

In: Lifespan Development and the Brain: The perspective of Biocultural Co-Constructivism, Eds : P. Baltes, P. Reuter-Lorenz and F. Rösler. Cambridge University Press.

The Influence of Organized Violence and Terror on Brain and Mind – a Co-Constructive Perspective

by Thomas Elbert, Brigitte Rockstroh, Iris Kolassa, Maggie Schauer, Frank Neuner
University of Konstanz, Germany and NGO vivo international (www.vivo.org)

Address of the authors:

Thomas.Elbert@Uni-Konstanz.de

Brigitte.Rockstroh@Uni-Konstanz.de

Iris.Kolassa@Uni-Konstanz.de

Maggie.Schauer@vivo.org

Frank.Neuner@Uni-Konstanz.de

Acknowledgement: The research was supported by the Deutsche Forschungsgemeinschaft and the European Refugee Fund (EFF)

„Genetic interventions make us better animals. Humans, we become, however, because of the ways that culture and our individual constructions exploit the brain and make it our servant.“ (Baltes & Singer, 2001).

INTRODUCTION

In this day and age human beings are raised and live in a complex socio-cultural environment with increased demands for the brain, the body, and the social structures to adapt. More information at increasingly complex levels has to be processed than ever before at an ever increasing velocity and over an extended life-span. This places a high pressure on the individual and the society to continuously adjust to new environmental conditions resulting in a stream of continuous *microstressors*. At the same time, modern societies are becoming increasingly aware of the effects of *macrostressors* including traumatic stress, which, although seemingly transient, may be changing the brain's processing modes, resulting in characteristic behavioral, physiological, and psychological (mal)adaptations to environmental conditions and – when a whole community is affected – in a change in the local culture. This in turn will impinge on all individuals in the community, even those not affected by the traumatic experience in the first place, as human behavior represents the co-constructive expression of biological-genetic and socio-cultural conditions. While recent neuroscientific advances have substantially improved our understanding of neuroplasticity, i.e., the brain's extraordinary ability to change its structure and function in response to experience, less effort has been devoted to understanding mechanisms in relation to affect and distress and little is known how changes on the level of the individual interact with those on a community level. Social stress and organized violence serve as good models for studying this interaction of adaptive alterations in individual and societal minds. We currently witness a qualitative change in the way wars are waged and organized violence is exerted; in other words, a transformation in the “culture of violence” cannot be overlooked. Moreover, scientific methods are available to study how traumatic stressors change individuals and communities, so that we can expect increasing knowledge about how social stressors and related learning conditions shape

the structure and function of the brain, the “societal mind” including individual behavior and interactions on the community level.

Brain and body constitute the joint product or co-construction of two interactive systems of impact; the internal genetic-biological and the external socio-cultural systems (Baltes, 1999). The social environment into which malleable individuals are born, together with their response to this environment – the way, in which individuals live their lives – lead to the cultural determination of gene expression. In turn, individuals shape their socio-cultural environment by imposing structure and function resulting from a history of genetic expression. Stressors exert a powerful influence on the brain, at the same time modifying brain structure, brain function, neuropsychological performance, and peripheral physiological responses. Extreme or continuous stress may drive the individual into an increasingly maladaptive range with the potential for mental disorders. Cultural settings can support only a limited fraction of such individuals before these become a driving force in cultural mal-adaptations.

NEURAL ADAPTATION AND PLASTICITY: EXPERIENCE SHAPES THE BRAIN

The brain is continuously modified by experience. The study of sensory representations in the cortex has provided an excellent model for studying how the brain’s representations of the periphery are dynamically modified (Elbert & Heim, 2001; Elbert & Rockstroh, 2004). Cortical representations mirror the spatial arrangement of the corresponding peripheral receptors in the form of cortical maps. Although genetically encoded programs control the connections of these maps from the periphery to the cortical destination, their organization ultimately depends on the efficacy of the synapses connecting the nerve cells within the network, which is affected by external input. For instance, two receptors of the same fingertip are more frequently activated in synchrony than two receptors in different digits. According to Hebb’s learning model, synchronous stimulation should lead to connections between the representations of the same fingertip but to a separation from those of the other digits: representational zones are shaped by the temporal pattern of such coincident experience. An alteration in behaviorally relevant afferent input will trigger a reorganization of the map. The representation of a fingertip, for instance, can be enlarged;

representations of adjacent fingers can invade its territory; or the representation of two fingers can get fused. Using magnetic source imaging, we have demonstrated that skilled string instrument players – a category for which both cultural and psychological preconditions are necessary – have larger representational zones of their left hand in their cerebral cortex compared to the brains of people who do not engage in such extensive practice (Elbert et al., 1995). Using the same culturally determined quasi-experimental setup, structural MRI reveals that the change in function is intertwined with structural alterations (the depth of the left-hemispheric central sulcus) that extend into the macroscopic range (Schlaug et al., 1995; Amunts et al., 1997). Moreover, the musician’s brain not only exemplifies adaptation (or in some cases maladaptation; Elbert et al., 1998) to somatosensory requirements but differs from “normal” brains on many levels (Münte et al., 2002; Christian & Schlaug, 2003). Use-dependent reorganization also holds for other sensory modalities than the somatosensory cortex: whenever musicians hear their own instrument, larger responses are elicited in the auditory cortex than when they hear tones from other instruments (Pantev et al., 1998). Reorganization varies with perceptual correlates of superior performance – an adaptive advantage of cortical plasticity.

Adaptive cortical plasticity can also be observed in disabled persons – as in blind individuals who are forced by the nature of their disability to rely on non-visual modalities, including hearing, for information about their external environment. Sensory input via non-visual avenues thus gains greater behavioral relevance and becomes a focus of greater attention to enable effective interaction with the world. For instance, when attention is directed to peripheral auditory space, localization of sounds is better in blind than in sighted people (Lessard et al., 1998; Muchnik et al., 1991; Röder et al., 1999). Individuals who lost their sight at an early age may outperform sighted persons in non-visual tasks, including speech perception (Muchnik et al., 1991; Niemeyer & Starlinger, 1981; Röder et al., 2003), verbal memory (Hull & Mason, 1995; Röder et al., 2001), and musical abilities (Gougoux et al., 2004; Hamilton et al., 2004). Behaviorally relevant stimulation over extended periods has been found to produce a substantial enlargement in the representational zones of the involved portions of the tonotopic system in animals (Recanzone et al., 1992) and humans (Elbert et al., 2002). In addition, there is cross-modal plasticity in the blind, such that auditory (Ahlo et al., 1993; Gougoux et al., 2005; Kujala et al., 1992, 1995, 1997) and tactile (Kujala et al., 1995; Röder et al., 1996; Rösler et al., 1993;

Uhl et al., 1993) stimuli come to be processed in visual cortex. Obviously, environmental requests and the individual experiences can dramatically remodel the brain's functional organization.

During “critical periods” of development, sensory stimulation without explicit behavioral significance is sufficient to alter the organization of the sensory cortex (Bao et al., 2003; Wiesel & Hubel, 1965; Zhang et al., 2001). In contrast, functional reorganization in the adult cortex seems to be driven mainly by stimuli related to reinforcement, i.e., it requires a behaviorally relevant context (e.g., Diamond & Weinberger, 1989; Recanzone et al., 1992, 1993; review Elbert & Rockstroh, 2004). The effects of reinforcement are mediated by cholinergic (Kilgard & Merzenich, 1998) and dopaminergic pathways (Bao et al., 2001) which by themselves are subject to plastic alterations, as seen in psychosis. While the model of the blind demonstrates that similar mechanisms also act beyond the representational cortex, additional mechanisms may come into play in associative or polymodal areas. Already at the level of the primary sensory cortex, context and top-down modulation driven by attention and motivation affect reorganization (Braun et al., 2000, 2002).

On the other hand, stressful life experiences during “critical periods” may affect brain organization in harmful ways. As childhood and adolescence are determined as critical periods of cognitive and emotional development (Paus, 2005; Steinberg, 2005), they are particularly vulnerable phases for the development of the stress system (Charmandari et al., 2003). Even a single traumatic experience can initiate a cascade of dynamic brain processes which may result in enhanced vulnerability to subsequent stressors (Schauer et al., 2003; Neuner et al., 2004a) or even in a break-down of normal functioning as seen in the pathologies of the trauma spectrum. Consequently, the question arises to what extent brain and mind are affected in individuals living or even growing up in a culture of violence. Before we will examine the latter, we turn to the question whether violent and traumatic experiences by altering the individual brain and mind can induce a spiral of violence in which an increasing number of individuals influenced by traumatic experiences themselves commit crimes in the community.

STRESSED BRAINS – TRAUMATIZED MINDS

Wartime strategies are increasingly characterized by mutual hate of ethnicities. Forcible displacement of civilians is used by both guerrilla and anti-guerrilla forces, in an attempt to unite one's own group through crimes against humanity. Wars are accompanied by systematic killings and ethnic cleansings, whole regions are left uninhabitable for the local people since towns were devastated, infrastructure ruined, and landmines make much of the land inaccessible. In an attempt to understand the consequences of these atrocities on brain and mind (Elbert & Schauer, 2002; Neuner, 2003), we have studied refugees in Germany and war victims in crises regions such as the Balkans (Neuner et al., 2002), the West Nile (Neuner et al., 2004a,b; Karunakara et al., 2004), Rwanda (Schaal & Elbert, 2005; Onyut et al., 2004), and Somalia (Odenwald et al., 2005). In all these regions, we conducted diagnostic interviews and found high prevalence rates of posttraumatic stress disorder ranging from 19 to 51%. An amazing finding was the ability of displaced persons from remote areas in Southern Sudan, who previously had almost no contact with the outside world and who were illiterate, to describe the classic symptoms of severely traumatized individuals as if taken from a psychiatric textbook. The core symptoms of Posttraumatic Stress Disorder (PTSD) are: (1) re-experiencing symptoms that manifest at night in the form of nightmares and in the waking state as flashbacks and intrusive recollections which are so intense that the victim actually believes to be back amidst the atrocities, (2) an exaggerated startle response and a persistent hyperarousal, difficulties in calming down or falling sleep; all these symptoms describe a readiness for fight or flight rather than a permanently enhanced autonomic activation, and (3) an active avoidance of places or thoughts associated with traumatic experiences and/or passive avoidance symptoms, i.e., numbing emotional responsiveness as a way to cope with unbearable feelings. In severe cases this may include dissociative symptoms, e.g., feelings of detachment or estrangement from the external world (derealization) and of oneself (depersonalization), or even persecutory delusions.

PTSD symptomatology can be understood as a consequence of plastic changes in memory through stressful, traumatic experiences. Life experiences are stored in autobiographical memory. The autobiographical context memory has been called “cold memory” (Metcalf & Jacobs, 1996). It contains knowledge about life-time periods and specific

events. The sensory-perceptual representations of a traumatic event have been called “hot” or non-declarative (implicit) memory. It comprises emotional and sensory memories of all modalities. Cold memories (e.g., on March 24 at 3:30 I was living on my farm in Djakovica, we had three cows) are usually connected with hot sensory memories (e.g., black-masked, dark night, shooting, burning smell) as well as with cognitive (e.g., I can’t do anything), emotional (e.g., fear, sadness), and physiological elements (e.g., heart racing, fast breathing, sweating).

In individuals who are not affected by trauma or fear, hot memories are linked with autobiographic, declarative memories. However, in traumatized persons, sensory and emotional memories are activated by environmental stimuli without being related to autobiographic, declarative items (i.e., dates and places of autobiographical occurrences) – those autonomous hot memories form a fear network. An example of such a network is outlined in Figure 1. The activation of a single memory item (e.g., seeing a man in a uniform or feeling ones heartbeat) will cause the whole network to be activated. According to Hebbian learning, this will not only strengthen the interconnections between existing network units, but will also lead to an inclusion of additional network element, namely of those that are synchronously activated. Such an inclusion of additional nodes will be strongest during subsequent traumatic experiences or experiences with a strong emotional component that co-activate motivational and reward systems which enhance the brain’s plasticity. As a consequence, the number of non-declarative (hot) elements in the fear network and their interconnectivity will increase. At the same time, co-activation of declarative autobiographical memories becomes less likely since with an increasing number of experiences, the network contains more and more conflicting information: a person will not be able to retrieve all contexts in which the fear network was previously activated. As a result, hot and cold memory will separate, and only few connections of hot memory contents with declarative, autobiographic memory will survive, leading to a fragmentation of the latter. The described model assumes the fear network to be an example of plastic changes in the human brain in response to traumatic life events. The validation of the proposed model provides a challenge for future neuroscientific research.

Memory is the ability to recall events from the past. However, it is a common mistake to restrict the function of memory to recollections of the past. What seems more

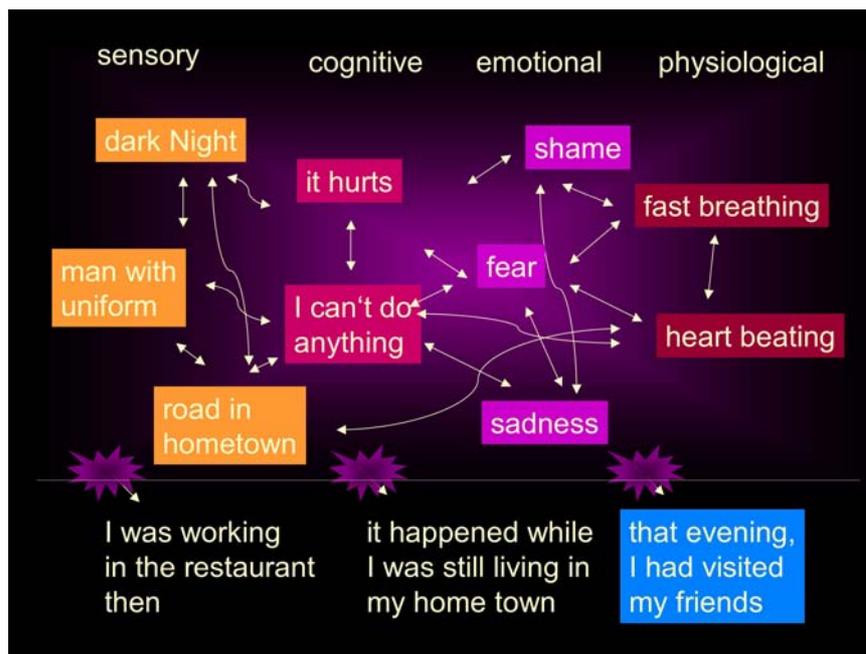


Figure 1: Example of a traumatic memory structure: the hot (non-declarative) memory builds a fear network. With each additional traumatic experience this fear network gets more and more extended, while the connections to the specific cold autobiographic events come more and more loose or even get lost.

likely is that the ability to envisage future scenarios was a driving force in the evolution of memory, and of episodic memory in particular. Hebbian types of memory fit this explanation of memory well: every time a cell assembly is activated for "read-out", the content of the respective memory will not only be read but also modified, i.e., neurons of an activated cell assembly are not only activated, but will also modify their connections with each other. This activation-modification takes place both through imagination and present experiences. Since each time a memory is activated, it is modified, a fear network, although originally formed by traumatic experience, may become connected to present conditions, e.g. when the survivor is forced to live under unsafe conditions.

How can we detect signs of a fear network? One would expect that stimuli specifically related to the individual traumatic experience are necessary to activate the fear network. However, contrary to expectations, it seems that emotionally arousing stimuli, not necessarily related to the traumatic event, suffice to activate the whole fear network. Junghöfer et al. developed a rapid serial visual presentation (RSVP) paradigm whereby

stimuli from the International Affective Picture System (IAPS; Lang et al., 1999) are presented in fast succession (3 or 5 Hz). We suggest that each emotional stimulus, even when presented for some 300ms only activates one or more elements in a large fear network. Using this technique, Junghöfer et al. (2003) demonstrated that indeed, many elements or nodes of the network will be activated and the whole network will ignite. This stimulation proved as powerful to evoke affective processing and even provoked flash-backs in severely traumatized survivors of organized violence including torture. (It should be noted that this was the case in individuals for whom flash-backs were so common that the one contingent on the RSVP didn't add to their suffering, as they reported). In both, controls and traumatized individuals, the affective material activated the visual cortex and associated areas. However, only in traumatized survivors suffering from PTSD, the pre- and orbitofrontal areas and the cingulate gyrus were also activated (Junghöfer et al., 2003; the group difference is displayed in Figure 2). Thus, the data suggest that torture, like any other massive experience, dramatically alters the functional organization of the brain: an enlarged fear network is activated by aversive material. Obviously, the (medial) prefrontal cortex (including the anterior cingulate) lost its ability to regulate the hyperresponsive fear structures, i.e., presumably the interplay between amygdala and frontal cortex is disturbed.

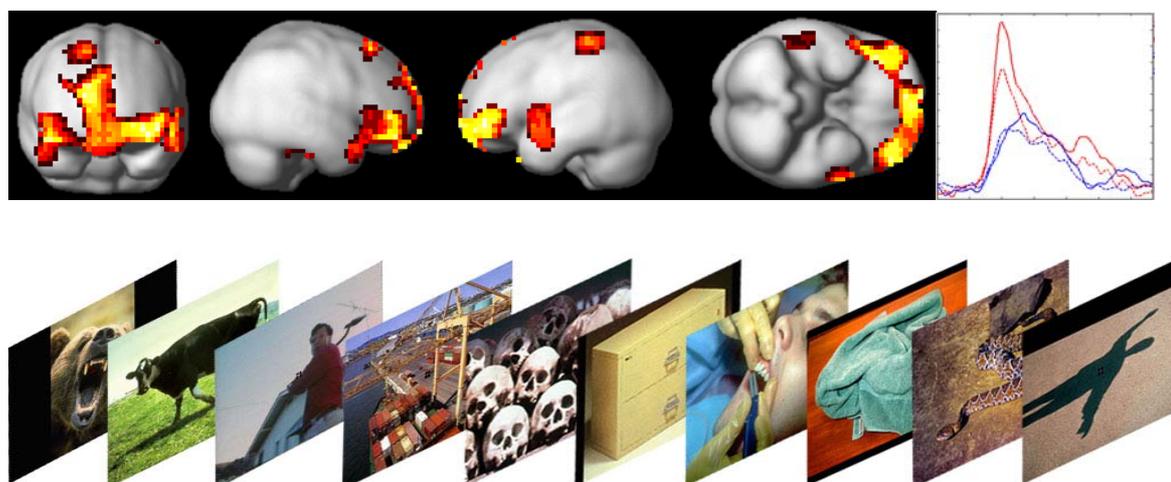


Figure 2: Event-related magnetic fields were recorded from 13 torture victims with a current diagnosis of PTSD and a group of 13 controls, matched for age, gender as well as ethnic back-

ground. Aversive and neutral IAPS pictures (2x100 per affective condition) were presented for 333ms each in an alternating fashion without ISI. The global activation in the time interval from 0 to 300ms post stimulus onset is presented in the inset in the upper right. Red lines represent PTSD patients, blue lines controls. Responses to aversive stimuli are marked by solid lines, responses to neutral ones by dashed lines. Inverse source analyses (L2-Minimum-Norm) were performed, and group differences were tested for an early 60-110ms interval based on Statistical Parametric Mapping (SPM). Sources were localized bilaterally in occipital and occipito-parietal areas with a right hemispheric dominance. Frontal difference activations were distinctly stronger in PTSD patients in both hemispheres (with right hemisphere dominance) as illustrated in the top row (data from Junghöfer et al., 2003)

Severely traumatized persons like torture victims often describe dissociative experiences during or immediately after trauma exposure (peritraumatic dissociation), but also later on (posttraumatic dissociation). These episodes are characterized by feelings of detachment from the self (depersonalization), an unreal or distorted perception of internal and external reality (derealization), and a reduced awareness of surroundings. During such episodes, they experience time in a nonlinear way. Persons who report more peritraumatic dissociation are at greater risk for developing PTSD (Fullerton et al., 2000; Marmar et al, 1998; Koopman et al, 1994; Shalev et al., 1996). In addition, they have the most difficulties to later assess information about traumatic experiences in a systematic manner (Michelson & Ray, 1996; McNally, 2003).

Using magnetic source imaging procedures we mapped (Schauer et al., 2005b) abnormal slow wave activity in 23 survivors of severe torture with a current diagnosis of PTSD, who had experienced multiple forms of psychological traumata. The number of dissociative experiences was significantly and positively related to the density of abnormal slow wave generators in the left ventral region of the anterior cortical structures and to the left hemisphere as a whole. The inverse relationship was found for the right hemisphere as a whole and for the right anterior superior areas (correlations range from .4 to .65). Statistically partialling out the level of posttraumatic stress disorder did not influence these relationships suggesting that the level of dissociation contributes a separate component over and above PTSD symptoms to abnormal brain activity. This is theoretically consistent with DSM-IV not including dissociation as a PTSD criterion. Further-

more, the patient group showed significantly more abnormal slow waves in the left ventral region than a culturally matched control group without torture experience.

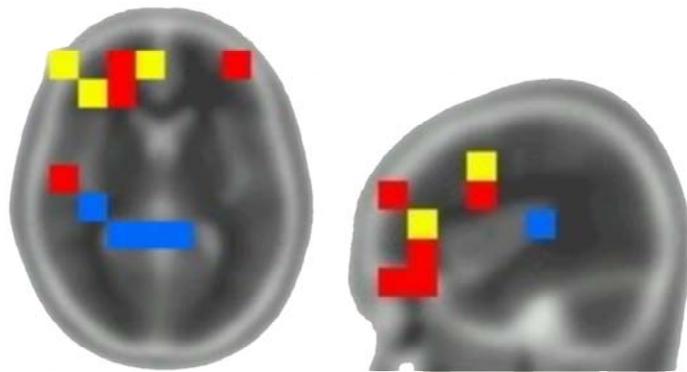


Figure 3: An example of abnormal neural generators in a PTSD patient scoring high in dissociative symptoms: red voxels depict deviations of more than 2 standard deviations (SD) from the norm (healthy controls) and yellow voxels deviations of more than 3 SD. Blue indicates activity below normal. The patient displays a high density of focally generated slow waves in the left frontal region and the region of the anterior cingulate. During the recording session, the patient was fully awake and had less than average global power in the delta band (Schauer et al, 2005b).

Left frontal areas subserve language and executive function. However, more recently neuroimaging studies showed the left ventral prefrontal cortex also to be involved in both verbal memory encoding and retrieval (Iidaka et al., 2000). This might explain why dissociative individuals lack conscious, verbal access to certain previous traumatic experiences. It is a common experience in clinical practice that patients with PTSD have difficulties in verbalizing their traumatic experiences. The quality of emotional memories during re-experiencing symptoms is more emotional and sensoric in nature, while literally being out of touch verbally with patients' feelings. A disruption of these frontal networks would explain why individuals experiencing intrusions and dissociative episodes are unable actively retrieve and verbalize previous traumatic experiences. Our current model suggests that torture initially prompts active or passive avoidance strategies in an attempt to reduce overwhelming fear, that finally result in a permanent disruption of left frontal networks. The neural mechanisms of this disconnection may in part be explained by long-term depression (LTD) as indicated above, but also by anti-Hebbian learning (i.e., the strengthening of inhibitory synapses). The functional disconnection of affective from lan-

guage processing areas as a consequence of trauma-induced plastic changes in the brain's emotion and memory systems remains to be further validated in future studies.

Central-peripheral circuits triggered by specific environmental cues that activate the fight-flight-freeze defense cascade strongly affect the dynamic storage of various elements of memory. The body's stress response is regulated by three systems (overview Teicher et al., 2002; McEwen et al., 2002; McEwen, 2004; Elbert & Rockstroh, 2003; Steckler et al. 2005): First, the hippocampus and the hypothalamic-pituitary-adrenal (HPA) axis play a major role in the defense cascade and are involved in the feedback regulation of cortisol excretion. Second, the amygdala, the locus coeruleus, the adrenal gland, and the sympathetic nervous system are crucial in the stress-induced mobilization for fight or flight; they are involved in sharpening awareness in alarm situations and directing blood flow toward the brain and major muscles and away from surface of skin in hands and feet as well as away from digestive and reproductive organs. A third, less well explored axis involves the vasopressin-oxytocin peptides (Heinrichs et al., 2001, 2003). When functioning properly, these systems secure survival in alarm situations. They also play a role in the stress-protective effects of positive social interactions. Dysregulations in these systems may be associated with clinical disorders. This happens, for instance, when neural representations of fearful past experiences activate the HPA axis permanently. The excreted stress hormones ultimately make their way back to the brain, affecting both behavior and health.

Allostasis¹, the adaptation of the internal milieu to meet perceived or anticipated threats in the environment, has evolved as a survival securing response to escape acute danger. However, it may be an inappropriate response in the modern human. The same physiological responses (like the supply of additional blood and oxygen to muscles, etc.) are still activated in the face of modern stressors, which can, however, neither be attacked nor escaped from by running away. Thus, prolonged stress turns adaptive allostasis into

¹ The body, including the brain, is able to deal with dangers in a flexible and adaptive way. In contrast to *homeostasis*, i.e., the organism's ability to maintain a steady internal state, *allostasis* refers to the flexibility in the adjustment to stressors that range from Hans Selye's types of physical deprivation (cold, noise, deprivation of food, sleep, etc.) to the real or imagined fear-provoking situations that trigger an alarm response. The Greek word 'allo', meaning 'variable', is used by McEwen (2002) to emphasize the ability to choose various attack and defence mechanisms to counter negative impact.

allostatic load. Permanently warding off stress turns the adaptive physiological responses into maladaptive diseases in the form of aches and pains, loss of appetite or overeating. A chronically high allostatic load damages organs, including the brain (McEwen, 2004).

Increasing evidence suggests that the brain is affected in various ways by stressful experiences. Two prime targets for stress hormones in the brain are the hippocampus and the amygdala. It is well established that acute elevations of adrenal stress hormones (catecholamines and glucocorticoids) enhance memory consolidation of emotionally arousing, contextual (hippocampus-dependent) information in a dose-dependent manner in animals (Roozendaal et al., 2001) and humans (Cahill et al., 1994; Buchanan et al., 2001). These enhancing effects of stress hormones are mediated by the basolateral nucleus of the amygdala (Cahill et al., 1995; McGaugh, 2002).

Although the memory-supporting effects of stress hormones are certainly adaptive when lasting memories of vital information (e.g., dangerous situations) have to be established, this mechanism may become maladaptive under conditions of extreme stress: persistent and intrusive memories of the traumatic event might be formed which promote the development of PTSD. Yet, elevated glucocorticoid levels do not only enhance memory consolidation, but also impair memory retrieval (de Quervain et al., 1998, 2000; Roozendaal et al., 2003). In addition, chronic glucocorticoid excess can lead to disturbances of synaptic plasticity, atrophy of dendritic branching, and an enhanced susceptibility to other neurotoxic insults (Sapolsky, 1999). Moreover, stress-related enhanced corticotrophin releasing hormone (CRH) secretion during “sensitive periods” of brain plasticity in childhood and adolescence (Paus, 2005; Steinberg, 2005; Thompson et al., 2004) enforces hippocampal volume loss, sensitization of hippocampal glucocorticoids receptors, and altered feedback properties of the HPA axis, which in turn promote endocrine hyperresponsivity to subsequent social stress. The seminal studies by Meaney and his group provide clear evidence that perinatal stress already changes the HPA-axis, delays cognitive and emotional development, and may impair avoidance learning for the rest of the life (Meaney et al., 1988; Bock et al., 2003; Teicher et al., 2002).

Thus, stressful experiences differentially activate a variety of responses designed by evolution to counter danger. The different chemical messengers may cause deficits in hippocampus-based learning and memory, and their effects on the amygdalae and the medial prefrontal and cingulate cortex may lead to an impaired inhibition of fear responses. A fragmentation of autobiographical memory due to a separation of emotional and declarative autobiographic memory contents is further promoted by these mechanisms. Repeated exposure to traumatic or chronic stress may lead to long-term dysregulations and impaired functioning of these systems and may cause symptoms of stress-related disorders such as hyperarousal, dissociation, flashbacks, avoidance, and depression. These symptoms in turn may promote maladaptive behaviors like social withdrawal, inappropriate aggression, and self-sedation with drugs.

Such an impact on the brain may change its functionality, alter its basic rhythms, and may even cause structural abnormalities. As described in Figure 2, the responsiveness of traumatized brains to emotionally arousing material differs from that of controls. Abnormal slow wave activity may serve as a measure for (plastic) changes in brain architecture and communication. Slow brain waves, when focal and present during the waking state, signal cortical dysfunction (Rockstroh et al., 2005). In psychiatric disorders, slow waves may indicate altered neural networks even when macroscopic structural lesions are not detectable. Indeed, the distribution of slow wave generators determined from the spontaneous magnetoencephalogram by fitting single equivalent dipoles differed in patients with PTSD from patients with other psychiatric diagnoses. When compared to the norm group of healthy controls, PTSD patients displayed high concentrations of abnormal brain waves in the pre- and orbitofrontal cortex while depressed patients showed hypoactive regions, particularly in frontal areas (see Figure 4). The “dysfunctional” significance of this brain activity is supported by its variation with successful therapeutic intervention in PTSD (Elbert et al., 2005).

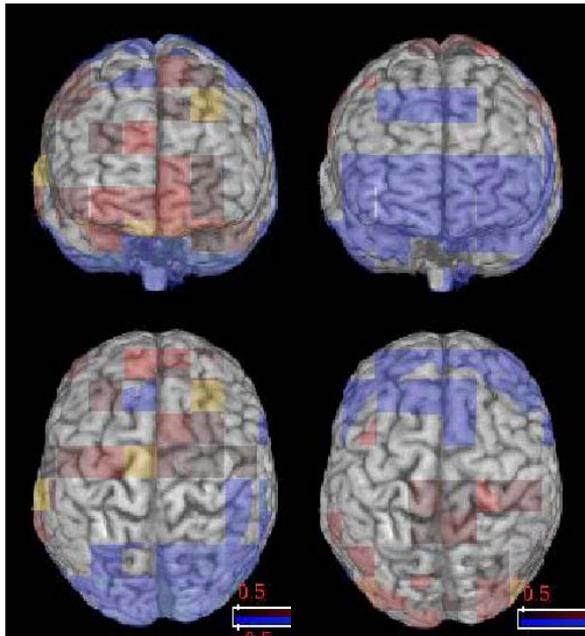


Figure 4: Mean regional distribution of abnormal slow waves for a group of 22 PTSD patients (left) and 15 depressive patients (right) relative to 25 normal controls. The top row offers a front view, the bottom row the perspective from above. Voxels with significant differences to control groups are marked with color. Yellow to red color indicate higher abnormal activity relative to controls, grey to blue colors indicate hypoactive areas. Displayed are mean z-values (data from Rockstroh et al., 2005 and Kolassa in preparation)

Several studies on PTSD patients with traumata resulting from combat, prolonged childhood abuse, rape, and traffic accidents have been conducted that analyzed structural changes associated with PTSD. As summarized by Geuze et al. (2005), the findings are mixed: A number of studies found evidence for significantly smaller right (Bremner et al., 1995), left (Bremner et al., 1997; Stein et al., 1997), or bilateral hippocampal volumes (Gurvits et al., 1996; Villarreal et al., 2002; Hedges et al., 2003) relative to comparison subjects. In contrast, a series of studies did not find evidence for hippocampal volume loss in PTSD (Bonne et al., 2001; Fennema-Notestine et al., 2002; Schuff et al., 2001; Neylan et al., 2003; Yamasue et al., 2003). The results are complicated even more by the findings of Gilbertson et al. (2002) who studied pairs of identical twins in which one member of each pair experienced combat in Vietnam, while the other stayed home. Combat veterans who developed PTSD had smaller hippocampi than combat veterans without PTSD. Furthermore, more severe PTSD was associated with an even smaller hippocam-

pus. However, the crucial finding was that the stay-at-home siblings of PTSD combat-veterans also had smaller hippocampi and the hippocampal volume of the stay-at-home siblings was even equally predictive of the severity of the combat sibling's PTSD. These results suggest that instead of being a consequence of stress, a small hippocampus might represent an increased vulnerability for developing PTSD in the aftermath of a trauma. On the other hand, we have described that each stressful experience increases the vulnerability to develop PTSD (Neuner et al., 2004a). Given that siblings share a history of traumatic stressors and assuming that the resulting vulnerability is somehow reflected in the hippocampal size, the results of Gilbertson may not contradict the assumption that traumatic stress affects the morphology of the brain and that of medial temporal lobe in particular.

Very little knowledge exists as to whether hippocampal damage in PTSD is reversible. Only two studies (Vermetten et al., 2003; Bremner et al., 2005) investigated this issue. The study by Vermetten et al. (2003) found that after 1-year of treatment with paroxetine (an SSRI), PTSD patients showed increased hippocampal volumes compared to baseline. These findings were paralleled by an amelioration of verbal declarative memory. Similarly, after a 3 months administration of phenytoin (an antiepileptic drug), Bremner et al. (2005) observed a significant increase in right brain volume. While the increase in right hippocampal volume was not statistically significant, a significant correlation between increases in hippocampal volume and reduction of PTSD symptoms was found.

Previous research mostly focused on the effects of stress on plasticity in the hippocampus. However, more recently a few studies have shown that other structures known to be involved in the fear circuitry of PTSD are also affected by stress. These are the amygdala and the medial prefrontal cortex (mPFC), and here in particular the anterior cingulate cortex (ACC). For these regions high concentrations of mineralocorticoid (MR) and glucocorticoid (GR) receptors have been described, highlighting the pivotal role of these regions in mediating stress-induced changes in attention and (emotional) memory (Erickson et al., 2002).

The amygdala plays a pivotal role in mediating stress-related effects on behaviour and modulating hippocampal function. One would expect similar structural changes in the

amygdala of severely traumatized persons as in the hippocampus. However, no differences in amygdala size or volume between patients with PTSD and subjects without PTSD have been found (de Bellis et al., 2002; 2001; Fennema-Notestine et al., 2002; Gurvits et al., 1996).

It has been suggested that the amygdala, and here in particular the basolateral nucleus (BLA), modulates hippocampus-dependent memory storage (Packard et al., 1994; Roozendaal & McGaugh, 1996). The BLA projects to the hippocampal dentate gyrus (Thomas et al., 1984). Lesions of the BLA and the basomedial amygdala (BMA), but not the central or medial nuclei, attenuate hippocampal LTP in the dentate gyrus (Ikegaya et al., 1994, 1996b), while stimulation of the BLA and BMA facilitate LTP in the dentate gyrus of rats (Ikegaya et al., 1996a). Thus, the amygdala seems to play an important role in mediating hippocampal neuroplasticity.

The human ACC is implicated in evaluating the emotional significance of stimuli, in attentional function, and in detecting errors of performance (Cardinal et al., 2002). More recently it has been suggested that the ACC “disambiguates” similar conditioned stimuli depending on their association with reinforcement to prevent generalization between conditioned stimuli, i.e. the ACC appears to discriminate similar stimuli (stimuli that share common elements) on the basis of their differential association with reinforcement. Some evidence for structural alterations in the ACC of traumatized patients exists. Rauch et al. (2003) found decreased volumes of the pregenual portion of the ACC (>25%) in combat nurses from the Vietnam War with PTSD compared to combat nurses without PTSD. The pregenual ACC, to which the results were specific, is thought to subserve affective function while the dorsal ACC is thought to subserve cognitive motor functions. However, more research on structural (and functional) alterations of the ACC in traumatized individuals with PTSD is necessary.

In conclusion, while animal research suggests a coherent picture of stress-induced changes in hippocampal structure and function, studies in individuals with PTSD provide a more complicated picture. The exact relationship between hippocampal atrophy and PTSD remains to be clarified. Animal research implies that the amygdala plays an important role in modulating hippocampal plasticity, but compared to the knowledge on hippocampal changes, little is known about the role of structural alterations of the amygdala in

the brains of individuals with PTSD. In addition, even less is known about structural changes in the anterior cingulate cortex in patients with PTSD and how these changes relate to PTSD symptomatology.

Finally, research indicates that instead of focusing on stress-induced changes in hippocampal plasticity and instead of investigating single brain structures, a shift to a more systemic approach is called for which takes possible modifications of interactions between relevant brain areas into account. Such evidence comes from a recent study by Maroun & Richter-Levin (2003) who showed that stress in rats was effective in blocking LTP in the CA1 region of the hippocampus as well as in the pathway from the BLA to the medial PFC. Thus, stress seems not only to affect neuronal plasticity in the hippocampus, but also in the medial prefrontal cortex, with the amygdala additionally being able to modulate hippocampal plasticity. It has been suggested that it may be an adaptive response to emotionally stressful conditions to block structures such as the hippocampus and the PFC that mediate higher-order behavior and to allow subcortical areas such as the amygdala to take more automatic control of behavioral responses (Mauron & Richter, 2003).

In sum, structural, functional, and neuroendocrine changes can be observed in the brains of survivors of organized violence that can be linked to the (re)organization of memory. In threat situations when flight is impossible, fight futile, and only freezing is left as a response option in our evolutionary repertoire, the functioning of frontal and medial temporal lobe structures, which form the gateway to autobiographical memory, is altered: “hot” and “cold” memories lose their connection. We suggested that this disconnection of hot and cold memories, accompanied by an enlarging fear network, explains flashbacks and the individual’s entrapment in speechless terror and fear. Since the hot emotional memory is disconnected from autobiographical cold memory, the victim is unable to relate the proper autobiographical dates and places of occurrence to the flashback episodes. If one could restore this connection, the horror of the memories might be alleviated. However, the psychic scar inflicted to the mind cannot be undone, but there are narrative approaches that can help to alleviate PTSD symptoms. Reweaving hot memory contents back into cold memory networks can bring relief to the injured mind. In addi-

tion, documenting and acknowledging human rights violations dignifies the victims of terror and organized violence, and enables them to tell their stories (Neuner et al., 2004b; Onyut et al., 2004; Schauer et al., 2005a).

VIOLENCE BREEDS VIOLENCE

In the previous section, we discussed how brain and mind are affected by experiences of organized violence. A resulting question is how stressful experiences influence individual behavior with its consequences for the society, including the way of dealing with violence. The saying “violence breeds violence” was coined 40 years ago by Curtis, in 1963, who expressed the concern that “abused and neglected children would become tomorrow’s murderers and perpetrators of other crimes of violence”. The validity of this finding obviously cannot be assessed by direct experimental manipulations. However, converging evidence exists that experiencing violence is intimately related to expressing violence: For instance, parents who were abused as children are more likely to abuse their own kids. Rates of abuse double for parents who themselves grew up in violent environments compared to parents who did not. Prospective and retrospective studies on children who were abused or neglected disclose a high incidence of later delinquency. Children clinically referred to residential treatment with a history of abuse scored significantly higher on measures of reactive and verbal aggression than non-abused control children (Conner et al., 2003). Finally, a large proportion of homicide offenders come from unfavorable home environments and up to 80% of subjects within delinquent samples report witnessing of violence in their childhood or adolescence.

It is important to note that effects are exerted from early on, i.e., when plasticity for the brain is greatest. Developmental studies indicate that abuse and neglect are related to aggression and later antisocial behavior in children as young as infants and toddlers. Thus, violent childhood experiences may leave their mark on the brain and mind of the affected individuals, a vulnerability that interacts with future stressful experiences. Indeed, childhood experience seem to be an important factor in this dynamical interaction, as Van der Kolk & Fisler (1994) emphasize: ‘Abused children often fail to develop the capacity to express specific and differentiated emotions: Their difficulty putting feelings into words interferes with flexible response strategies and promotes acting out’.

If stressful childhood experiences make susceptible to further stressors and the use of violence, can violence thus result as a consequence of traumatic stress or allostatic overload? Many studies addressing consequences of traumatic experiences in war veterans found increased impulsive aggression towards intimate partners (Byrne & Riggs, 1996) and unknown persons (e.g., Begic & Jokic-Begic, 2001; Silva et al., 2001). Similarly, high rates of traumatic experiences were found in a sample of juvenile delinquents (Abram et al., 1994). Over 90% of the sample (N = 898) had experienced one or more traumatic events; the most prominent event was witnessing violence. Approximately 11% of the sample even met criteria for PTSD in the past year. Thus, traumatic events seem to play an important role in individuals with violent or antisocial behavior.

Similar results come from two studies of our group on forensic psychiatric patients (Salepsi et al., 2004; Garieballa et al., submitted), which found higher than normal rates of PTSD among those patients. Similarly, Timmerman & Emmelkamp (2001) found sexual and emotional abuse to be significantly more prevalent among forensic patients than among prisoners. Using structural equation modelling, Orcutt et al. (2003) examined the impact of early-life stressors, war-zone stressors, and PTSD symptom severity on partner's reports of recent male-perpetrated intimate partner violence among 376 Vietnam veteran couples. Results revealed a direct relationship of war-zone stressors (i.e., traumatic stress) and PTSD symptom severity with intimate partner violence. In addition, indirect effects (i.e., via PTSD) of stressful early life experiences, childhood antisocial behavior, and traumatic war-zone experiences were found on intimate partner violence. Thus, experiencing PTSD symptoms as a result of previous trauma appears to increase an individual's risk for perpetrating intimate partner violence.

Another example of the relation between stressful life experiences and subsequent violent behavior becomes obvious in the results of a study by Freeman et al. (2003). War veterans with PTSD (Freeman et al. 2003) owned more than four times as many firearms as comparison groups of patients with schizophrenia or substance abuse, and they reported significantly higher levels of potentially dangerous firearm-related behaviors. Thus, experiencing violence that leads to trauma and PTSD lowers the threshold to exert violence. PTSD may increase the vulnerability for violence and impulsive aggression, in

particular when confronted with stress. However, it seems important to note that violent outbursts increase, but not organized violence!

Beyond trauma-related factors, cultural factors, i.e. the societies attitude to violence, plays a role in the prevalence and spread of violence. DeFronzo & Prochnow (2004) analyzed the rate of serial homicide across 50 States within the US and found that 34-45% of the interstate variation in rates of serial killer activity could be accounted for by dimensions of local culture, with higher rates of violence being found in states supporting game hunting, military training, and a local culture supporting punitive violence!

THE CULTURE OF ORGANIZED VIOLENCE

Currently, violence and atrocities in many regions of the world dominate every day's news. Rebels use fear and violence to maintain on the one hand control within the rebel movement, and on the other hand to sustain the conflict. Torture, cutting ears, lips, and throats are but one example of the instruments used to achieve these aims. The ongoing violence has displaced tens of millions of people. In many places, the economic base of whole regions was completely destroyed. In large epidemiological studies we showed that more than one fifth, in some refugee camps up to over 50% of the population, suffers from trauma spectrum disorders. This impairs not only the mental health and daily functioning on an individual level, but also compromises the reconstruction and development of society (Karunakara et al., 2004; Neuner et al., 2004a; Onyut et al., 2005).

Organized violence comprises war, torture, and other severe human rights violations that wound the psyche and cause mental illnesses. However, organized violence can not be understood just on the individual level, i.e., exclusively as a psychiatric problem, but must also be considered on the societal level, i.e., as a problem in politics or micropolitics. It is important to be aware of the political context of wars and torture to comprehend the meaning of organized violence for the individual and the community. Likewise, it is necessary to study the conditions under which violence breeds new violence, i.e., when does a victim become a perpetrator. In other words, in order to understand violence we need to study the co-constructive shaping of brain/mind and culture. Let us therefore consider the societal level first, and then discuss the co-constructivism in the next section.

Organized violence encompasses three types of violence (for a definition see Neuner, 2003). The first type is the permanent state-sponsored persecution that is present in all dictatorships, and even in some countries that are considered democracies. This harassment includes different forms of violence like torture, extralegal executions, disappearances etc. The second type is the massive violence committed against people in an interstate or civil war. The third type of organized violence is characterized by violence committed by terror organizations.

The public view of wars is dominated by knowledge about the 20th century's World Wars. However, research on current wars shows that wars characterized by two or more fighting countries are the exception rather than the rule. In 2001, more than nine out of ten wars (91%) were inner-state conflicts or civil wars (Schreiber, 2002). Although foreign armies may participate in the fights, these wars do not originate from conflicts between nations but arise within a country. There are two different reasons for civil wars: Currently, in about half of the inner-state conflicts, a rebel army fights for the autonomy or secession of a region. In the other half of the wars, rebels aim to overthrow the ruling regime. Kaldor (1999) introduced the term "New War" to describe these currently dominant ways of warfare. She recognized that the general characteristics of inner-state warfare, although not really new, have largely been neglected. Several characteristics of "New Wars" have been suggested since (Neuner, 2003):

- Irregular forces: The fighting is dominated by irregular forces, including paramilitary units, rebel forces, mercenary troops, and foreign armies that intervene in civil wars on one side. The majority of fighters on all sides of the conflicts have limited military training. Since many characteristics of regular armies, like uniforms and regular salaries, are not applicable to the majority of fighters, the clear separation between civilians and soldiers disappears. Forcibly recruited child soldiers belong to the usual repertoire of most forces in the new wars. The advantage of using children as fighters is that it is assumed that one can get children easily under control and can manipulate them to become unscrupulous fighters; children still lack norms and values and cannot judge risks and dangers in the same way as adults can (Schreiber, 2002).

- Justification on identities of the conflict partners, based on their affiliation to different ethnic groups, cultures, or religions. Myths about ancient rivalries and wars between the ethnic groups are used to motivate the public for the war.
- Warfare targets civilians since the best way to gain power in new wars is by controlling and frightening the civilian population, and by expelling civilians who do not belong to the group in power. Small weapons which are easily available worldwide will be sufficient for this type of warfare. Thus, new warfare strategies include systematic atrocities like massacres and mass rapes to frighten civilians and to make regions uninhabitable for the group to be expelled. The widespread use of landmines and the destruction of monuments are other means to achieve this goal. Another reason for the prevalence of atrocities in current wars is the assumption that they help to unite the group committing the atrocities. Once a person has participated in committing war crimes, it is almost impossible to leave the group since the perpetrator will always be rejected by others. At the beginning of the Rwandese genocide in 1994, which resulted in the killing of more than 800 000 Tutsis by the Hutus, each Hutu was under pressure to participate in the killings. Many reports state that the children who were recruited as soldiers were forced to commit atrocities in their own village. This prevented the children from fleeing from the forces and returning to their home villages, since they would be rejected if they returned.
- Economic factors: The observation of current wars suggests that rational motives cannot explain these excessively violent conflicts. Yet, detailed analyses of these wars show that it is not the case that wild and fanatic fighters kill each other at random. Instead, tangible interests motivate the main actors to wage these wars. Economic factors play an increasing role in the onset and maintenance of wars. In a global economy, the war parties are usually not self-sufficient but get resources from supporting foreign countries and exile communities. Very often, the conflicts are fought to win or keep control over local resources like diamonds, minerals, oil, and drugs. This consequently leads to powerful warlords who do not depend on governments. Since war offers them the opportunity to maintain power and to gain money without the control of any regulating institution, they have no immediate interest in a termination of war. Consequently, many wars are extended by deliber-

ately delaying peace negotiations and an unwillingness of both war parties to fight deciding battles against each other.

The consequences of violence and the resulting traumatic stress severely impact the daily lives of millions of people on the run. In the New Wars, more than 80% of casualties are civilians. In the next section, we will address the question of how this culture of violence affects the individuals living in it.

CULTURAL ADAPTATION AND MALADAPTATION – THE INTERACTION OF INDIVIDUAL AND COMMUNAL MIND

One to two months after the September 11th terrorist attacks, PTSD prevalence was 20% among New York inhabitants living close to the World Trade Center, while attack-related PTSD in other areas of New York was 7.5% (Galea et al., 2002). This study showed that organized violence in the form of terrorist attacks, at least in the immediate aftermath of the event, can have severe consequences for the mental health of populations even in industrialized countries. Notwithstanding the attention this attack received in the industrialized world's public, these kinds of terrorist attacks are still more the exception rather than the rule when compared to other forms of organized violence. The prevailing forms of organized violence are wars and persecution, which can cause forced migration.

The prevalence of PTSD in populations living in war regions varies with the type and number of experienced traumatic events. In some cases, prevalence rates of more than 50% can be found, i.e., more than half of the community suffers from this disabling condition, which impairs normal family life and renders the person unable to earn a living. This assumption is supported by one of our large scale projects (Karunakara et al., 2004) which screened 3.231 refugees in northern Uganda and southern Sudan. As mentioned earlier, this demographic survey revealed a surprisingly high prevalence of chronic mental illness. In one settlement with a count of approximately 12,000 refugees, 70% had experienced war, 78% had been threatened with a weapon, 61% had been assaulted, and 49% had been abused or tortured. The prevalence of disabling chronic mental illness in six camps ranged from 20% up to 56%. In one camp, more than half of the population was unable to function due to persistent mental problems. These and other epidemiological findings demonstrate that communities at large are affected and not just a few indi-

viduals. In a quantitative analysis of the health consequences of civil wars, Ghobarah et al. (2003) point out that “the direct and immediate casualties from civil wars are only the tip of the iceberg of their longer-term consequences for human misery” (p. 1).

How can we model this co-constructivism of individual and societal cycle of stress and violence? Beyond the fact that traumata and resulting trauma spectrum disorders are a *consequence* of violence, traumatic experiences can also *cause* domestic violence as well as violent wars and conflicts, a circumstance that receives more and more consideration at a political level. Organizations providing psychosocial interventions in war-affected societies justify their interventions not only as a means of improving mental health care for individuals, but also by referring to sociopolitical factors. A common statement is that the treatment of “traumatized societies” is necessary to break the “cycle of trauma” (Tauber, 2003; UNICEF, 2001). This reasoning is based on the assumption that traumatized individuals are more likely to become perpetrators themselves. However, through treatment one aims not only to reduce PTSD symptoms but also to foster reconciliation and forgiveness. It is unclear whether this truly is the case. While some investigations indicate that traumatized individuals are more likely to become perpetrators themselves, there is not enough evidence that confirms a “cycle of violence”.

Societies deal with matters of conflict resolution in different ways. One typical reaction of societies to political repression is to deny and minimize violence and its consequences. In times of political suppression this can be understood as a response to the threat of the ruling regime and the general climate of intimidation. Countries that have overcome war and dictatorship have to manage the incompatible interests of the victims on the one hand, who demand reparations and justice, and the interests of the perpetrators on the other hand. This conflict results in different developments within these societies.

One way to offer justice after conflict is to set up international tribunals that deal with war crimes and human rights violations committed during conflicts and dictatorships. For example, the UN set up tribunals after the Balkan war and the Rwandese genocide. The experiences from these tribunals show that they have to deal with two major difficulties. One problem is that the capacity of the courts is limited and only a small minority of the perpetrators can be charged. The other problem is that the danger of juridical consequences might prevent the perpetrators from withdrawing their power and this

might cause further conflicts. To overcome these problems, many countries, e.g., Nigeria and more recently Kenya, have set up truth commissions following the example of South Africa after the Apartheid regime. Truth commissions offer perpetrators the opportunity to confess their deeds in public, but they do not have judicial power, i.e., perpetrators who are willing to confess do not have to fear judicial consequences. Although truth commissions offer a chance to balance justice and peace, the consequence is that neither the victims nor the offenders are fully satisfied, and thus, the contribution of truth commissions to reconciliation is not yet proven. At present, are not experienced enough to make recommendations about the right way to deal with these issues. Justice and peace should be the major outcome variables to evaluate the efficacy of social institutions in processing the violent past.

OUTLOOK

Societal conflicts and civil war-affected communities provide an example of the interaction of brain, mind, and culture. This interaction should be of utmost interest to scientific investigation, public health, and politics. The brain's ability to adapt and reorganize also helps to understand the enduring effects of social stress and trauma on brain systems involved in the regulation of affect and memory. Another challenge for future research is to apply this knowledge of the stress/trauma-induced brain plasticity to the level of the society. Developing countries provide the most dramatic examples of "societal trauma". Although globalization contains a great deal of developmental chances for developing countries, the last decade has been characterized by a stunning stagnation of progress. Even though developing countries are oftentimes blessed with a wealth of natural resources and thus, should have the potential to develop to globally acting economy, they are handicapped by ongoing political instability, armed conflicts, and civil wars.

More than 10 sub-Saharan African countries are currently affected by civil wars. Many of these conflicts have lasted for decades. With few exceptions, most African countries have a recent history of armed conflicts and currently suffer from war's consequences. The devastating effects of civil wars result from the appalling degree of violence as well as from the destructive consequences of inner-state conflicts. These wars differ radically from traditional inter-state wars and thus, have been termed "new wars". Civil

wars are fought beyond international laws. Almost all civil wars are conducted by irregular forces, i.e. rebel armies, paramilitary forces, mercenary forces, and foreign armies. War parties commonly commit massacres and atrocities against the civilian population which serve to unite the troop. Documentations on current civil wars are full of reports on atrocities such as systematic killings, massacres, genocides, ethnic cleansings, and systematic strategies to make regions uninhabitable (e.g., laying landmines, destroying buildings, agricultural land, and equipment). Oftentimes, whole populations are forced to flee their homes. The effects of these conflicts on politics, society, economy, and (mental) health last for decades and have been termed as “development in reverse”.

Currently, psychosocial services in conflict and post-conflict settings have no feasible guidelines on how to treat mental disturbances caused by traumatic experiences. Very little is known about the usefulness of psychiatric concepts and therapeutic approaches for survivors of severe violence who above all still live in stressful and potentially dangerous conditions such as refugee settlements. Furthermore, it is unpredictable how long-term development will be influenced by the common mental health problems in the aftermath of trauma, particularly if one considers the lack of access to good-quality treatment.

It has also been argued that violence, conflict, and demoralization in these communities feed further violence, reinforcing a downward spiral. As pointed out there is some evidence that particularly early traumatic experiences may foster interfamilial and intimate partner violence, empirical evidence is weak though. There is no sufficient evidence to suggest that traumatic experiences promote organized violence on an individual or societal level. However, we only begin to understand the consequences of violence on the individual’s brain, mind, and behavior and how this impacts society. Perhaps, some day we will also comprehend the roots of violence and how they can be counteracted – a great endeavor. As long as the underlying mechanisms of violence are not properly understood, we will have focus on improving the therapeutic approaches for helping victims of severe violence.

REFERENCES

- Abram, K.M., Teplin, L.A., Charles, D.R., Longworth, S.L., McClelland, G.M., Dulcan, M. K. (1994). Posttraumatic stress disorder and trauma in youth in juvenile detention. *Arch Gen Psychiatry*, 61(4), 403-410.
- Alho K, Kujala T, Paavilainen P, Summala H, Näätänen R (1993). Auditory processing in visual brain areas of the early blind: evidence from event-related potentials. *Electroenceph Clin Neurophys*, 86, 418-427.
- Amunts, K., Schlaug, G., Jäncke, L., Steinmetz, H., Schleicher, A., Zilles, K. (1997). Motor cortex and hand motor skills: structural compliance in the human brain. *Human Brain Mapping* 5, 206–215.
- Baltes, P. & Singer, T. (2001). Plasticity and the ageing mind: An exemplar of the bio-cultural orchestration of brain and behaviour. *European Review* 9, 59-76.
- Baltes, P. (1999). Age and aging as incomplete architecture of human ontogenesis. *Z. Gerontol Geriatr.* 32, 433-448.
- Bao, S., Chan, V.T., Merzenich, M.M. (2001). Cortical remodeling induced by activity of ventral tegmental dopamine neurons. *Nature* 412, 79–83.
- Bao, S., Chang, E.F., Davis, J.D., Gobeske, K.T., Merzenich, M.M. (2003). Progressive degradation and subsequent refinement of acoustic representations in the adult auditory cortex. *J. Neurosci.*, 23(34), 10765–10775.
- Begic D, & Jokic-Begic N. (2001). Aggressive behavior in combat veterans with post-traumatic stress disorder. *Mil Med*, 166(8), 671-676.
- Bock, J., Helmeke, C., Ovtsharoff, W., Groß, M., Braun, K. (2003). Frühkindliche emotionale Erfahrungen beeinflussen die funktionelle Entwicklung des Gehirns. *NeuroForum* 2.03, 51-57.
- Bonne, O., Brandes, D. Gilboa, A., Gomori, J. M., Shenton, M. E., Pitman, R. K., Shalev, A. Y. (2001). Longitudinal MRI study of hippocampal volume in trauma survivors with PTSD. *American Journal of Psychiatry*, 158, 1248-1251.
- Braun, C. Haug, M., Wiech, K., Birbaumer, N., Elbert, T, Roberts, L. (2002). Functional organization of primary somatosensory cortex depends on the focus of attention. *Neuroimage* 17, 1451–1458.
- Braun, C., Schweizer, R., Elbert, T., Birbaumer, N., Taub, E. (2000) Differential activation in somatosensory cortex for different discrimination tasks. *J. Neurosci.* 20 :446-50.
- Bremner, J. D., Mletzko, T., Welter, S., Quinn, S., Willisams, C., Brummer, M., Siddiq, S., Reed, L., Heim, C. M., Nemeroff, C.B. (2005). Effects of phenytoin on memory, cognition and brain structure in post-traumatic stress disorder: a pilot study. *Journal of Psychopharmacology*, 19(2), 159-165.

- Bremner, J. D., Randall, P., Scott, T. M., Bronen, R. A., Seibyl, J. P., Southwick, S. M., Delaney R.C., McCarthy, G., Charney, D.S., Innis, R.B. (1995). MRI-based measurement of hippocampal volume in patients with combat-related posttraumatic stress disorder. *American Journal of Psychiatry*, 152, 973-981.
- Bremner, JD, Randall P, Vermetten E, Staib L, Bronen RA, Mazure C, Capelli S, McCarthy G, Innis RB, Charney DS (1997). Magnetic resonance imaging-based measurement of hippocampal volume in posttraumatic stress disorder related to childhood physical and sexual abuse – a preliminary report. *Biological Psychiatry*, 41(1): 23-32.
- Buchanan, T.W. & Lovallo, W.R. (2001). Enhanced memory for emotional material following stress-level cortisol treatment in humans. *Psychoneuroendocrinology*, 26, 307-317.
- Byrne, C.A. & Riggs, D. S. (1996). The cycle of trauma; relationship aggression in male Vietnam veterans with symptoms of posttraumatic stress disorder. *Violence Vict*, 11(3), 213-225.
- Cahill, L., Babinsky, R., Markowitsch, H.J., McGaugh, J.L. (1995). The amygdala and emotional memory. *Nature* 377, 295-6.
- Cahill, L., Prins, B., Weber, M., McGaugh, J.L. (1994). Beta-adrenergic activation and memory for emotional events. *Nature* 371, 702-704.
- Cardinal, R. N., Parkinson, J. A., Hall, J., Everitt, B. J. (2002). Emotion and motivation: the role of the amygdala, ventral striatum, and prefrontal cortex. *Neuroscience and Biobehavioral Reviews*, 26, 321-352.
- Charmandari E, Kino T, Souvatzoglou E, Chrousos GP (2003). Pediatric stress: hormonal mediators and human development. *Horm Res*, 59(4),161-179.
- Christian G, Schlaug G (2003). Brain Structures Differ between Musicians and Non-Musicians, *The Journal of Neuroscience*, 23(27), 9240-9245.
- Conner, D. F., Doerfler, L. A., Volungis, A. D., Steingard, R. J., & Melloni, R. H. (2003). Aggressive behavior in abused children, *An N Y Acad Sci*, 1008: 79-90.
- Curtis, G.C. (1963). Violence Breeds Violence – Perhaps? *Am J Psychiatry*, 120, 386.
- De Bellis, M. D., Hall, J., Boring, A. M., Frustaci, K., Moritz, G. (2001). A pilot longitudinal study of hippocampal volumes in pediatric maltreatment-related posttraumatic stress disorder. *Biological Psychiatry*, 50, 305-309.
- De Bellis, M. D., Keshavan, M. S., Spencer, S., Hall, J. (2000). N-Acetylaspartate concentration in the anterior cingulate of maltreated children and adolescents with PTSD. *American Journal of Psychiatry*, 157, 1175-1177.
- de Quervain, D.J.-F., Roozendaal, B., McGaugh, J.L. (1998). Stress and glucocorticoids impair retrieval of long-term spatial memory. *Nature* 394, 787-790.

- de Quervain, D.J.-F., Roozendaal, B., Nitsch, R.M., McGaugh, J.L., Hock, C. (2000). Acute cortisone administration impairs retrieval of long-term declarative memory in humans. *Nature Neurosci.* 3, 313-314.
- DeFronzo, J. & Prochnow, J. (2004). Violent cultural factors and serial homicide by males. *Psychol Rep*, 94(1), 104-108.
- Diamond DM, Weinberger NM. (1989) Role of context in the expression of learning-induced plasticity of single neurons in auditory cortex. *Behav Neurosci*, 103(3), 471-494.
- Elbert T, Neuner F, Schauer M, Odenwald M, Ruf M, Wienbruch C, Rockstroh B (2005) Successful psychotherapy modifies abnormal neural architecture in frontal cortex of traumatised patients. Paper presented at the conference of the European Society for Traumatic Stress Studies, Stockholm.
- Elbert T, Sterr A, Rockstroh B, Pantev C, Müller MM, Taub E (2002). Expansion of the tonotopic area in the auditory cortex of the blind. *J Neurosci*, 22, 9941-9944.
- Elbert T. & Rockstroh B. (2004). Reorganization of human cerebral cortex: the range of changes following use and injury. *The Neuroscientist*, 10(2):129-141.
- Elbert T. & Schauer, M. (2002). Psychological trauma: Burnt into memory. *Nature* 419, 883.
- Elbert, T. & Heim, S. (2001). Cortical Reorganization, a light and a dark side. *Nature* 411, 139.
- Elbert, T. & Rockstroh, B. (2003). Stress factors. The science of our flexible responses to an unpredictable world. *Nature* 421, 477-478.
- Elbert, T., Candia, V., Altenmüller, E., Rau, H., Sterr, A., Rockstroh, B., Pantev, C., Taub, E. (1998). Alteration of digital representations in somatosensory cortex in focal hand dystonia. *Neuroreport* 16, 3571-5.
- Elbert, T., Pantev, C., Wienbruch, C., Rockstroh, B., Taub, E. (1995). Increased use of the left hand in string players associated with increased cortical representation of the fingers. *Science*, 270, 305-307.
- Erickson, K., Drevets, W., Schulkin, J. (2003). Glucocorticoid regulation of diverse cognitive function in normal and pathological emotional states. *Neuroscience and Biobehavioral Reviews*, 27, 233-246.
- Fennema-Notestine, C., Stein, M. B., Kennedy, C. M., Archibald, S. L., Jernigan, T. L. (2002). Brain morphometry in female victims of intimate partner violence with and without posttraumatic stress disorder. *Biological Psychiatry*, 52, 1089-1101.
- Freeman, T.W., Roca, V., & Kimbrell, T. (2003). A survey of gun collection and use among three groups of veteran patients admitted to veterans affairs hospital treatment programs. *South Med J*, 96(3), 240-243.
- Fullerton CS, Ursano RJ, Epstein RS, Crowley B, Vance KL, Kao TC, Baum A (2000). Peritraumatic dissociation following motor vehicle accidents: relationship to prior trauma and prior major depression. *J Nerv Ment Dis*, 188, 267-272.

- Galea, S., Resnick, H., Ahern, J., Gold, J., Bucuvalas, M., Kilpatrick, D., Stuber, J., Vlahov, D. (2002). Posttraumatic stress disorder in Manhattan, New York City, after the September 11th terrorist attacks. *J Urban Health*, 79(3), 3340-353.
- Gariebballa S., Schauer M., Saleptsi E., Kluttig T., Hoffmann K., Neuner F., Rockstroh B. and Elbert T. (2005). Traumatic Events, PTSD, and Psychiatric Comorbidity in Forensic Patients (submitted).
- Geuze, E., Vermetten, E. & Bremner, J. D. (2005). MR-based in vivo hippocampal volumetrics: 2. Findings in neuropsychiatric disorders. *Molecular Psychiatry*, 10, 160-184.
- Ghobarah, H., Huth, P., & Russett, B. (2003). Civil wars kill and maim people - long after the shooting stops. *American Political Science Review*, 97.
- Gilbertson, M. W., Shenton, M.E., Ciszewski, A., Kasai, K., Lasko, N.B., Orr, S.P., Pitman, R.K. (2002). Smaller hippocampal volume predicts pathologic vulnerability to psychological trauma. *Nature Neuroscience* 5, 1242-1247.
- Gougoux F, Lepore F, Lassonde M, Voss P, Zatorre RJ, et al. (2004) Neuropsychology: Pitch discrimination in the early blind. *Nature* 430: 309.
- Gougoux F, Zatorre RJ, Lassonde M, Voss P, Lepore F, (2005) A Functional Neuroimaging Study of Sound Localization: Visual Cortex Activity Predicts Performance in Early-Blind Individuals. *PLoS Biology*, 3(2), e27.
- Gurvits, T. V., Chenton, M. E., Hokama, H., Ohta, H., Lasko, N. B., Gilbertson, M. W., Orr, S.P., Kikinis, R., Jolesz, F.A., McCarley, R.W., Pitman, R.K. (1996). Magnetic resonance imaging study of hippocampal volume in chronic, combat-related posttraumatic stress disorder. *Biological Psychiatry*, 40, 1091-1099.
- Hamilton RH, Pascual-Leone A, Schlaug G (2004) Absolute pitch in blind musicians. *Neuroreport* 15: 803–806.
- Hedges, D. W., Allen, S. Tate, D. F., Thatcher, G. W., Miller, M. J., Rice, S. A., Cleavinger, H.B., Sood, S., Bigler, E.D. (2003). Reduced hippocampal volume in alcohol and substance naïve Vietnam combat veterans with posttraumatic stress disorder. *Cognitive and Behavioral Neurology*, 16, 219-224.
- Heinrichs M, Baumgartner T, Kirschbaum C, Ehlert U. (2003). Social support and oxytocin interact to suppress cortisol and subjective responses to psychosocial stress. *Biol Psychiatry*, 54(12), 1389-1398.
- Heinrichs M, Meinlschmidt G, Neumann I, Wagner S, Kirschbaum C, Ehlert U, Hellhammer DH. (2001). Effects of suckling on hypothalamic-pituitary-adrenal axis responses to psychosocial stress in postpartum lactating women. *J Clin Endocrinol Metab*, 86(10), 4798-4804.
- Hull T, Mason H (1995) Performance of blind children on digit-span tests. *J Vis Impair Blindn* 89: 166–169.
- Iidaka T, Sadato N, Yamada H, Yonekura Y, (2000) *Brain Res Cogn Brain Res*. 9, 73.

- Ikegaya Y, Saito H, Abe K (1996). Attenuated hippocampal long-term potentiation in basolateral amygdala-lesioned rats. *Brain Res*, 656(1), 157-164.
- Ikegaya Y, Saito H, Abe K (1996a). Dentate gyrus field potentials evoked by stimulation of the basolateral amygdaloid nucleus in anesthetized rats. *Brain Res*, 718(1-2), 53-60.
- Ikegaya Y, Saito H, Abe K (1996b). The basomedial and basolateral Amygdaloid nuclei contribute to the induction of long-term potentiation in the dentate gyrus in vivo. *Eur J Neurosci*, 8(9), 1833-1839.
- Junghöfer M, Schauer M, Neuner F, Odenwald M, Rockstroh B, Elbert T (2003) Enhanced fear-network in torture survivors activated by RVSP of aversive material can be monitored by MEG. *Psychophysiology*, 40, Supplement, S51.
- Junghöfer, M., Bradley, M., Elbert, T., Lang, P. (2001) Fleeting images: A new look at early emotion discrimination. *Psychophysiology*, 38, 175-178.
- Kaldor, M. (1999). *New and old wars: organized violence in a global era*. London: Blackwell.
- Karunakara UK, Neuner F, Schauer M, Singh K, Hill K, Elbert T, Burnha G. (2004). Traumatic events and symptoms of post-traumatic stress disorder amongst Sudanese nationals, refugees and Ugandans in the West Nile. *Afr Health Sci*, (2), 83-93.
- Kilgard, M.P., Merzenich, M.M. (1998). Cortical map reorganization enabled by nucleus basalis activity. *Science* 279, 1714–1718.
- Koopman C, Classen C, Spiegel D (1994). Predictors of posttraumatic stress symptoms among survivors of the Oakland/Berkeley, Calif., firestorm. *Am J Psychiatry*, 151, 888-894.
- Kujala T, Alho K, Huotilainen M, Ilmoniemi RJ, Lehtokoski A, Leinonen A, Rinne T, Salonen O, Sinkkonen J, Standertskjöld-Nordenstam CG, Näätänen R. (1997). Electrophysiological evidence for cross-modal plasticity in humans with early- and late-onset blindness. *Psychophysiology*. 34(2), 213-216.
- Kujala T, Alho K, Paavilainen P, Summala H, Näätänen R. (1992). Neural plasticity in processing of sound location by the early blind: an event-related potential study. *Electroencephalogr Clin Neurophysiol*, 84(5), 469-472.
- Kujala T, Huotilainen M, Sinkkonen J, Ahonen AI, Alho K, Hämäläinen MS, Ilmoniemi RJ, Kajola M, Knuutila JE, Lavikainen J, Salonen, O., Simola, J., Standertskjöld-Nordenstam, C.-G., Tiitinen, H., Tissari, S.O. & Näätänen, R. (1995). Visual cortex activation in blind humans during sound discrimination. *Neurosci Lett*, 183(1-2), 143-146.
- Lang, P. J., Bradley, M. M., & Cuthbert, B. N. (1999). *International affective picture system (IAPS): Instruction manual and affective ratings*. Technical Report A-4. Gainesville, FL: The Center for Research in Psychophysiology, University of Florida.

- Lessard, N., Pare, M., Lepore, F., & Lassonde, M. (1998). Early-blind human subjects localize sound sources better than sighted subjects. *Nature*, 392, 811-814.
- Marmar CR, Weiss DS, Metzler T (1998). Peritraumatic dissociation and posttraumatic stress disorder, in trauma, memory, and dissociation. Bremner JD, Marmar CR (Eds.) Washington, DC, American Psychiatric Press, 229-252.
- Maroun M, Richter-Levin G. (2003). Exposure to acute stress blocks the induction of long-term potentiation of the amygdala-prefrontal cortex pathway in vivo. *J Neurosci*, 23(11), 4406-4409.
- McEwen, B. (2004) Protection and damage from acute and chronic stress. *Ann Y Acad Sci*, 1032, 1-7.
- McEwen, B.S., Lasley, E. N., Lasley, E. (2002). *The End of Stress as We Know It*. Joseph Henry Press and Dana Press, Washington, DC, 2002.
- McGaugh, J.L. (2002). Memory consolidation and the amygdala: a systems perspective. *Trends in Neuroscience* 25, 456.
- McNally RJ, (2003). Psychological mechanisms in acute response to trauma. *Biol Psychiatry* 53(9), 779-788.
- Meaney, M., Aitken, D., van Berkel, C. Bhatnagar, C., Sapolsky, R. (1988). Effects of neonatal handling on age-related impairments associated with the hippocampus. *Science* 239, 766-770.
- Metcalve, J., & Jacobs, W. (1996). A "hot-system/cool-system" view of memory under stress. *PTSD Research Quarterly*, 7, 1-3.
- Michelson, L Ray WJ, Eds. (1996). *Handbook of Dissociation: Theoretical, Empirical, and Clinical Perspectives*. Plenum Publishing, New York.
- Muchnik C, Efrati M, Nemeth E, Malin M, Hildesheimer M (1991) Central auditory skills in blind and sighted subjects. *Scand Audiol* 20: 19–23.
- Münte, T.F., Altenmüller, E., Jäncke, L. (2002). The musician's brain as a model of neuroplasticity. *Nat.Rev. Neurosci.* 3, 473-478.
- Neuner F, Schauer M, Klaschik C, Karunakara U, Elbert T. (2004b) A comparison of narrative exposure therapy, supportive counselling, and psychoeducation for treating posttraumatic stress disorder in an African refugee settlement. *J Consult Clin Psychol*, 72(4), 579-587
- Neuner, F (2003). *Epidemiology and Treatment of Posttraumatic Stress Disorder in West-Nile Populations of Sudan and Uganda*. Dissertation. Universität Konstanz. www.ub.uni-konstanz.de/kops/volltexte/2003/1082/pdf/dissNeuner.pdf
- Neuner, F., Schauer, M., Karunakara, U., Klaschik, C., Robert C., Elbert, T. (2004a). Psychological trauma and evidence for enhanced vulnerability for PTSD through previous trauma in West Nile refugees *BMC Psychiatry*, 4(1), 34.

- Neuner, F., Schauer, M., Roth, W.T. Elbert, T. (2002) Testimony Therapy as an Acute Intervention in a Macedonian Refugee Camp: Two Case Reports. *Behav Cogn Psychotherapy*, 30, 205-209.
- Neylan, T. C., Shuff, N., Lenoci, M., Yehuda, R., Weiner, M. W., Marmar, C. R. (2003). Cortisol levels are positively correlated with hippocampal N-acetylaspartate, *Biological Psychiatry*, 54(10), 1118-1121.
- Niemeyer W, Starlinger I (1981) Do the blind hear better? Investigations on auditory processing in congenital or early acquired blindness. II. Central functions, *Audiology* 20, 510-515.
- Odenwald M, Neuner F, Schauer M, Elbert T, Catani C, Lingenfelder B, Hinkel H, Hafner H, Rockstroh B. (2005). Khat use as risk factor for psychotic disorders: a cross-sectional and case-control study in Somalia. *BMC Med*, 3(1), 5.
- Onyut LP, Neuner F, Schauer E, Ertl V, Odenwald M, Schauer M, Elbert T. (2005). Narrative Exposure Therapy as a treatment for child war survivors with posttraumatic stress disorder: two case reports and a pilot study in an African refugee settlement. *BMC Psychiatry*, 5(1), 7.
- Onyut, PL, Neuner F, Schauer E, Ertl V, Odenwald, M., Schauer, M, Elbert T (2004) The Nakivale Camp Mental Health Project: Building local competency for psychological assistance to traumatised refugees. *Intervention* 2 (2), 90-107.
- Orcutt, H.K., King, L.A., & King, D.W. (2003). Male-perpetrated violence among Vietnam veteran couples: relationships with veteran's early life characteristics, trauma history, and PTSD symptomatology. *J Trauma Stress*. 16(4), 381-390.
- Packard, M. G., Cahill, L., McGaugh, J. L. (1994). Amygdala modulation of hippocampal-dependent and caudate nucleus-dependent memory processes. *Proceedings of the National Academy of Sciences of the USA*, 91, 8477-8481.
- Pantev, C., Oostenveld, R., Engelien, A., Ross, B., Roberts, L.E., & Hoke, M. (1998). Increased auditory cortical representation in musicians. *Nature*, 392, 811-814.
- Paus, T. (2005) Mapping brain maturation and cognitive development during adolescence. *Trends in Cognitive Sciences*, 9, 60-68.
- Rauch, S. L., Shin, L. M., Segal, E., Pitman, R. K., Carson, M.A., McMullin, K., Whalen, P.J., Makris, N. (2003). Selectively reduced regional cortical volumes in post-traumatic stress disorder. *Neuroreport*, 14: 913-916.
- Recanzone, G.H., Merzenich, M.M., Jenkins, W.M., Grajski, K.A., Dinse, H.R. (1992). Topographic reorganization of the hand representation in cortical area 3b owl monkeys trained in a frequency-discrimination task. *J Neurophysiol*. 67, 1031–1056.
- Recanzone, G.H., Schreiner, C.E., Merzenich, M.M. (1993). Plasticity in the frequency representation of primary auditory cortex following discrimination training in adult owl monkeys. *J. Neurosci*. 13, 87–103.

- Rockstroh, B, Ray, W, Wienbruch, C, Elbert, T (2005). Identification of dysfunctional cortical network architecture and communication: abnormal slow wave activity mapping (ASWAM) in neurological and psychiatric disorders (submitted).
- Röder B, Gelmuth L, Rösler F (2003) Semantic and morpho-syntactic priming in auditory word recognition in congenitally blind adults. *Lang Cogn Processes* 18: 1–20.
- Röder B, Rösler F, Hennighausen E, Nacker F. (1996). Event-related potentials during auditory and somatosensory discrimination in sighted and blind human subjects. *Brain Res Cogn Brain Res*, 4(2), 77-93.
- Röder B, Rösler F, Neville HJ (2001) Auditory memory in congenitally blind adults: A behavioural-electrophysiological investigation. *Brain Res Cogn Brain Res* 11: 289–303.
- Röder B, Teder-Sälejärvi W, Sterr A, Rösler F, Hillyard SA, et al. (1999) Improved auditory spatial tuning in blind humans. *Nature* 400: 162–166.
- Rooszendaal, B. & McGaugh, J. L. (1996). Amygdaloid nuclei lesions differentially affect glucocorticoids-induced memory enhancement in an inhibitory avoidance task. *Neurobiology of Learning and Memory*, 65, 1-8.
- Rooszendaal, B., de Quervain, D.J.-F., Ferry, B., Setlow, B., McGaugh, J.L. (2001). Basolateral amygdala-nucleus accumbens interactions in mediating glucocorticoid enhancement of memory consolidation. *J. Neurosci.* 21, 2518-2525.
- Rooszendaal, B., Griffith, Q.K., Buranday, J., de Quervain, D.J.-F., McGaugh, J.L. (2003). The hippocampus mediates glucocorticoid-induced retrieval impairments of spatial memory: Dependence on the basolateral amygdala. *PNAS* 100, 1328-1333.
- Rösler, F., Röder, B., Heil, M., & Hennighausen, E. (1993). Topographic differences of slow event-related brain potentials in blind and sighted adult human subjects during haptic mental rotation. *Cognitive Brain Research*, 1, 145-159.
- Saleptsi, E., Bichescu, D., Rockstroh, B., Neuner, F., Schauer, M., Studer, K., Hoffmann, K., Elbert, T. (2004). Association between psychiatric diagnoses and negative and positive childhood experiences during the different developmental periods. *BMC Psychiatry*, 4, 40.
- Sapolsky, R.M. (1999). Glucocorticoids, stress, and their adverse neurological effects: relevance to aging. *Exp. Gerontol.* 34, 721-732.
- Schaal S, Elbert T (2005.) Ten years after the genocide: Trauma confrontation and posttraumatic stress in Rwandan adolescents. *J Traumatic Stress* (in press).
- Schauer M, Ray WJ, Odenwald M, Neuner F, Ruf M, Rockstroh B, Elbert T (2005b) Decoupling neural networks from reality: Dissociative experiences modify the neural architecture in left frontal cortex. Paper presented at the conference of the European Society for Traumatic Stress Studies, Stockholm.

- Schauer, M., Neuner, F. & Elbert, T. (2005a) Narrative Exposure Therapy. A short-term intervention for traumatic stress disorder after war, terror or torture. Göttingen, Germany: Hogrefe & Huber.
- Schauer, M., Neuner, F., Karunakara, U., Klaschik, C., Robert, C., Elbert, T. (2003). PTSD and the “building block” effect of psychological trauma among West Nile Africans. *ESTSS Bulletin*, 10 (2), 5-6.
- Schlaug, G., Jäncke, L., Huang, Y., Staiger, J.F., Steinmetz, H. (1995). Increased corpus callosum size in musicians. *Neuropsychologia* 33, 1047-1055.
- Schreiber, W. (2002). *Das Kriegsgeschehen 2001*. Opladen: Leske und Budrich.
- Schuff, N., Neylan, T. C., Lenoci, M. A., Du, A. T., Weiss, D. S., Marmar, C. R., Weiner, M.W. (2001). Decreased hippocampal N-acetylaspartate in the absence of atrophy in posttraumatic stress disorder. *Biological Psychiatry*, 50, 952-959.
- Shalev AY, Peri T, Canetti L, Schreiber S (1996). Predictors of PTSD in injured trauma survivors: a prospective study. *Am J Psychiatry*, 153, 219-225.
- Silva JA, Derecho DV, Leon GB, Weinstock R, Ferrari MM (2001). A classification of psychological factors leading to violent behavior in posttraumatic stress disorder. *J Forensic Sci*, 46(2), 309-316.
- Steckler, T., Kalin, N., & Reul, J. (Eds.) (2005). *Handbook of Stress and the Brain*, Vol. 1 Amsterdam, Elsevier.
- Stein, M. B., Koverola, C., Hanna, C., Torchia, M. G., McClarty, B. (1997). Hippocampal volume in women victimized by childhood sexual abuse. *Psychological Medicine*, 27, 951-959.
- Steinberg, L. (2005) Cognitive and affective development in adolescence. *Trends in Cognitive Sciences*, 9, 69-74.
- Tauber, C. D. (2003). Psychological trauma, physical health and conflict resolution in Croatia, Serbia and Bosnia: lessons for the future. Retrieved 17.02, 2003, from www.conflictres.org/vol184/tauber.htm
- Teicher, M.H., Andersen, S.L., Polcari, A., Anderson, C.M., Navalta, C.P. (2002). Developmental neurobiology of childhood stress and trauma. *Psychiatr. Clin. North Am.* 25, 397-426.
- Thomas, S. R., Assaf, S. Y., Iversen, S. D. (1984). Amygdaloid complex modulates neurotransmission from the entorhinal cortex to the dentate gyrus of rat. *Brain Research*, 307, 363-365.
- Thompson, J., Pogue-Geile, M., Grace, A. (2004) Developmental pathology, dopamine, and stress: A model for the age of onset of schizophrenia symptoms. *Schiz Bulletin*, 30, 875-900.
- Timmerman, I.G. & Emmelkamp, P.M. (2001). The relationship between traumatic experiences, dissociation, and borderline personality pathology among male forensic patients and prisoners. *J Personal Disord*, 15(2), 136-149.
- Uhl F, Franzen P, Podreka I, Steiner M, Deecke L. (1993). Increased regional cerebral blood flow in inferior occipital cortex and cerebellum of early blind humans. *Neurosci Lett*, 150(2),162-164.

- UNICEF. (2001). *The state of the world's children 2001*. New York: UNICEF.
- Van der Kolk, BA & Fisler, R.E. (1994). Childhood abuse and neglect and loss of self-regulation. *Bull Menninger Clin*, 58(2),145-68.
- Vermetten, E., Vythilingam, M., Southwick, S. M., Charney, D. S., Bremner, J. D. (2003). Long-term treatment with paroxetine increases verbal declarative memory and hippocampal volume in posttraumatic stress disorder. *Biological Psychiatry*, 54(1), 693-702.
- Villareal, G., Hamilton, D. A., Petropoulos, H., Driscoll, I, Rowland, L. M., Griego, J. A., Kodituwakku, P.W., Hart, B.L., Escalona, R., Brooks, W.M. (2002). Reduced hippocampal volume and total white matter volume in posttraumatic stress disorder. *Biological Psychiatry*, 52, 119-125.
- Wiesel, T.N., Hubel, D.H. (1965) Comparison of the effects of unilateral and bilateral eye closure on cortical unit responses in kittens. *J Neurophysiol* 28, 1029–1040.
- Yamasue, H., Kasai, K., Iwanami, A., Ohtani, T., Yamada, H., Abe, O., Kuroki, N., Fukuda, R., Tochigi, M., Furukawa, S., Sadamatsu, M., Sasaki, T., Aoki, S., Ohtomo, K., Asukai, N., Kato, N. (2003). Voxel-based analysis of MRI reveals anterior cingulate gray-matter volume reduction in posttraumatic stress disorder due to terrorism. *Proceedings of the National Academy of Sciences of the USA*, 100, 9039-9043.
- Zhang, L.I., Bao, S., Merzenich, M.M. (2001). Persistent and specific influences of early acoustic environments on primary auditory cortex. *Nat. Neurosci.* 4, 1123–1130.