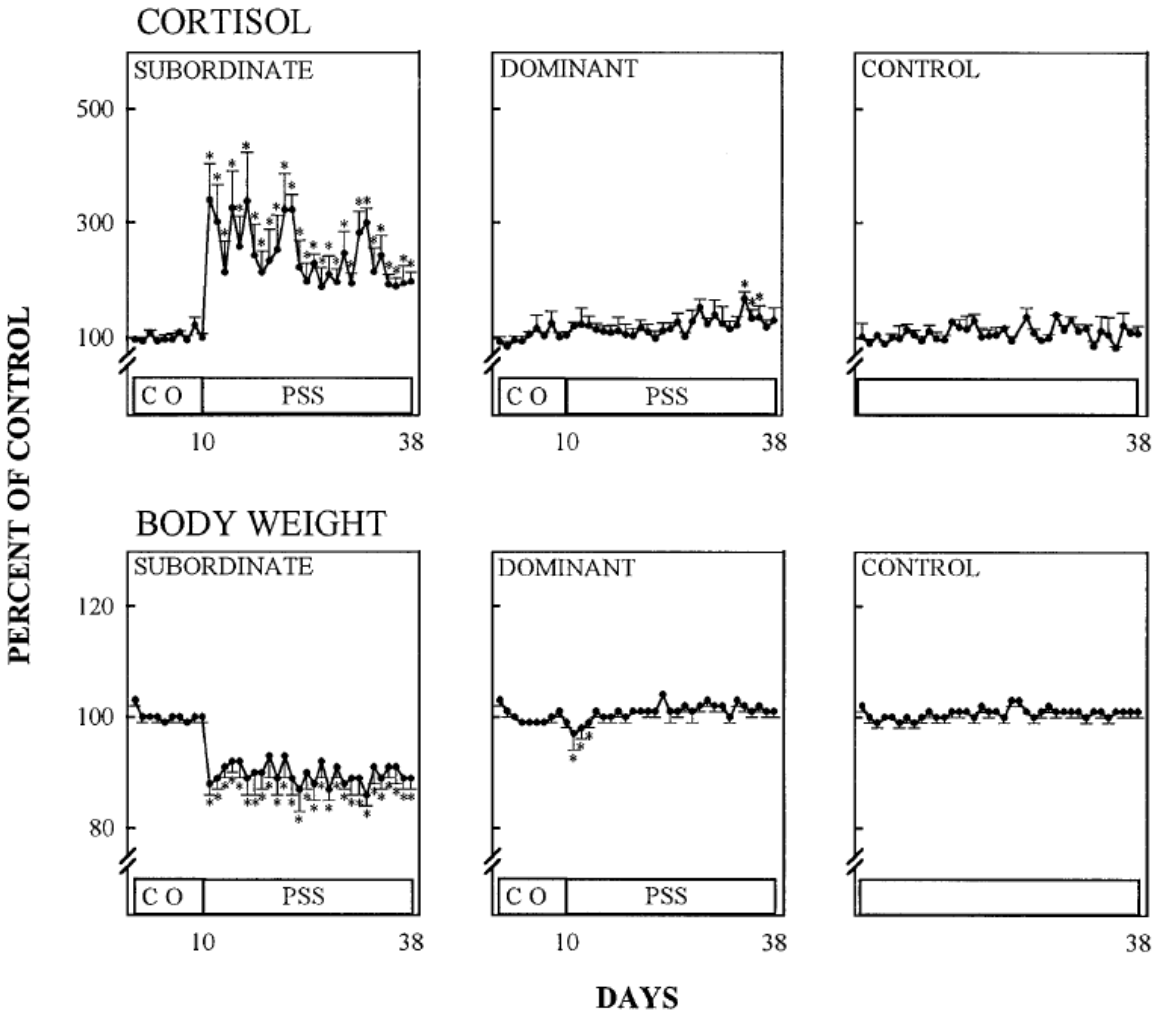


Figure 4 | **A brief exposure to stress triggers a delayed increase in anxiety and spine density in the BLA.** **a** | A single episode of acute stress (immobilization for 2 h) had no significant effect on spine density on principal neurons in the basolateral complex of the amygdala (BLA) 1 day later, which was quantified by counting the number of spines per unit length of dendrite in Golgi-stained neurons. However, 10 days after the same acute stress there was an increase in spine density that was localized to proximal dendritic segments of BLA neurons. **b** | The delayed increase in spine density was paralleled by a gradual build-up in anxiety-like behaviour measured on the elevated plus maze — there was a significant reduction in the number of entries into the open arm of the plus maze 10 days but not 1 day after acute stress[115]. * p<0.05; ** p<0.01; ***p<0.001 (all versus control). Figure is modified, with permission, from REF. 115 © (2005) National Academy of Sciences.

Chronischer Stress: Tupaias

Chronischer Stress: Tupaias

(Magarinos et al., 1996)



Chronischer Stress: Tupaias

(Magarinos et al., 1996)

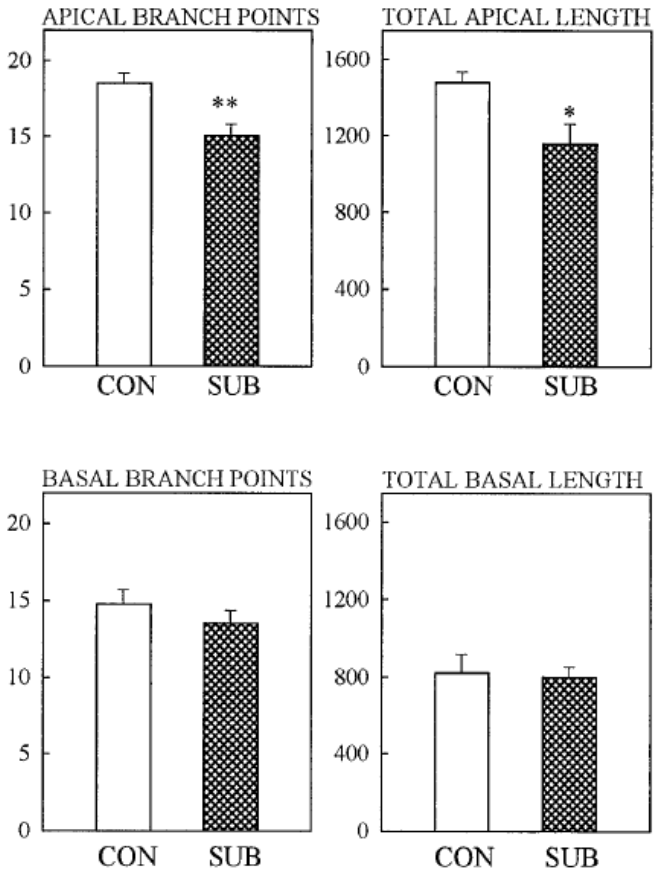


Figure 1. Effect of 28 d of psychosocial stress on dendritic morphology of CA3 pyramidal neurons is shown for apical and basal dendrites. Note that psychosocial stress reduces the number of apical branch points in subordinates (*SUB*) as compared with controls (*CON*). *Bars* represent the mean ± SEM; *double asterisk* indicates $p < 0.01$, and *asterisk* indicates $p < 0.05$ as compared with controls (Student's t test).

Ergebnis: reduzierte Anzahl von Dendriten im Hippocampus

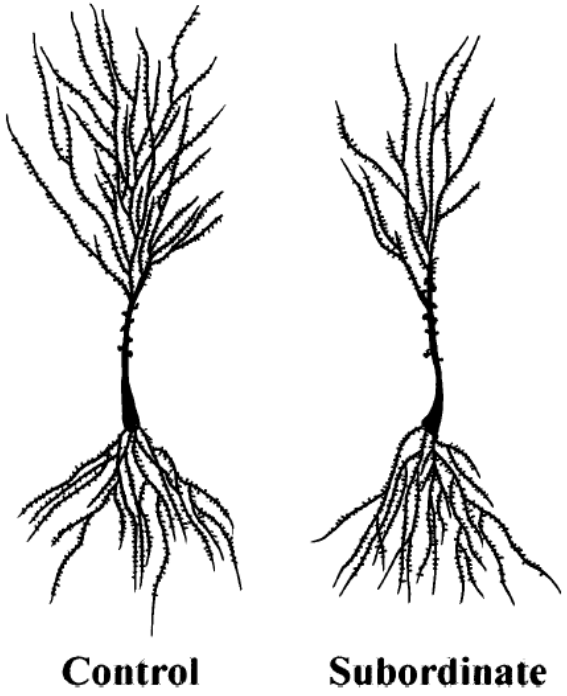


Figure 2. Camera lucida drawings of representative Golgi-impregnated CA3 pyramidal neurons from control (not subjected to stress) and subordinate tree shrews (after 28 d of psychosocial stress). Notice the decreased branching pattern in the subordinate apical dendritic tree as compared with the control.

Chronischer Stress: Hippocampus

(Herbert et al., 2006)

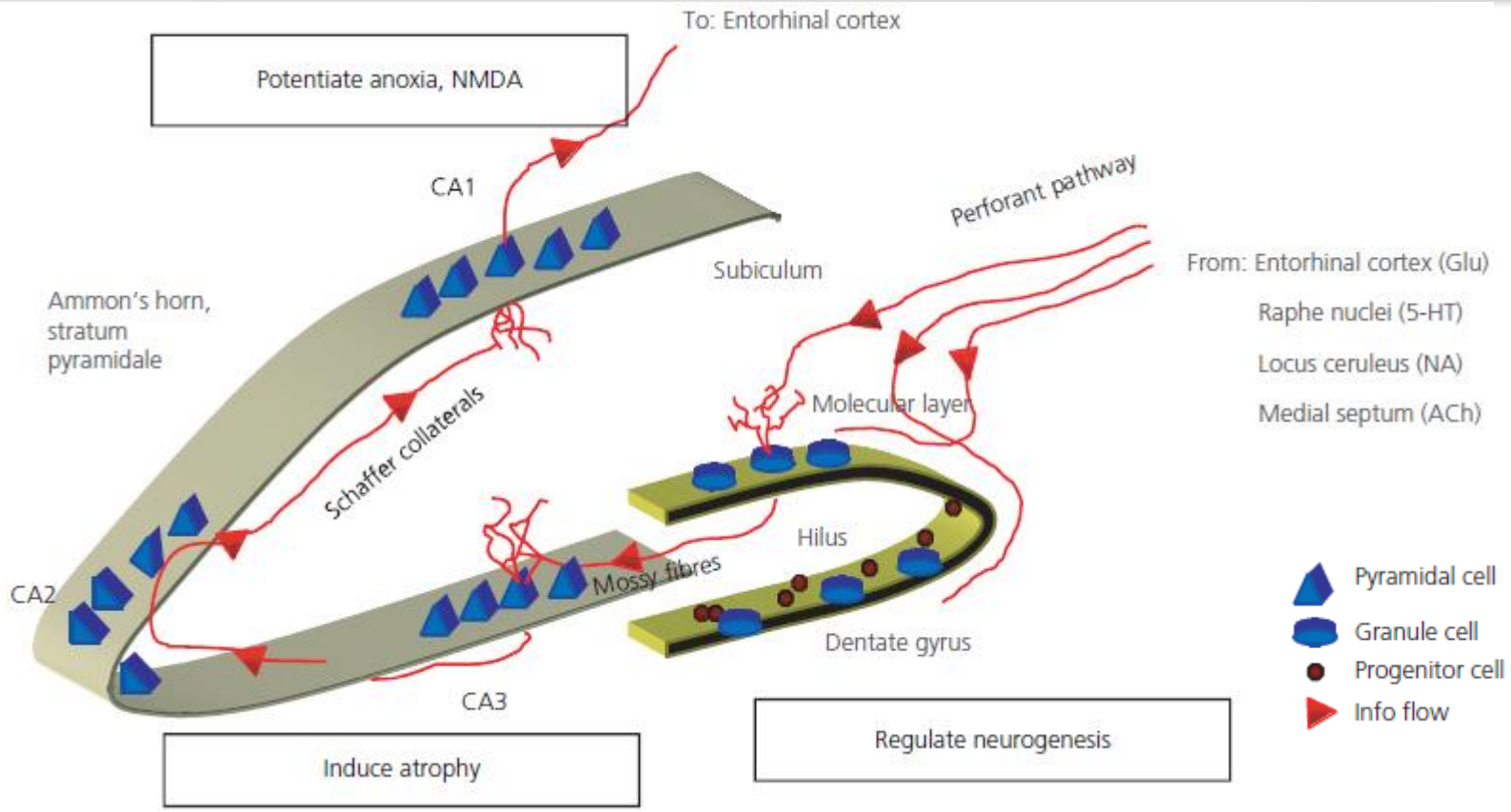
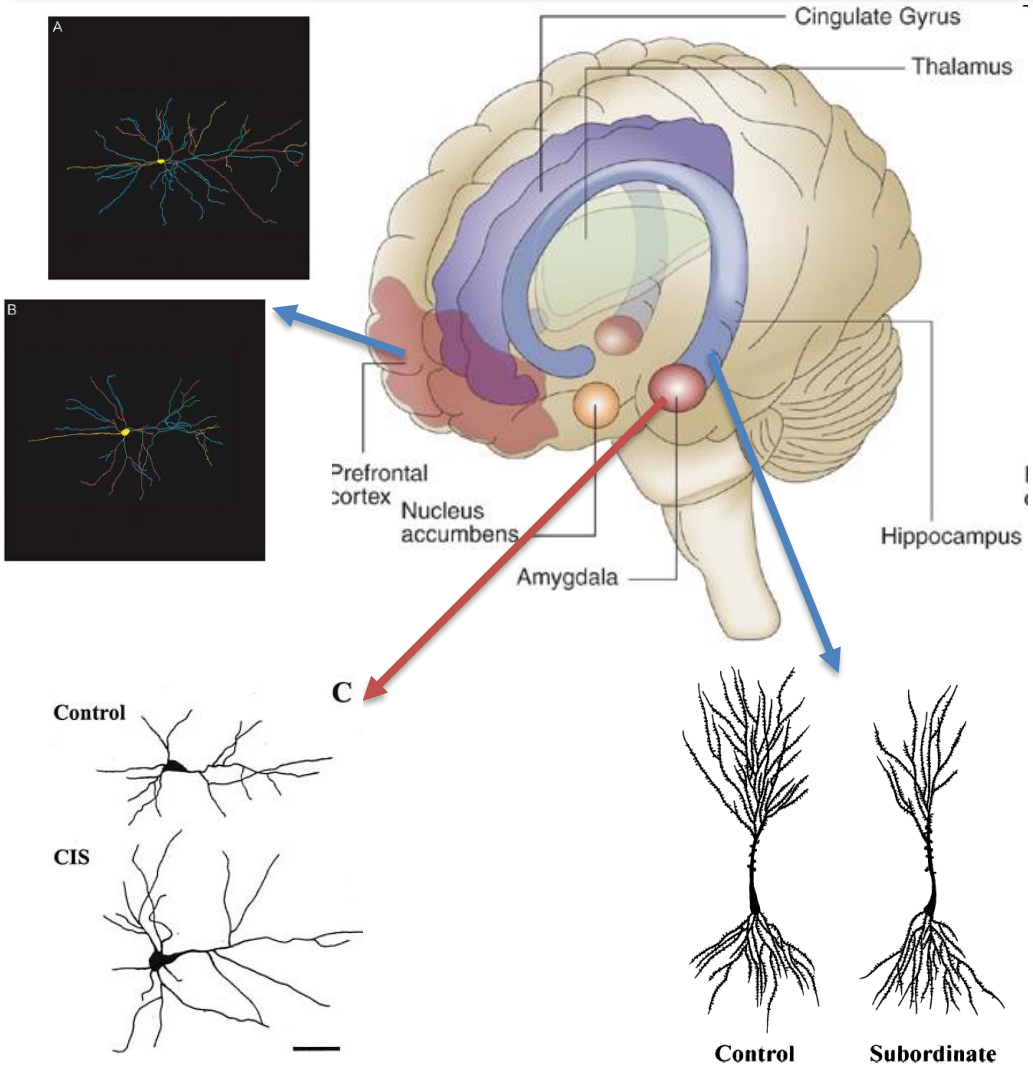


FIG. 3. The sites of glucocorticoid action in the adult hippocampus. The diagram shows the basic circuitry of the hippocampus. Input from various sources (the entorhinal cortex, together with aminergic fibres from the brain stem and basal forebrain) enters mainly through the dentate gyrus. This consists of small granular cells. The progenitor cells from which neurogenesis derives lie on the innermost surface. As they proliferate and then mature, these new neurones migrate outwards. Axons from the dentate granule cells project to the pyramidal cells of CA3, which in turn project to CA1 via the Schaffer collaterals. Glucocorticoids have distinct actions of the dentate granule cells (inhibiting progenitor proliferation), and the pyramidal cells of CA3 (encouraging dendritic atrophy) and CA1 (accentuating neurotoxic actions). The output from the hippocampus comes mainly from CA1.

Chronischer Stress & Gehirn: Atrophien & Hypertrophien



im **Hippocampus**:

- Dendritenatrophie
- reduzierte Neurogenese

im **PFC**:

- Dendritenatrophie
- reduzierte Neurogenese

in der **Amygdala**:

- Dendritenhyperthrophie

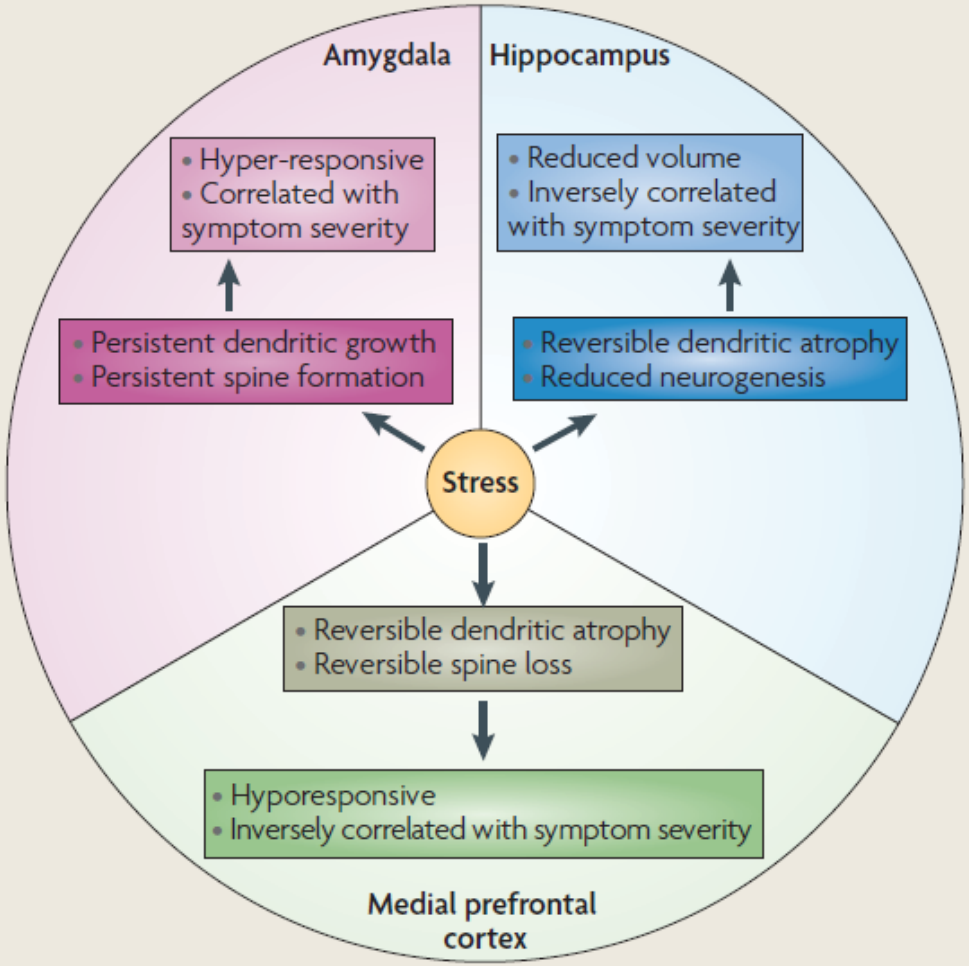
→ Gleichgewicht zwischen diesen Strukturen verschiebt sich unter chronischem Stress

Chronischer Stress & Gehirn: regionsspezifische Effekte (Roozendaal et al., 2009)

RUB

Box 1 | **Structural changes in interconnected brain regions**

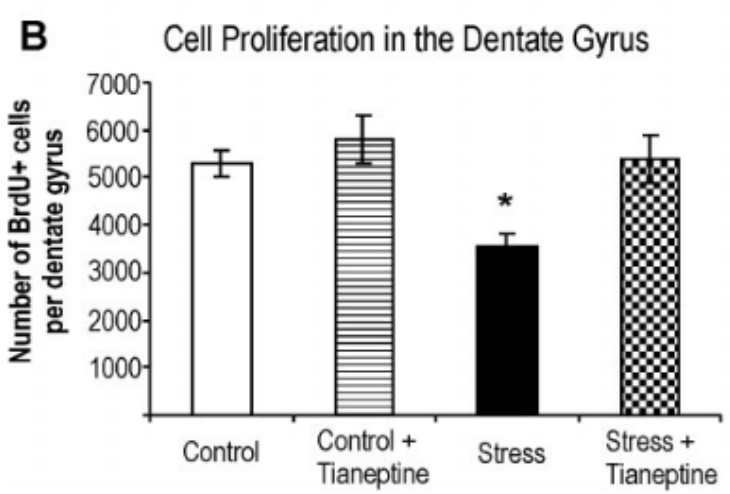
Repeated stress of the type that causes remodelling of neurons and connections in the amygdala produces concurrent neuronal remodelling in the prefrontal cortex and hippocampus (see the figure), two structures that regulate the activity of the hypothalamus-pituitary-adrenal axis[117]. These changes include shrinkage of dendrites and a reduction of spine density in medial prefrontal cortex neurons and, in the hippocampus, shrinkage of dendrites in CA3 pyramidal neurons and dentate gyrus granule neurons. Chronic stress also decreases neurogenesis and neuron number in the dentate gyrus. However, dendritic branching in the orbitofrontal cortex increases as a result of chronic stress. For the most part, these stress-induced changes in the hippocampus and medial prefrontal cortex are reversible over time, at least in the animal models that have been investigated so far. As these brain regions are interconnected, it is likely that the structural remodelling in one region will influence the functions of other brain regions.



Chronischer Stress & Gehirn: Hippocampus & Antidepressiva

Stress-induced changes in cerebral metabolites, hippocampal volume, and cell proliferation are prevented by antidepressant treatment with tianeptine

Boldizsár Czéh*, Thomas Michaelis†, Takashi Watanabe†, Jens Frahm†, Gabriel de Biurrun*, Marja van Kampen*, Alessandro Bartolomucci*, and Eberhard Fuchs*‡



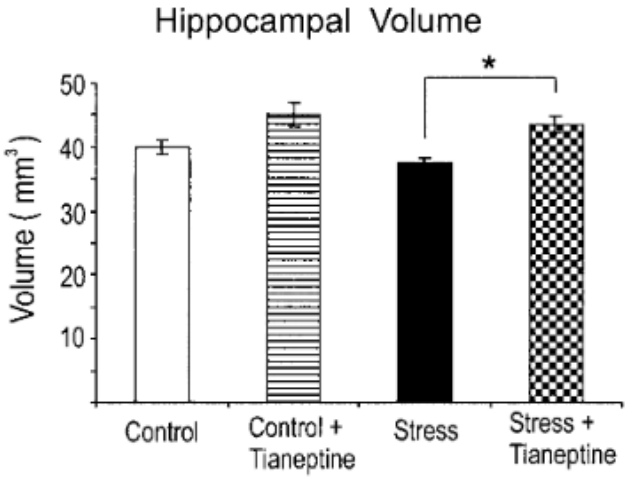


Fig. 3. Postmortem volumetry of the hippocampus. Chronic psychosocial stress resulted in a decrease (7%) of the hippocampal volume compared with unstressed controls ($P = 0.40$). This decrease was prevented by tianeptine treatment (Stress + Tianeptine vs. Stress; *, $P < 0.05$).

→ Schutz des Hippocampus durch Antidepressiva vor den Folgen von chronischem Stress

Chronischer Stress & Gehirn: Dendritenatrophie im PFC

Brief Communication

Reversibility of apical dendritic retraction in the rat medial prefrontal cortex following repeated stress

Jason J. Radley[a,c,*], Anne B. Rocher[a], William G.M. Janssen[a], Patrick R. Hof[a,c], Bruce S. McEwen[b,c], John H. Morrison[a,c]

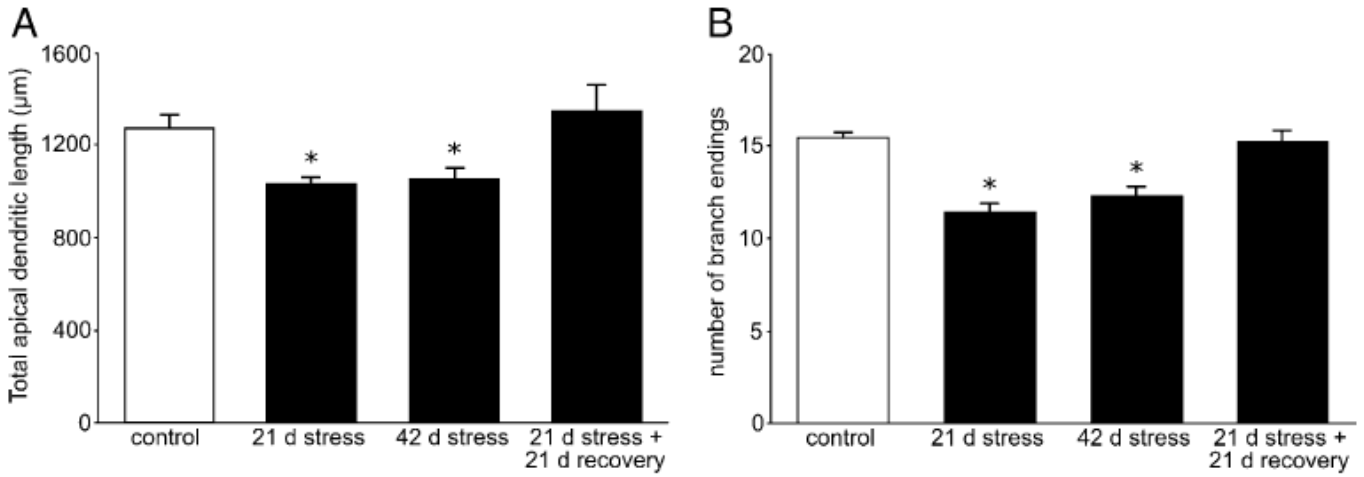
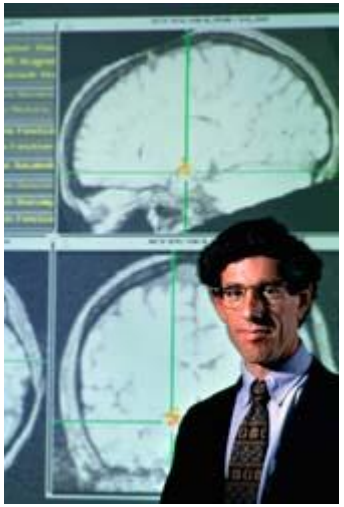


Fig. 3. After 3 and 6 weeks of restraint stress, the total length (A) and branch ending number (B) for apical dendrites of medial PFC pyramidal neurons were significantly reduced compared to unstressed control and 3-week stress/3-week recovery groups. Asterisks indicate significant differences (see text for *P* values) between stress vs. control and stress vs. stress/recovery.

- reversible Dendritenatrophie im PFC (nach 3-wöchiger Erholungsphase)

Chronischer Stress & Gehirn: Neuroplastizität (Davidson & McEwen, 2012)

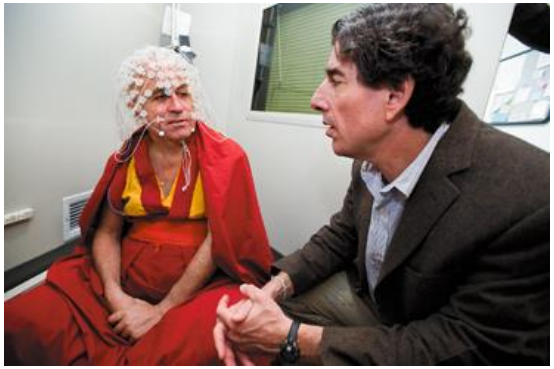


nature neuroscience

Social influences on neuroplasticity: stress and interventions to promote well-being

Richard J Davidson[1] & Bruce S McEwen[2]

Experiential factors shape the neural circuits underlying social and emotional behavior from the prenatal period to the end of life. These factors include both incidental influences, such as early adversity, and intentional influences that can be produced in humans through specific interventions designed to promote prosocial behavior and well-being. Here we review important extant evidence in animal models and humans. Although the precise mechanisms of plasticity are still not fully understood, moderate to severe stress appears to increase the growth of several sectors of the amygdala, whereas the effects in the hippocampus and prefrontal cortex tend to be opposite. Structural and functional changes in the brain have been observed with cognitive therapy and certain forms of meditation and lead to the suggestion that well-being and other prosocial characteristics might be enhanced through training.



Chronischer Stress & Gehirn: Neuroplastizität (Davidson & McEwen, 2012)

https://www.richardjdavidson.com/

Chronischer Stress & Gehirn: Neuroplastizität (Davidson & McEwen, 2012)

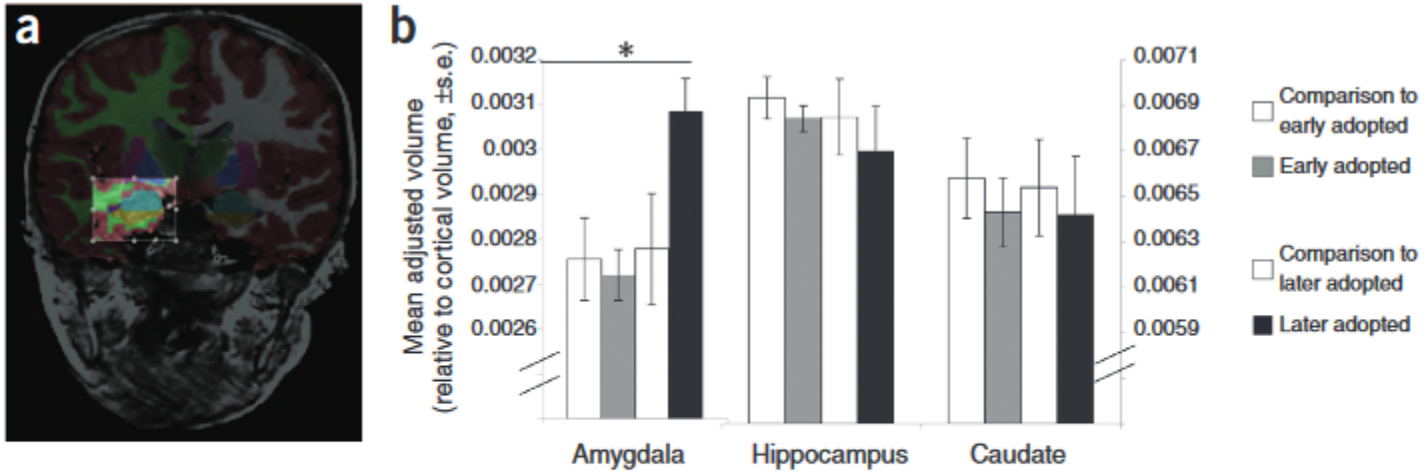


Figure 3 Anatomically segmented amygdala volumes are larger in later-adopted post-institutionalized children. (**a**) Anatomical segmentation of the amygdala. (**b**) Later-adopted post-institutionalized children show larger amygdala volume compared with both early adopted children and with typically developing controls. No differences among groups were found in hippocampus or caudate[71]. Asterisk indicates that the later adopted group exhibited significantly larger amygdala volume compared with each of the comparison groups.

- vergrößerte Amygdala bei Kindern mit langem Waisenhausaufenthalt

Chronischer Stress & Gehirn: Neuroplastizität

Larger amygdala but no change in hippocampal volume in 10-year-old children exposed to maternal depressive symptomatology since birth

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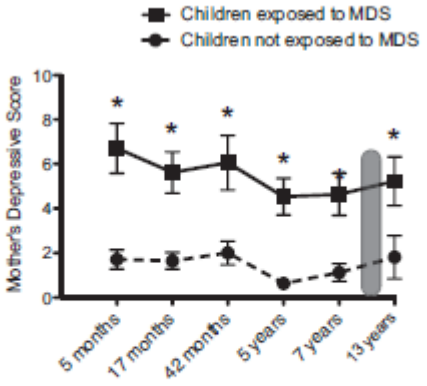

Fig. 1. Scores of mothers on the CES-D at child's age 5, 17, 30, 42, 60 (5 y), 84 (7 y), and 156 mo (13 y). Mothers of children exposed to MDS presented significantly higher MDS at all timepoints compared with mothers of children not exposed to MDS (*$P < 0.001$). The gray zone represents time of scanning at 120 mo (10 y). Error bars represent SEMs.

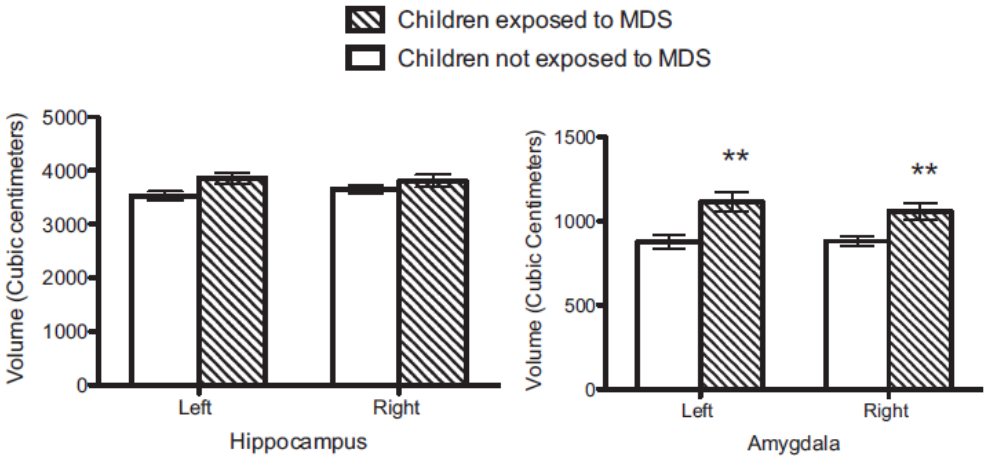

Fig. 2. Left and right hippocampal (*Left*) and amygdala (*Right*) volumes (cubic centimeters) in children exposed to MDS since birth and children not exposed to MDS since birth. Error bars represent SEMs. **$P < 0.01$ for left and right amygdala volume.

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Stress reduction correlates with structural changes in the amygdala

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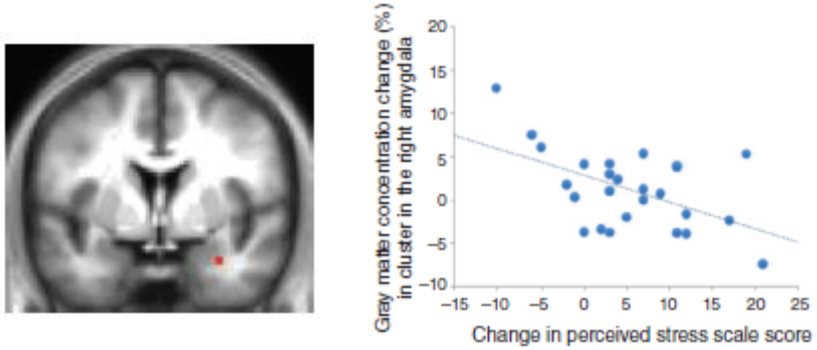

Figure 4 Change in gray matter volume in the right basolateral amygdala from pre to post 8 weeks of MBSR was associated with decreases in perceived stress over this same time period. Individuals undergoing MBSR who showed the largest decreases in perceived stress also showed the largest decreases in basolateral amygdala gray matter volume[11].

Several previous cross-sectional studies have investigated the impact of mindfulness meditation on brain morphology by comparing groups of experienced mindfulness meditators to nonmeditators (Lazar *et al.*, 2005; Pagnoni and Cekic, 2007; Hölzel *et al.*, 2008; Luders *et al.*, 2009). These studies identified several regions of altered brain morphology, but none within the amygdalae. However, none of these studies assessed the participants' perceived stress levels. Again, these data highlight the limitations of the cross-sectional study design. The unique hypothesis-driven, focused analysis employed in the present study revealed a novel link between changes in amygdaloid gray matter density and decreases in self-reported stress following stress-reduction training, marking a significant advance in our understanding of the association between both. Whereas previous studies have demonstrated that gray matter modifications can result from the acquisition of abstract information (Draganski *et al.*, 2006), motor skills (Draganski *et al.*, 2004) and language skills (Mechelli *et al.*, 2004), this is the first study to demonstrate neuroplastic changes associated with changes in a measure of a psychological state.

Interventionsstudie: ReSource

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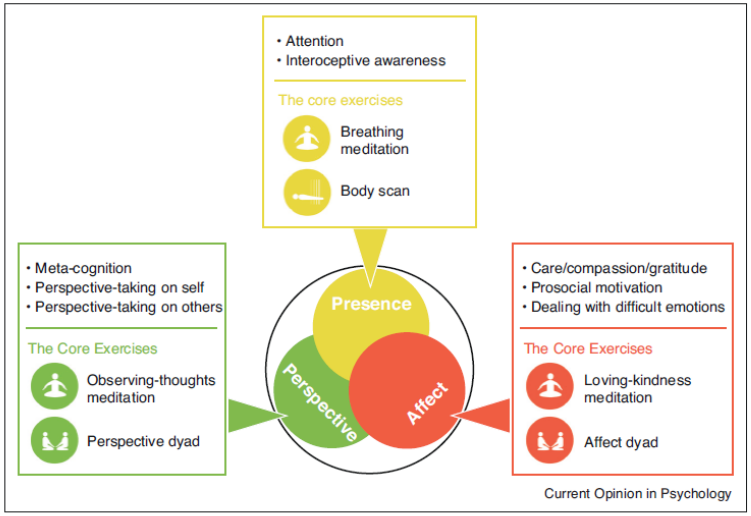
Current Opinion in Psychology

It matters what you practice: differential training effects on subjective experience, behavior, brain and body in the *ReSource Project*

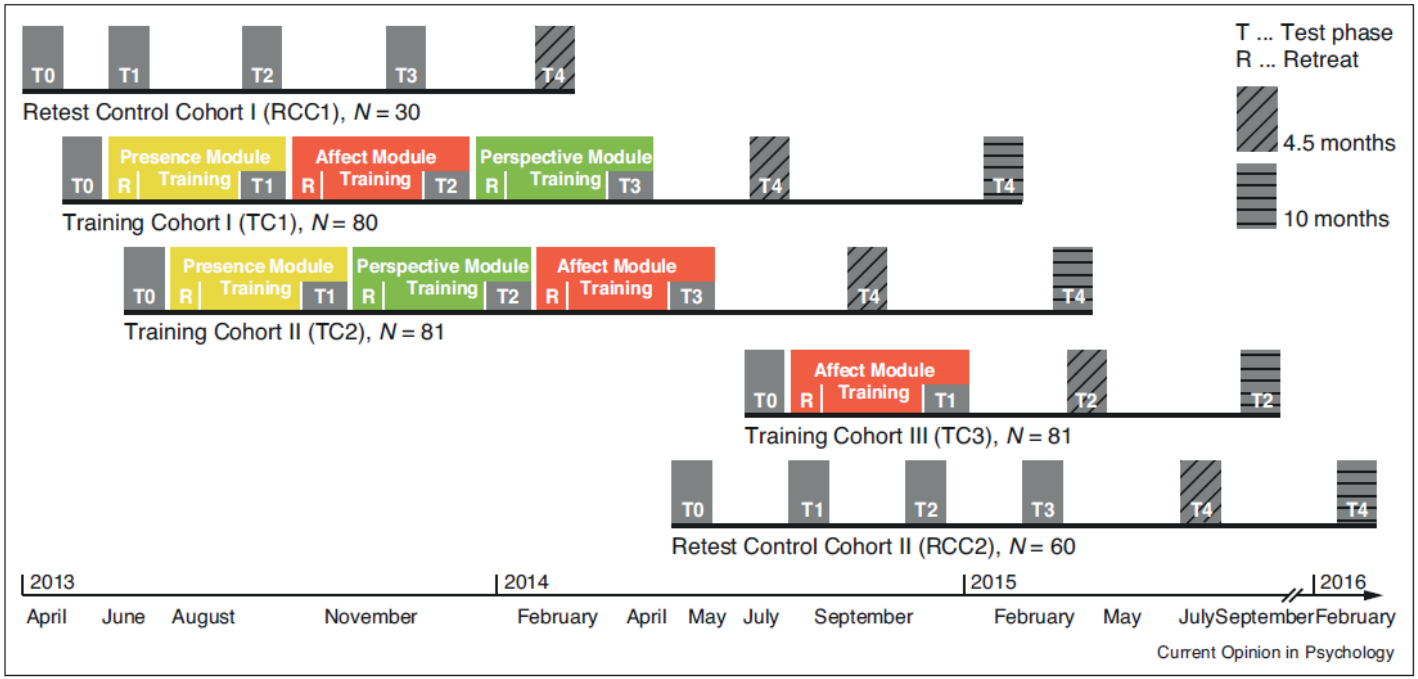
Tania Singer[1,2] and Veronika Engert[1,3]



Core processes and practices of the *ReSource* training. The processes targeted in the Presence Module are attention and interoceptive body awareness, which are trained daily through the two classic meditation-based practices Breathing Meditation and Body Scan. The Affect Module targets social emotions such as compassion, loving kindness and gratitude. Additionally, it aims to enhance prosocial motivation and dealing with difficult emotions. The two daily practices trained in the Affect Module are Loving-kindness Meditation and Affect Dyad. In the Perspective Module, metacognition and perspective-taking on self and others are trained through Observing-thoughts Meditation and Perspective Dyad.

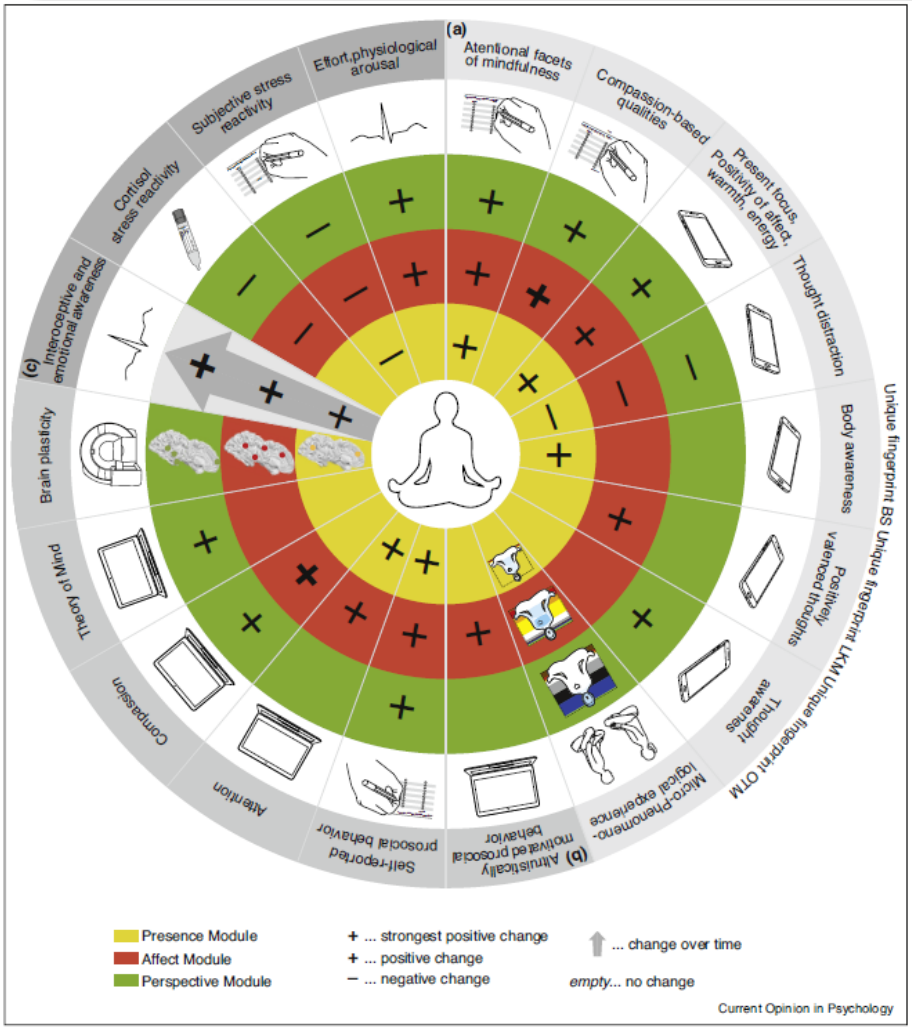
Interventionsstudie: ReSource

Figure 2



Design and timeline of the *Resource Project*. Two training cohorts (TC1 and TC2) started their training with the mindful attention-based Presence Module. They then underwent the social Affect and Perspective Modules in different orders, thereby acting as mutual active control groups. To isolate the specific effects of the Presence training, a third training cohort (TC3) underwent a 3-month Affect Module only. The total training time for TC1 and TC2 was 39 weeks (13 weeks per module). TC3 only trained for 13 weeks [for more detailed information see Ref. 7, chapter 4].

Interventionsstudie: ReSource

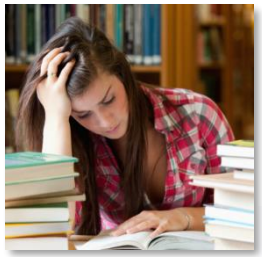


https://www.resource-project.org/

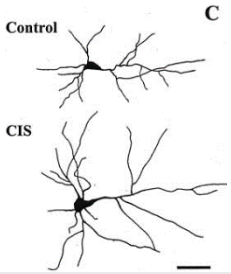
Schematic representation of *ReSource* training effects and utilized methods of data assessment (for more details on the complex result patterns, please check original articles). (**a**) Subjective experience. On the trait level, Hildebrandt *et al.* [13] assessed attentional facets of mindfulness and compassion-based qualities once per module using self-report questionnaires [18–23]. On a more fine-grained level, state ratings of subjective experience as measured by Kok and Singer [15] were recorded immediately after a daily meditation practice using a custom-designed app (BS: Body Scan; LKM: Loving-kindness Meditation; OTM: Observing-thoughts Meditation). In the study by Pzyrembel and Singer [16], micro-phenomenological experience was measured once per module in an extensive 1-hour interview. The depicted results focus on differential color sensations and activated body areas during the core meditation practices. (**b**) Behavior and structural brain plasticity. Böckler *et al.* [28•] assessed three facets of prosocial behavior (altruistically motivated, self-reported and norm-based prosocial behavior; 29,30) using a battery of measures including game theoretical paradigms, interactive computer tasks, hypothetical distribution tasks and multiple trait questionnaires. Norm-based behavior is not depicted here because it showed no change after mental training. In Trautwein *et al.* [24], the cued flanker task [45,46] was applied to assess attentional performance. For measures of compassion and Theory of Mind, participants conducted the EmpaToM video task [32,33]. In the study by Valk *et al.* [34•], brain plasticity was measured in terms of cortical thickness assessed with magnetic resonance imaging. (**c**) Peripheral physiology. Bornemann *et al.* [40] measured interoceptive awareness in terms of heart beat perception accuracy. The Toronto

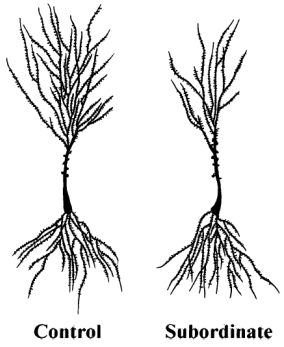
Zusammenfassung

- **akuter Stress & Gedächtnis**:
  - verbesserte Konsolidierung
  - verschlechterter Abruf



- **chronischer Stress & Gehirn**:
  - Dendritenatrophie in Hippocampus & PFC
  - Dendritenhypertrophie in Amygdala
  - Teilw. reversibel → Neuroplastizität





Vorläufige Terminübersicht

20.10.25	Übersicht und Einführung
27.10.25	Stress und das SNS: Walter Cannon (Dr. Katja Langer)
03.11.25	Stress und die HHNA: Hans Selye
10.11.25	Stress und die HHNA: Munck und Sapolsky Die kognitive Wende: Lazarus
17.11.25	Stress und Gesundheit: McEwen und die allostatische Belastung Stress im Arbeitsleben: Siegrist und die Effort Reward Imbalance
24.11.25	Burnout (Dipl. Psych. Natalie Freund)
01.12.25	Soziale Evaluation als bedeutsamer Stressor: Dickerson & Kemeny (Dr. Katja Langer)
08.12.25	Soziale Unterstützung als Stresspuffer/Oxytozin
15.12.25	Stress und Gehirn: akute und chronische Effekte
12.01.26	Pränataler Stress und seine Folgen
(Aufzeichnung)	Frühkindlicher Stress und seine Folgen (Prof. Robert Kumsta)
19.01.26	Posttraumatische Belastungsstörung