Early Stress and its Long-term Effects on Health – State of the Art and Implications for Future Research[1]

U.T. Egle[2], J. Hardt[2], R. Nickel[2], B. Kappis[2], S.O. Hoffmann[2]

Abstract

There is evidence from some prospective and various retrospective studies that early biological and psychosocial stress in childhood is associated with long-term vulnerability to various mental and physical diseases. Recent research has detected emotional, behavioral and psychobiological factors that can affect health and well-being over an entire life span. They lead to an increased risk of somatization and other mental disorders. Anxiety, depression, and personality disorders particularly often result in high risk behavior that itself is associated with physical disease (cardiovascular, stroke, viral hepatitis, type 2 diabetes, chronic lung diseases) as well as with aggressive behavior. An overview is presented here on how these various factors interact, and a bio-psychopathological model of vulnerability is described. Implications for future research are outlined and contrasted to actual political trends in Germany.

Key words

Adverse childhood experience – Stress – Developmental psychopathology – Psychobiology – Attachment behavior – Family – Vulnerability

It was not until the Industrial Revolution of the 19th century that western industrial countries became widely sensitized to the abuse and neglect of children. Childhood came to be recognized as a particularly vulnerable phase of life in need of special protection (Ariès 1975). Early psychoanalytical work (Freud 1917, Abraham 1924) pointed out the connection between an early traumatic experience of the loss of a parent and the later appearance of a depressive disorder. However, the systematic investigation of the effects of early stress factors did not begin until the second half of the 20th century with Bowlby's work (1951) commissioned by the WHO. Bowlby came to the conclusion that long periods of deprivation

[1] Supported by the German Research Association (DFG) and the Ministry of Education, Women, and Youth, Rheinland-Pfalz, Germany.

[2] Dep. of Psychosomatic Medicine and Psychotherapy, University Mainz.

from maternal care during early childhood can have serious and far-reaching consequences for personality development and, as such, have lasting effects across the entire life span of an individual.

Early psychosocial risk factors with long-term effects

In the wake of Bowlby's discoveries, the long-term effects of childhood risk factors on mental and physical health in adulthood were further investigated. Significant contributions were made through research on attachment behavior driven decisively by Bowlby himself against the background of early attachment experiences and a series of prospective longitudinal studies on birth cohorts. These studies include the Kauai study (Werner 1989, 1992, 1997) as well as three national birth cohort studies in Great Britain (e.g. Wadsworth 1987, Wadsworth & Kuh 1997, van Os et al. 1997, Schoon & Montgomery 1997), and one study in New Zealand (Fergusson & Lynskey 1995, 1996). Perhaps the most significant of these was the Kauai study which recruited all 698 children born in 1955 on Kauai, one of the Hawaiian Islands. This study revealed a distinct connection between a cumulative effect of biological and psychosocial factors during childhood on the one hand, and psychosocial abnormalities in adulthood on the other. Specific early risk factors emerged as indicators for the later appearance of delinquency and criminality: the lack of a secure care giver and/or long periods of separation from this person during the first year of life. In contrast to many later studies, the Kauai study not only narrowed its focus to investigate the meaning of psychosocial stress factors, it also took into account protective factors. For the first time, the partially compensatory function of protective factors could be proven prospectively. In this context, Werner (1989) differentiated between three types of protective factors:

- individual factors: degree of activity and good social behavior, at least average intelligence, good communicative abilities (language, reading), and internal locus of control;
- an emotionally stable and empathetic relationship with at least one parent, sibling, or later partner or friend that affords support in stressful situations;
- external support systems: school, work, or religious institutions that contribute to promoting individual talents and to the development of a positive attitude towards life.

Thus, there exists by all means a certain flexibility in the development of an individual, which today is subsumed under the term ”resilience”: Even under the most difficult developmental circumstances, a positive development can be possible, if appropriate protective factors are present during certain developmental phases. This can even contribute to a relatively higher resistance to stress in later life (Bender & Lösel 2000). If we take into account the results of

other longitudinal studies that only partly cover the very broad spectrum of stress and protective factors in the Kauai study and the results of carefully conceptualised retrospective studies (for a review see Egle & Hoffmann 2000, Egle et al. 2002), however, a limited number of evidence-based psychosocial stress factors emerges. Multiple occurrence of these factors can lead to long-term effects which themselves can, nonetheless, be at least partially alleviated by a significantly fewer number of compensatory protective factors (see Table 1). Even though the risk factors include some genetically determined parameters, such as temperament, intelligence, and sex, a weighting of ”nature” versus ”nurture” has yet to be carried out with regard to the occurrence of mental disorders and/or physical diseases in later life. This research deficit continues to prevail despite Rutter’s call for such an investigation eight years ago (1994). One of the few exceptions is the study by Kendler et al. (1992) on depressive and anxiety diseases in a large sample of twins. This study was able to establish that the ”environmental factor” of separation from the parents or loss of a parent contributes to increased vulnerability in adulthood, independent of genetic predisposition. In one of our own studies on n=407 consecutive patients from the psychosomatic outpatient department (Nickel & Egle 2001, Egle et al. in prep.), the factor ”loss of a parent by death” ranked in frequency in last position out of a total of 14 systematically surveyed childhood risk factors. Indeed, experiences of severe sexual abuse (penetration and/or genital manipulation) at a frequency of occurrence of just under 10% did not belong to the most frequent stress factors of this selected patient population (see Table 1). Emotional neglect, chronic family discord, and chronic physical or mental illness in one parent occurred with much greater frequency during childhood in this sample. Along with the factor of regular physical punishment, which occurs with even greater frequency in our (German) societal environment, sexual abuse represents only one marker for a high-risk childhood: on average, patients with these two risk factors also have experienced five additional other risk factors. An Australian study also doubts the reliance on the long-term effects of sexual abuse only, whose importance has often been overly exaggerated during the past decade in the wake of a (by all means necessary) lifting of taboos (Mullen et al. 1993, 1996). In Germany, physical maltreatment, persistent family conflicts – at times physical fights – and in particular emotional neglect occur with much higher frequency than sexual abuse (Engfer 2000). A father who is absent during the sensitive years of childhood development can represent a risk factor for the later emotional health of children (Werner & Smith 1982, Franz et al. 1999). In Germany, the proportion of single parent families has grown over the past years and is currently approximately 20% (2.82 million, approx. 90% of whom are single mothers). Studies show a decline in social status and increase in the overall burden on health in this population group. The socio-economic status

of single mothers is distinctly reduced (lower income, lower education level, dependence on welfare 39.0% versus 2.5% in the control group) and mental stress is significantly increased (Franz et al. 2001). Few differences were found between younger (year of birth 1965-75) and older (year of birth 1935-50) patients from the psychosomatic department of a university hospital (Egle et al. in prep.): The loss of a parent was, as was to be expected, significantly more frequent in the older patients, while separation and divorce of the parents were more frequent in the younger generations. As for the frequency of physical and sexual abuse, the differences were just as minimal as for the other factors assessed. Highly significant sex effects (increased rate for women) could only be observed in severe sexual abuse, which was reported by women about three times as often as by men. Compared to the prevalence rates in the German general population both female as well as male patients reported 2 to 3 times more cases of sexual abuse(see Wetzels 1997).

Table 1: Empirically established risk factors and protective factors with potentially long-term effects

Risk factors

- Low socio-economic status
- Poor education level of the parents
- Unemployment
- Large families and very little living space
- Contact with institutions of ”social control” (e.g. youth welfare department)
- Criminality or anti-sociality of a parent
- Chronic discord in the primary family
- Mother working during first year of life
- Insecure attachment behavior
- Mental disorder of the mother/father
- Severe physical illness of the mother/father
- Chronically ill sibling
- Single mother
- Autoritarian behavior of the father
- Loss of the mother
- Long period of separation from the parents during the first 7 years of life
- Persistent conflicts as a result of divorce or separation of the parents
- Frequently changing early relationships
- Sexual and/or physical abuse
- Poor contacts with peers in school
- Age difference to next sibling <18 months
- High overall risk
- Gender: Boys more vulnerable than girls

Compensatory protective factors

- Long-term good relationship with at least one primary care giver
- Secure attachment behavior
- Large family, compensatory parental relationships
- Relief of the mother (above all in single mothers)
- Good substitute environment following early loss of mother
- Above average intelligence

- Robust, active, and communicative temperament
- Internal locus of health control, ”self-efficacy”
- Social support (e.g. youth groups, school, church)
- Reliably supportive care giver(s) in adulthood
- Later founding of a family (in the sense of taking on responsibility)
- Low overall risk
- Gender: girls less vulnerable than boys

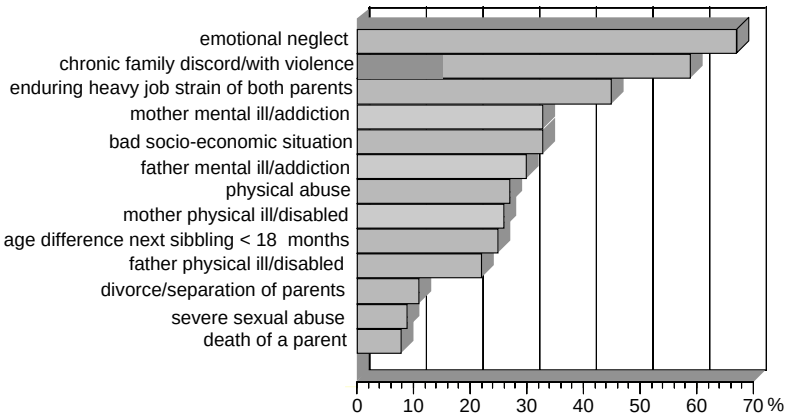

Fig. 1: Childhood adversities: frequencies of the population (n=407) of a psychosomatic outpatient department of an university hospital

Long-term effects of attachment disorders

The human being’s need for attachment is part of his evolutionary heritage. What becomes of this genetically determined attachment need of a newborn is essentially decided in the relationship with the primary caregiver. That person’s sensitivity, her reactions to the infant’s signals appropriate to its developmental phase, essentially determine whether after 12 to 18 months a secure or an insecure form of attachment behavior develops. If the primary caregiver responds to the attachment needs with rejection, or if she responds rather inconsistently, then insecure attachment behavior arises. Prospective longitudinal studies show that this form of attachment behavior, determined early on, affects with quite high consistency how the individual forms relationships in adulthood (Fremmer-Bombik & Grossmann 1993). The passing on of attachment behavior across generations has been proven both in animal experiments (Francis et al. 1999a, Champagne & Meaney 2001) and in human beings (Fonagy et al. 1991, Egeland & Susman-Stillman 1996). Insecure attachment behavior as a form of early emotional neglect is therefore included in the evidence-based list of risk factors of the long-term effects. Secure attachment behavior, on the other hand, can act as a compensatory protective factor to counteract the long-term effects of early stress factors (Farber & Egeland

1987, Wyman et al. 1991, Radke-Yarrow & Brown 1993, Esser et al. 1993, McCarthy & Taylor 1999, Duggal et al. 2001). Early psychosocial stress factors coincide with insecure attachment behavior in adulthood, as was demonstrated in a large-scale American study on a representative sample population (Mickelson et al. 1997). 36% of the evaluated persons with insecure attachment behavior (25% insecure-avoiding, 11% insecure-anxious) showed a significantly higher rate of adverse childhood experiences when asked in adulthood. The association between insecure-disorganized attachment and the development of dissociative symptoms is also well established (Ogava et al. 1997).

Psychobiological effects of early stress experiences

In addition to these behavior-related effects which can contribute to the development of risk behavior (see below), an inadequate response to the need for attachment can also have psychobiological consequences which hinder the individual maturation of the stress response system. Animal experiments demonstrate a close tie between early attachment disorders on the one hand and endocrine reactions affecting the stress management system and physical growth on the other (Meaney et al. 1993, Hofer 1994, 1996). Accordingly, it is obviously of great importance for early development that new stimuli are presented adequate to the developmental stage of the infant or toddler. Even in infants and toddlers it can be observed that secure attachment accompanies better stress response, and insecure attachment correlates with poor stress response. Psychobiological studies have shown that if the newborn's biologically determined attachment need is met with appropriate maternal care and sensitivity (e.g. responsive facial expressions from the mother), then dopaminergic fibres are activated in the brainstem (Schore 1997, 2000, Glaser 2000). This triggers the release of endorphins which make social interaction and social affects a pleasant experience for the infant. Secure attachment behavior thus develops, behavior that protects the brain – particularly the hippocampus and prefrontal orbital cortex– from damage resulting from the release of stress hormones (glucocorticoids and/or norepinephrine/dopamine). Thus, secure attachment leads to an increase in the stress threshold, i.e. better damping of the stress response over the hypothalamic-pituitary-adrenal axis (HPA) and the locus coeruleus-norepinephrine (LC-NE) axis. In animal experiments it has been shown that postnatal care (especially intensive licking) on the part of the primary caregiver can compensate the effects of genetically determined stress vulnerability, stress effects on the embryo with respect to increased anxiety and activity of the HPA axis, as well as decreases in body weight, size and feeding behavior (Valee et al. 1996, 1997, Francis et al. 1999a). The HPA and the LC-NE axis are largely stimulated by the corticotropin-releasing hormone (CRH) which is mainly produced in the paraventricular

nucleus of the hypothalamus, and in other regions of the brain as well. The goal of both systems is to restore the biological homeostasis threatened by biological or psychosocial stress (Chrousos & Gold 1992). Early stress in children quite obviously leads to an increased ACTH reaction to CRH, i.e. to a sensitization of the HPA axis (Kaufman et al. 1997, Heim et al. 1998). Persistent or frequently recurring biological or psychosocial stress leads through an increased glucocorticoid level (DeBellis et al. 1994) over the course of life to damage of the hippocampus (Bremner et al. 1997, Stein et al. 1997) and losses in the declarative memory, i.e. to considerable cognitive deficits (Lupien et al. 1998), or through increased dopamine/ norepinephrine levels, to damage in the region of the prefrontal orbital cortex (Arnsten 1997, 1999, Francis et al. 1999b, Braun et al. 2000) and to sustained dysfunction of the autonomic nervous system or the LC-NE axis (Perry 2001). Risk of this response is particularly high when recurring stress occurs during a vulnerable developmental stage. The resulting dysfunction of the hippocampus has consequences for both the short-term memory and the dynamic-associative linking of experiences (dissociation), as well as for control over the release of cortisol (negative feedback), through which, in a vicious circle, the extent of glucocorticoid-caused damage is heightened (Sapolsky 1996). The abnormal function of the LC-NE system causes the development of multiple physical ailments or somatization (Heim et al. 2000) as well as impairment of cognitive stress management (coping, planning, and organization of actions, inhibition of inappropriate responses) through damage to the region of the prefrontal orbital cortex. This results in ”attention to distraction”, which today is regarded as one of the causes for the development of the attention deficiency-hyperactivity (ADH-) syndrome/disorder (Arnsten 2000).

Thus, early childhood stress factors such as early attachment disorders and other effects of lacking sensitivity to the needs of the child on the part of his primary care giver, as well as maltreatment and neglect, lead to increased stress vulnerability in which increased CRH release plays a central biological role (Heim & Nemeroff 1999, 2001). This release triggers a cascade of biological processes through the activation of the HPA axis and the LC-NE axis which, within the framework of feedback mechanisms, leads to damage in the hippocampus and prefrontal cortex and ultimately to a life-long dysfunction of the stress response system (McEwen 1999).

Cognitive dysfunction and immature defense or coping strategies

On the behavioral level, emotional and cognitive impairments emerge as a consequence of early stressors. The inability to modulate emotions contributes to a series of behaviors that can be understood as attempts at self-regulation (van der Kolk & Fisler 1994). Such behaviors

include substance abuse, self-destructive behavior, disturbed eating patterns, and aggression towards others. Traumatized children often lack the ability to express specific affects in a differentiated manner. Because they are limited in the range and flexibility of their reactions, they often act out their affects. The fact that the maturity of the available defense mechanisms influences the vulnerability to mental and physical illnesses is regarded as firmly established, particularly since the results of the 45-year prospective Grantstudy (Vaillant 1976, Vaillant et al. 1986). The defense and illness coping strategies of patients who were traumatized during childhood are generally immature. This means that these strategies are above all dominated by self-negating thoughts, projections or catastrophizing, generalizations, and ”black or white thinking” (Schmidt et al. 1993, Elton et al. 1994, Scarinci et al. 1994, Nickel & Egle 2001, Fearon & Hotopf 2001).

Risk behavior

Immature strategies for dealing with conflicts and coping with illness further increase the vulnerability to stress, which is already heightened, when confronted with psychosocial stress situations and internal conflicts during adulthood. Substance abuse (alcohol, drugs, nicotine), overweight (Body Mass Index > 35) resulting from a disturbed eating behavior, as well as depression, suicide attempts, a lack of physical leisure activities, or frequent change of sexual partners belong to risk health behavior which essentially determines the link of early stress factors to long-term health effects in adulthood (Felitti et al. 1998). Risk behavior hinders age-specific developmental steps in adolescents and young adults. Missing any of these steps can in turn be an additional risk factor. Academic and athletic achievement, affiliation to a peer group (in school, a religious institution, or a sport club), close friendships (Pawlby et al. 1997), and an emotionally sound partnership (Werner 1997) all contribute substantially to a stable feeling of self-esteem, and provide compensatory protection against adverse factors. On the other hand, physical microsomia resulting from family stress factors (Montgomery et al. 1997), failure to achieve, the experience of being an outsider, and early pregnancy all play a role in the development of increased mental and physical vulnerability in adulthood (Werner 1989, 1996, Romans et al. 1995).

Early stress and later violent behavior

Cluster A and B personality disorders and symptoms of a paranoid, narcissistic and passive-aggressive personality bring along an increased likelihood of violence (Johnson et al. 2000). Vulnerability to one of these personality disorders is significantly increased by sexual or physical abuse as well as emotional or physical neglect during childhood, as was shown in a

prospective study on a representative random sample (n=793) in New York (Johnson et al. 1999). A further adverse childhood experience that increases vulnerability to a personality disorder later in life was regular ”verbal abuse” (Johnson et al. 2001). In addition, these studies were able to differentiate between various personality disorders (PD) resulting from the effects of different adverse childhood experiences: emotional neglect led to an avoiding PD and symptom formation of a paranoid PD, while physical neglect was associated with an increased risk of a schizoid PD. Both emotional and physical neglect also increase the risk of cluster A symptom formation. A lack of supervision or setting limits make the later development of a passive-aggressive or cluster B personality disorder significantly more likely. More than one hour of daily television watching additionally increases the later potential for violence (Johnson et al. 2002). In the meantime, various studies have been able to firmly establish that a combination of biological factors (male sex, minor physical anomalies, perinatal complications), an early attachment disorder, hyperactive behavior and family risk factors also predispose an individual to later development of violent behavior and criminality (Raine et al. 1994, 1996, Arseneault et al. 2000). Thus, emotional neglect and family violence can ultimately lead to violent behavior towards the environment later on in life (Widom 1989; Fergusson & Horwood 1998; Marshall & Cooke 1999). Cierpka (1999) and Ratzke and Cierpka (2000) have summarized the multi-factorial combined effects of various biological, inter-individual, family, and social factors in a family risk model from which starting points for early prevention can be derived.

Bio-psychopathological model of vulnerability

Figure 2 summarizes in a ”bio-psychopathological model of vulnerability” the combined effects of biological and psychosocial factors with regard to long-term health effects in adulthood. It also makes clear that the discipline-related distinction often drawn today between biological-genetically (nature) and psychosocially (nurture) origins of mental and physical diseases is based on an antiquated scientific understanding. This is urgently in need of amendment for the future conception of scientific studies as well as for clinical work (Meaney 2001). The model illustrates what broad spectrum of mental disorders and physical diseases ultimately can develop on this basis (see Table 2). The results of the National Comorbidity Survey carried out on a representative random sample in the USA (Kessler et al. 1997, Molnar et al. 2001) and the Adverse Childhood Experience (ACE) Study performed in a Californian prevention center (Felitti et al. 1998, Anda et al. 1999) with retrospective surveys of samples of nearly 6000 and 10,000 subjects, respectively, demonstrate highly significant correlations. In his paper in this volume, Felitti (2002) deduces from these results

that some of the diseases resulting from mental processes (depression, suicide) or physical processes (stroke, coronary heart disease, diabetes, hepatitis, obstructive lung diseases) lead to an increase in mortality at a relatively young age. His postulation certainly sounds plausible, even if perhaps the final scientific proof has yet to be given within the framework of study specially designed for the diseases mentioned.

Table 2: Mental and Physical Illnesses as Potential Consequences of Early Stress Experiences

- Depressive disorders
- Anxiety disorders
- Eating disorders (above all bulimia)
- Somatoform disorders (above all pain, fibromyalgia)
- Post-traumatic risk disorders
- Dissociative and conversion disorders
- Severe personality disorders
- Addiction diseases
- Sexual dysfunctions

- Cardio-vascular diseases
- Stroke
- Hepatitis
- Chronic-obstructive lung disease
- Type 2 diabetes
- Osteoporosis

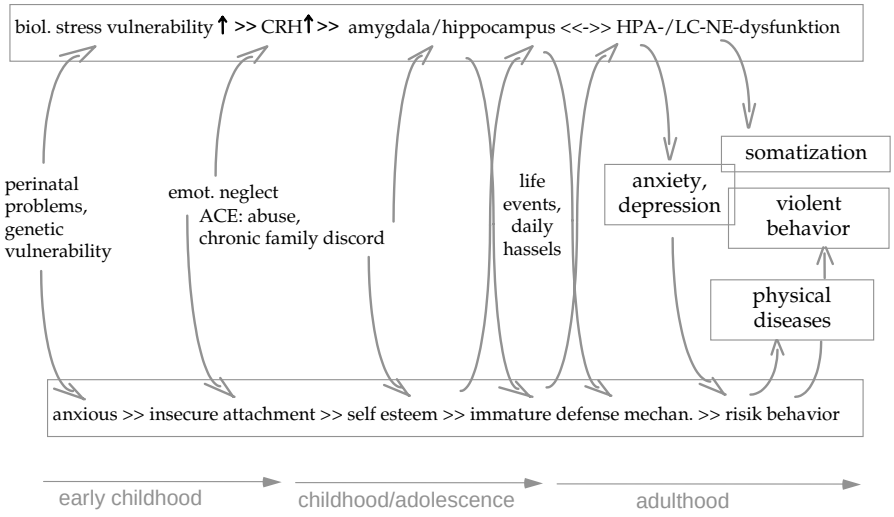

Fig. 2: Bio-psychopathological model of vulnerability

Methodological standards and traps

Although widely distributed, retrospective case control studies on the long-term effects of childhood risk factors are met by an equally wide-spread skepticism (i.e Yarrow et al. 1970, Bower 1981). The advantages of prospective longitudinal studies appear obvious at first (Rutter 1988): The researcher sets the time point and type of survey, and it is not the subject under investigation who has to remember a specific period in time. In addition, judgements from third parties, such as parents or teachers, can be collected. A further advantage lies in a broad definition of possible consequences. Even those individuals who were subjected to childhood risk factors yet show no signs of disorders in adulthood are adequately covered in longitudinal studies. Particularly in comparison with case control studies, longitudinal studies offer a better possibility to estimate the probability with which certain childhood risk factors lead to certain disorders. Often only a minority of the persons exposed to a stress factor later develops a specific disorder.

Yet these advantages are opposed by a series of disadvantages. Apart from the great effort involved and long duration of longitudinal studies, especially in young children there is danger that questions are not understood and answered as adults would (Ceci & Bruck 1993). For this reason, it is often the case that only behavior observations or indirect assessments via caregivers can be drawn up in these types of studies. For the entire range of criminally prosecuted offences, we must realize that a prospective study will only ever be able to survey a part of the adverse childhood experiences. Children and adolescents will only partially assent to various questions, even if the corresponding risk factors are present. Questioning caregivers, for instance parents, siblings, or teachers, in many cases will overlook adversities. Exemplary for this phenomenon is sexual abuse: Caregivers do not necessarily know about the abuse or may even have been the perpetrators themselves. The assurance of anonymity also could not be maintained in these cases; rather, it would be necessary to intervene immediately and protect the children. Amaya-Jackson et al. (2000) present a discourse on the problem of assuring confidentiality of information in a scientific investigation versus the necessity of ending continued sexual abuse. They show different practical solutions used in various longitudinal studies from the 1990’s that range from a large degree of restraint to an emergency hotline for children. Even falling back on objective data does not help much as only about 10% of abuse cases are reported to the authorities (Finkelhor 1994; Finkelhor & Dziuba-Leatherman 1994). The same holds true for other childhood risk factors, particularly when it comes to criminally prosecuted offences or socially undesirable events.

What are the specific risks involved in implementing the retrospective method? For one, test subjects can consciously decide to conceal events or may alter them when reporting. This applies especially to socially undesirable events, such as physical maltreatment, but also to

the separation of the parents (Cherlin & Horiuchi 1980). On the other hand, certain events, such as abuse, are sometimes concealed either because the interviewee wants to avoid the emotional distress caused by reporting, he or she may want to protect the perpetrator, or may not have sufficient trust in the interviewer (Femina et al. 1990). From experiment memory research we know that our memory does not work like a tape recording whose sound becomes muffled over time, until it completely fades away. Only essential core points of our experiences are stored in our memories and it is around these points that the events are reconstructed when we remember (Schacter 1996, 2001). This presents us with various possible problems with regard to the recollection of events that happened long ago. Many researchers suspect that memories are dependent on a person's mood. Bower (1981) was able to verify this in free recall under experimentally induced hypnosis. In contrast, Gerlsma et al. (1993, 1994) investigated mood-dependence in accounts of parental rearing practices by analyzing the influence of anxiety and depression measured by a questionnaire on their recollections. Their results showed depression to have no effect on their accounts of rearing practices. With regard to anxiety, the results showed that those individuals who at that moment reported greater anxiety stated that they had had an emotionally warmer relationship with their parents. However, the observed effect size was low. None of the cases showed more than 6% of the variance of a scale to the rearing practices described. Gerlsma et al. interpret their results to mean that an influence of the momentary emotions on the reported parental rearing practices does indeed exist, although its extent is marginal, and thus from a scientific standpoint it can almost be ignored. Furthermore, there is a risk that healthy and sick individuals differ in their memories of their childhood. An investigation by Maugham et al. (1995) shows an alarming finding in this connection. The authors explored the relationship between prospective and retrospective surveys of parental hostility. Among those individuals whose parents were categorized as rejecting over the course of the study, the rejection on the part of the parents was significantly more frequently reported by high-risk adults than low-risk ones in the retrospective survey. In contrast, false positive assignments rarely occurred. A similar result was found in answer to the question as to whether or not the parents suffered from a mental illness, since mental illnesses are seldom recognized by laymen (Heun et al. 1996; Hardt in prep.). Yet the extent of this selective distortion of memories appears altogether to be only moderate, though until today it is not yet clear which factors are at work here (Hardt & Rutter submitted).

A multitude of standardized instruments exists for surveying manifest adverse childhood experiences and the parent-child relationship (Melchert 1998; O'Leary 1999). Some comprehensive interviews are, for instance, the Childhood Experiences of Care and Abuse

(CECA, Bifulco et al. 1994) and, in German, the Mainz Structured Biographical Anamnesis (MSBA, Egle 1992, 1993). Various questionnaires exist on individual aspects of childhood risk factors. Physical maltreatment, for instance, can be surveyed using the Conflict Tactics Questionnaire (CTQ, Straus 1979; German: Wetzels 1997). Probably the most widespread questionnaire for surveying the parent-child relationship is the Parental Bonding Instrument (PBI, Parker 1979, 1984), also available in German in a modified form (Parker et al. 1997; Rumpold et al. 2002). It contains the scales care, overprotection, and abuse. A further instrument implemented in many languages is the Swedish ”Egna Minnen Beträffende Uppfostan” (EMBU, Perris et al. 1980). It is available in German in a validated and normed form (Questionnaire on Remembered Parental Rearing Practices, FEE, Schumacher et al. 1999) and contains the dimensions rejection and punishment, emotional warmth, and overprotection. One comprehensive instrument is the Kindheitsfragebogen (KFB, Childhood Questionnaire, Hardt et al. submitted). It contains eight dimensions for each parent (perceived love, punishment, trivialization of the punishing behavior, parents as a role model, role reversal, ambition, control, and sibling rivalry) that closely follow the evaluation of the Adult Attachment Interview (Main & Goldwyn 1984).

In summary we can statethat recollections of traumatic experiences can be surveyed with relative validity regarding the question as to whether an event took place or not. Details, in contrast, frequently are recalled in altered form (Brewin, 1993, Banyard & Williams 1996). Epidemiological investigations tend to underestimate the real prevalences. Concrete questions on events deliver more valid results than global assessments (see Hardt & Rutter submitted).

Summary of the current status of research and perspectives for the future

Within the framework of a scientific expert symposium (at Kloster Eberbach 20 and 21 Oct 2001), the current status of research and research perspectives was summarized in a joint concluding statement as follows:

Scientific evidence has emerged from various specialized areas indicating that it is not the individual risk factors alone but rather their sum that exerts a decisive influence on later life. Thus, sexual abuse, for instance, has been given considerable attention in research and in the media, while other factors that can have an equal influence have been examined to a much lesser extent. This is true of emotional neglect, for example. It is difficult to operationalize, which possibly is why it has received so little consideration in research. However, this difficulty should hinder future investigation of this factor. Styles of attachment can be regarded as an indicator of early neglect. Additionally, this research would integrate results on

the protective function of attachment. However, attachment styles should always be investigated in connection with manifest stress or protective factors. A one-sided concentration on attachment, as has been the case for previous concentration on sexual abuse, is to be avoided. Models exist that integrate different risk and protective factors. They should be validated and modified as needed. In doing so, the developmental age during which these factors take effect should be given greater consideration in the future. In addition to the attention given to the interaction of various influencing factors, researchers must increasingly focus on the different consequences of risk factors. Apart from mental, psychosomatic, and somatic illnesses, these consequences also include factors such as unemployment, academic achievement, antisocial behavior, criminality, and others. Resilience as a form of interaction between risk factors and protective factors appears to be of particular importance: We know today that certain factors accompany better coping with stress, but little is known about the exact mechanisms at work.

On the basis of these points, we can derive the following research strategies: The era of the small-scale case control studies, maybe with the exception of some more specific questions, is over. Large, multi-factorial investigations are necessary. If possible, these studies should take place more frequently on a joint international scale, as there are obviously significant differences in terms of prevalence for individual factors in various countries. Various standardized procedures are available that are also suitable for the valid retrospective survey of adverse childhood experiences. Psychobiological research should be increasingly integrated. Recently, essential findings and models have emerged from this field, and more is expected. All basic research aims towards better prevention and therapy. Cooperation with clinical disciplines should be strengthened in two regards: For one, it is necessary to integrate the discoveries of research into therapeutic and prevention programs. For another, these programs have to be evaluated in the future. Yet as long as we lack more exact data on the frequency of psychosocial risk factors in children in Germany, and as long as only limited empirically founded knowledge is available on the meaning of compensatory protective factors for clinically meaningful long-term consequences, it will probably be difficult to develop specific broad-scale preventative measures. The numbers on prevalence available for the USA do not appear to be applicable to western Europe due to the distinctly different societal values placed on the family (see Amato 2001).

The connections outlined here appear vital, particularly in light of the current political objectives of nearly all relevant German political parties: From an economic standpoint it may appear sensible to re-integrate mothers with a high level of professional training back into the

work force as quickly as possible. More daycare facilities would therefore need to be created for their infants and toddlers even before the age of pre-school. However, the early separation from the primary caregiver, in such arrangements increases the probability of long-term effects, both on a biological level (stress response system) and on a behavioral level (attachment disorders). Consequently, if we pay heed to the current state of scientific knowledge, then it must be assumed that the implementation of such a political agenda could have considerable consequences for the mental and physical health of the children and long-term effects regarding their health in adulthood. Based on scientific knowledge, politicians would have to think about other solutions, such as greater flexibility in the work hours of parents. With the current contrary political objectives, the question should be raised as to whether the committees responsible for allocating public funding for research regard more research in this field as necessary. Nonetheless, the continued exploration of the connections outlined here is one of the central tasks of psychosomatic medicine in the future, with the goal of defining and operationalizing preventative approaches.

We owe great thanks to the participants of the Expert Symposium at Kloster Eberbach/Rheingau on October 20 and 21, 2001 directly following the 1st International Conference ”Adverse Childhood Experiences and Adult Health” in Mainz: H. Aro, Helsinki/SF; P. Amato, Pennsylvania/USA; F. Becker-Stoll, Munich/D; K. Braun, Magdeburg/D; M. Cierpka, Heidelberg/D; A. Engfer, Paderborn/D; V. Felitti, San Diego/USA; D. Fergusson, Christchurch/NZ; M. Franz, Düsseldorf/D; U. Gast, Hannover/D; T. Harris, London/GB; B. van Houdenhove, Leuven/B; J. Johnson, New York/USA; P. Joraschky, Dresden/D; P. Mullen, Melbourne/AUS; A. Lampe, Innsbruck/A; F. Lösel, Erlangen/D; F. Resch, Heidelberg/D; M. Richards, London/GB; Sir M. Rutter, London/GB; C. Scheidt, Freiburg/D, U. Schnyder, Zurich/CH; I. Schoon, London/GB; G. Schüssler, Innsbruck/A; T. Sivik, Göteborg/S; W. Tress, Düsseldorf/D.

References

Abraham, K. (1924): Manic-depressive states and the pregenital level of libido. London.

Amato, P. R. (2001): Children of divorce in the 1990s: an update of the Amato and Keith (1991) meta-analysis. J Fam Psychol 15, 355-70.

Amaya-Jackson, L., Socolar, R. R. S., Hunter, W., Runyan, D. K., Colinders, R. (2000): Directly questioning children and adolescents about maltreatment. Interpersonal Violence 15, 725-759.

Anda, R. F., Croft, J. B., Felitti, V. J., Nordenberg, D., Giles, W. H., Williamson, D. F., Giovino, G. A. (1999): Adverse childhood experiences and smoking during adolescence and adulthood. JAMA 282, 1652-8.

Ariès, P. (1975): Geschichte der Kindheit. München.

Arnsten, A. F. (1997): Catecholamine regulation of the prefrontal cortex. J Psychopharmacol 11, 151-62.

Arnsten, A. F. (1999): Development of the cerebral cortex: XIV. Stress impairs prefrontal cortical function. J Am Acad Child Adolesc Psychiatry 38, 220-2.

Arnsten, A. F. (2000): Genetics of childhood disorders: XVIII. ADHD, Part. 2: Norepinephrine has a critical modulatory influence on prefrontal cortical function. J Am Acad Child Adolesc Psychiatry 39, 1201-3.

Arseneault, L., Tremblay, R. E., Boulerice, B., Seguin, J. R., Saucier, J. F. (2000): Minor physical anomalies and family adversity as risk factors for violent delinquency in adolescence. Am J Psychiatry 157, 917-23.

Banyard, V. L., Williams, L. M. (1996): Characteristics of child sexual abuse as correlates of women´s adjustment: a prospective study. J Marriage Fam 58, 853-865.

Bender, D., Lösel, F. (2000): Risiko- und Schutzfaktoren in der Genese und der Bewältigung von Misshandlung und Vernachlässigung. In: Egle, U.T., Hoffmann, S.O., Joraschky, P. (eds.) Sexueller Missbrauch, Misshandlung, Vernachlässigung. pp. 40-58. Stuttgart.

Bifulco, A., Brown, G. W., Harris, T. O. (1994): Childhood experience of care and abuse (CECA): a retrospective interview measure. J Child Psychol Psychiatry 35, 1419-35.

Bower, G. H. (1981): Mood and memory. American Psychologist 36, 129-148.

Bowlby, J. (1951): Mental care and mental health. WHO, Genf.

Braun, K., Lange, E., Metzger, M., Poeggel, G. (2000): Maternal separation followed by early social deprivation affects the development of monoaminergic fiber systems in the medial prefrontal cortex of Octodon degus. Neuroscience 95, 309-18.

Bremner, J. D., Randall, P., Vermetten, E., Staib, L., Bronen, R. A., Mazure, C., Capelli, S., McCarthy, G., Innis, R. B., Charney, D. S. (1997): Magnetic resonance imaging-

based measurement of hippocampal volume in posttraumatic stress disorder related to childhood physical and sexual abuse--a preliminary report. Biol Psychiatry 41, 23-32.

Brewin, C. R., Andrews, B., Gotlib, I. C. (1993): Psychopathology and early experience: a reappraisal of retrospective reports. Psychol Bull 113, 82-98.

Ceci, S. J., Bruck, M. (1993): Suggestibility of the child witness: a historical review and synthesis. Psychol Bull 113, 403-39.

Champagne, F., Meaney, M. J. (2001): Like mother, like daughter: evidence for non-genomic transmission of parental behavior and stress responsivity. Prog Brain Res 133, 287-302.

Cherlin, A. J., Horiuchi, S. (1980): Retrospective reports of family structure. Sociol Meth & Res 8, 454-469.

Chrousos, G. P., Gold, P. W. (1992): The concepts of stress and stress system disorders. Overview of physical and behavioral homeostasis. JAMA 267, 1244-52.

Cierpka, M. (1999): Kinder mit aggressivem Verhalten. Göttingen.

De Bellis, M. D., Chrousos, G. P., Dorn, L. D., Burke, L., Helmers, K., Kling, M. A., Trickett, P. K., Putnam, F. W. (1994): Hypothalamic-pituitary-adrenal axis dysregulation in sexually abused girls. J Clin Endocrinol Metab 78, 249-55.

De Bellis, M. D., Baum, A. S., Birmaher, B., Keshavan, M. S., Eccard, C. H., Boring, A. M., Jenkins, F. J., Ryan, N. D. (1999): A.E. Bennett Research Award. Developmental traumatology. Part I: Biological stress systems. Biol Psychiatry 45, 1259-70.

De Bellis, M. D., Keshavan, M. S., Clark, D. B., Casey, B. J., Giedd, J. N., Boring, A. M., Frustaci, K., Ryan, N. D. (1999): A.E. Bennett Research Award. Developmental traumatology. Part II: Brain development. Biol Psychiatry 45, 1271-84.

Duggal, S., Carlson, E. A., Sroufe, L. A., Egeland, B. (2001): Depressive symptomatology in childhood and adolescence. Dev Psychopathol 13, 143-64.

Egeland, B., Susman-Stillman, A. (1996): Dissociation as a mediator of child abuse across generations. Child Abuse Negl 20, 1123-32.

Egle, U.T. (1992): Die Strukturierte Biographische Anamnese für Schmerzpatienten (SBAS). Psychother Psychosom med Psychol Disk J, 3, 4.

Egle, U.T. (1993): Strukturierte Biographische Anamnese für Schmerzpatienten (SBAS-IV). In: Egle, U.T., Hoffmann, S.O. (eds.): Der Schmerzkranke, pp. 617-59. Stuttgart.

Egle, U.T., Hoffmann, S.O. (2000): Pathogene und protektive Entwicklungsfaktoren in Kindheit und Jugend. In: Egle, U.T., Hoffmann, S.O., Joraschky, P. (eds.): Sexueller Missbrauch, Misshandlung, Vernachlässigung, pp. 23-39. Stuttgart.

Egle, U.T., Hardt, J., Franz, M., Hoffmann, S.O. (2002): Belastungen in der Kindheit und Gesundheit im Erwachsenenalter. Möglichkeit der Prävention in der Psychosomatischen Medizin. Psychotherapeut 47, 124-27.

Elton, N. H., Hanna, M. M., Treasure, J. (1994): Coping with chronic pain. Some patients suffer more. Br J Psychiatry 165, 802-7.

Engfer, A. (2000) Gewalt gegen Kinder in der Familie. In: Egle, U.T., Hoffmann, S.O., Joraschky, P. (eds.): Sexueller Missbrauch, Misshandlung, Vernachlässigung, pp. 23-39. Stuttgart.

Esser, G., Dinter, R., Jörg, M., Rose, F., Villalba, P., Laucht, M., Schmidt, M. (1993): Bedeutung und Determinanten der frühen Mutter-Kind-Beziehung. Z psychosom Med Psychoanal 39, 246-64.

Farber, E. A., Egeland, B. (1987): Invulnerability among abused and neglected children. In: Anthony, E. J., Cohler, B. J. (eds.): The invulnerable child, pp. 253-88. New York, London.

Fearon, P., Hotopf, M. (2001): Relation between headache in childhood and physical and psychiatric symptoms in adulthood: national birth cohort study. BMJ 322, 1145.

Felitti, V. J., Anda, R. F., Nordenberg, D., Williamson, D. F., Spitz, A. M., Edwards, V., Koss, M. P., Marks, J. S. (1998): Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults. The Adverse Childhood Experience (ACE) Study. Am J Prev Med 14, 245-58.

Felitti, V.J. (2002): Z Psychosom Med Psychother 48, 359-69.

Femina, D. D., Yeager, C. A., Lewis, D. O. (1990): Child abuse: adolescent records vs. adult recall. Child Abuse Negl 14, 227-31.

Fergusson, D. M., Horwood, L. J. (1998): Exposure to interparental violence in childhood and psychosocial adjustment in young adulthood. Child Abuse Negl 22(5), 339-357.

Fergusson, D. M., Lynskey, M. T. (1995): Suicide attempts and suicidal ideation in a birth cohort of 16-year-old New Zealanders. J Am Acad Child Adolesc Psychiatry 34, 1308-17.

Fergusson, D. M., Lynskey, M. T. (1996): Adolescent resiliency to family adversity. J Child Psychol Psychiatry 37, 281-92.

Finkelhor, D. (1994): The international epidemiology of child sexual abuse. Child Abuse Negl 18, 409-17.

Finkelhor, D., Dziuba-Leatherman, J. (1994): Children as victims of violence: a national survey. Pediatrics 94, 413-20.

Fonagy, P., Steele, H., Steele, M. (1991): Maternal representations of attachment during pregnancy predict the organization of infant-mother attachment at one year of age. Child Dev 62, 891-905.

Francis, D. D., Champagne, F. A., Liu, D., Meaney, M. J. (1999a): Maternal care, gene expression, and the development of individual differences in stress reactivity. Ann N Y Acad Sci 896, 66-84.

Francis, D. D., Caldji, C., Champagne, F., Plotsky, P. M., Meaney, M. J. (1999b): The role of corticotropin-releasing factor--norepinephrine systems in mediating the effects of early experience on the development of behavioral and endocrine responses to stress. Biol Psychiatry 46, 1153-66.

Franz, M., Lieberz, K., Schmitz, N., Schepank, H. (1999): Wenn der Vater fehlt. Epidemiologische Befunde zur Bedeutung früher Abwesenheit für die psychische Gesundheit im späteren Leben. Z Psychosom Med Psychother 45, 113-127.

Franz, M. (2001): Aufwachsen mit nur einem Elternteil - Was bewirkt das Fehlen des Vaters? Vortrag bei der 1. Internationalen Konferenz "Belastungen in der Kindheit und Gesundheit im Erwachsenenalter", Mainz.

Fremmer-Bombik, E., Grossmann, K. E. (1993): Über die lebenslange Bedeutung früher Bindungserfahrungen. Petzold, H. (eds.): Frühe Schädigungen - späte Folgen? Psychotherapie und Babyforschung, pp. 83-110. Paderborn.

Freud, S. (1917): Trauer und Melancholie. Frankfurt/M.

Gerlsma, C., Das, J., Emmelkamp, P. M. (1993): Depressed patients' parental representations: stability across changes in depressed mood and specificity across diagnoses. J Affect Disord 27, 173-81.

Gerlsma, C., Kramer, J. J., Scholing, A., Emmelkamp, P. M. (1994): The influence of mood on memories of parental rearing practices. Br J Clin Psychol 33, 159-72.

Glaser, D. (2000): Child abuse and neglect and the brain--a review. J Child Psychol Psychiatry 41, 97-116.

Hardt, J. (in prep): Reliability and validity of the family history method: a meta- analysis.

Hardt, J., Rutter, M. (submitted): Validity of adult retrospective reports of adverse childhood experiences: review of the evidence.

Hardt, J., Egle, U. T., Engfer, A. (submitted): Der Kindheitsfragebogen (KFB): Ein Instrument zur Beschreibung der Kindheitsbeziehungen zu den Eltern.

Heim, C., Nemeroff, C. B. (1999): The impact of early adverse experiences on brain systems involved in the pathophysiology of anxiety and affective disorders. Biol Psychiatry 46, 1509-22.

Heim, C., Newport, D. J., Heit, S., Graham, Y. P., Wilcox, M., Bonsall, R., Miller, A. H., Nemeroff, C. B. (2000): Pituitary-adrenal and autonomic responses to stress in women after sexual and physical abuse in childhood. JAMA 284, 592-7.

Heim, C., Nemeroff, C. B. (2001): The role of childhood trauma in the neurobiology of mood and anxiety disorders: preclinical and clinical studies. Biol Psychiatry 49, 1023-39.

Heun, R., Hardt, J., Burkart, M., Maier, W. (1996): Validity of the family history method in relatives of gerontopsychiatric patients. Psychiatry Res 62, 227-38.

Hofer, M. A. (1994): Hidden regulators in attachment, separation, and loss. Monogr Soc Res Child Dev 59, 192-207. Hofer, M. A. (1996): On the nature and consequences of early loss. Psychosom Med 58, 570-81.

Johnson, J. G., Cohen, P., Brown, J., Smailes, E. M., Bernstein, D. P. (1999): Childhood maltreatment increases risk for personality disorders during early adulthood. Arch Gen Psychiatry 56, 600-6.

Johnson, J. G., Smailes, E. M., Cohen, P., Brown, J., Bernstein, D. P. (2000): Associations between four types of childhood neglect and personality disorder symptoms during adolescence and early adulthood: findings of a community-based longitudinal study. J Personal Disord 14, 171-87.

Johnson, J. G., Cohen, P., Smailes, E. M., Skodol, A. E., Brown, J., Oldham, J. M. (2001): Childhood verbal abuse and risk for personality disorders during adolescence and early adulthood. Compr Psychiatry 42, 16-23.

Johnson, J. G., Cohen, P., Smailes, E. M., Kasen, S., Brook, J. S. (2002): Television viewing and aggressive behavior during adolescence and adulthood. Science 295, 2468-71.

Kaufman, J., Birmaher, B., Perel, J., Dahl, R. E., Moreci, P., Nelson, B., Wells, W., Ryan, N. D. (1997): The corticotropin-releasing hormone challenge in depressed abused, depressed nonabused, and normal control children. Biol Psychiatry 42, 669-79.

Kendler, K. S., Neale, M. C., Kessler, R. C., Heath, A. C., Eaves, L. J. (1992): Childhood parental loss and adult psychopathology in women. A twin study perspective. Arch Gen Psychiatry 49, 109-16.

Kessler, R. C., Davis, C. G., Kendler, K. S. (1997): Childhood adversity and adult psychiatric disorder in the US National Comorbidity Survey. Psychol Med 27, 1101-19.

Lupien, S. J., de Leon, M., de Santi, S., Convit, A., Tarshish, C., Nair, N. P., Thakur, M., McEwen, B. S., Hauger, R. L., Meaney, M. J. (1998): Cortisol levels during human aging predict hippocampal atrophy and memory deficits. Nat Neurosci 1, 69-73.

Main, M., Goldwyn, R. (1984): Predicting rejection of her infant from mother's representation of her own experience: implications for the abused-abusing intergenerational cycle. Child Abuse Negl 8, 203-17.

Marshall, L. A.,Cooke, D. J. (1999). The childhood experiences of psychopaths: a retrospective study of familial and societal factors. J Personal Disord 13, 211-225.

Maughan, B., Pickles, A., Quinton, D. (1995): Parental hostility, childhood behavior, and adult social functioning. In: McCord, J. (ed.): Coercion and punishment in long term perspectives, pp. 34-58. New York.

McCarty, G., Taylor, A. (1999): Avoidant/ambivalent attachment styles as a mediator between abusive childhood experiences and adult relationship difficulties. J Child Psychol Psychiatry 40, 465-77.

McEwen, B. S. (1999): Stress and the aging hippocampus. Front Neuroendocrinol 20, 49-70.

Meaney, M. J. (2001): Nature, nurture, and the disunity of knowledge. Ann N Y Acad Sci 935, 50-61.

Meaney, M. J., Bhatnagar, S., Laroque, S., McCormick, C., Shanks, N., Sharma, S., Smythe, J., Viau, V., Plotsky, P.M. (1993): Individual differences in the hypothalamic-pituitary-adrenal stress responce and the hypothalamic CRF system. Ann N Y Acad Sci 29; 697, 70-85.

Melchert, T. P. (1998): A review of instruments for assessing family history. Clin Psychol Rev 18,

Mickelson, K. D., Kessler, R. C., Shaver, P. R. (1997): Adult attachment in a nationally representative sample. J Pers Soc Psychol 73, 1092-106.

Molnar, B. E., Buka, S. L., Kessler, R. C. (2001): Child sexual abuse and subsequent psychopathology: results from the National Comorbidity Survey. Am J Public Health 91, 753-60.

Montgomery, S. M., Bartley, M. J., Wilkinson, R. G. (1997): Family conflict and slow growth. Arch Dis Child 77, 326-30.

Mullen, P. E., Martin, J. L., Anderson, J. C., Romans, S. E., Herbison, G. P. (1993): Childhood sexual abuse and mental health in adult life. Br J Psychiatry 163, 721-32.

Mullen, P. E., Martin, J. L., Anderson, J. C., Romans, S. E., Herbison, G. P. (1996): The long-term impact of the physical, emotional, and sexual abuse of children: a community study. Child Abuse Negl 20, 7-21.

Nickel, R., Egle, U. T. (2001): Coping with conflict as pathogenetic link between psychosocial adversities in childhood and psychic disorders in adulthood. Z Psychosom Med Psychother 47, 332-47.

Ogava, J.R., Sroufe, L.A., Weinfield, N.S., Carlson, E.A., Egeland, B. (1997): Development and the fragmented self: longitudinal study of dissociative symptomatology in a nonclinical sample. Dev Psychopathol 9, 855-79.

O'Leary, K. D. (1999): Psychological abuse: a variable deserving critical attention in domestic violence. Violence Vict 14, 3-23.

Parker, G. (1979): Parental characteristics in relation to depressive disorders. Br J Psychiatry 134, 138-47.

Parker, G. (1984): The measurement of pathogenic parental style and its relevance to psychiatric disorder. Soc Psychiatry 19, 75-81.

Parker, G., Roussos, J., Hadzi-Pavlovic, D., Mitchell, P., Wilhelm, K., Austin, M. P. (1997): The development of a refined measure of dysfunctional parenting and assessment of its relevance in patients with affective disorders. Psychol Med 27, 1193-203.

Pawlby, S. J., Mills, A., Taylor, A., Quinton, D. (1997): Adolescent friendships mediating childhood adversity and adult outcome. J Adolesc 20, 633-44.

Perris, C., Jacobsson, L., Lindström, H., von Knorring, L., Perris, H. (1980): Development of a new inventory for assessing memories of parental rearing behavior. Acta Psychiatr Scand 61, 265-274.

Perry, B. D. (2001): The neurodevelopmental impact of violence in childhood. In: Schetky, D., Benedek, E. (eds.): Textbook of child and adolescent forensic psychiatry, pp. 221-238. Washington, D.C.

Radke-Yarrow, M., Brown E. (1993): Resilience and vulnerability in children of multiple-risk families. Develop Psychopathol 5, 581-92.

Raine, A., Brennan, P., Mednick, S. A. (1994): Birth complication combined with early maternal rejection at age 1 year predispose to violent crime at age 18 years. Arch Gen Psychiatry 51, 984-988.

Raine, A., Brennan, P., Mednick, B., Mednick, S. A. (1996): High rates of violence, crime, academic problems, and behavioral problems in males with both early neuromotor deficits and unstable family environments. Arch Gen Psychiatry 53, 544-549.

Ratzke, K., Cierpka, M. (2000): Familien von Kindern mit aggressiven Verhaltensweisen. In: Egle, U.T., Hoffmann, S.O., Joraschky, P. (eds.) Sexueller Missbrauch, Misshandlung, Vernachlässigung. pp. 99-114. Stuttgart.

Romans, S. E., Martin, J. L., Anderson, J. C., O'Shea, M. L., Mullen, P. E. (1995): Factors that mediate between child sexual abuse and adult psychological outcome. Psychol Med 25, 127-42.

Rumpold, G., Doering, S., Höfer, S., Schüßler, G. (2002): Der Fragebogen dysfunktionaler elterlicher Beziehungsstile (FDEB). Z Psychosom Med Psychother 48, 59-74.

Rutter, M. (1988): Longitudinal data in the study of causal processes: some uses and some pitfalls. In: Rutter, M. (ed.): Studies of psychosocial risks: the power of longitudinal data, pp. 1-28. Worcester.

Rutter, M. (1994): Psychiatric genetics: research challenges and pathways forward. Am J Med Genet 54, 185-98.

Sapolsky, R. M. (1996): Why stress is bad for your brain. Science 273, 749-50.

Scarinci, I. C., McDonald-Haile, J., Bradley, L. A., Richter, J. E. (1994): Altered pain perception and psychosocial features among women with gastrointestinal disorders and history of abuse: a preliminary model. Am J Med 97, 108-18.

Schacter, D. L. (1996): Searching for memory: The brain, the mind, and the past. New York.

Schacter, D. L. (2001): The seven sins of memory: how the mind forgets and remembers. Boston. Schmidt, U., Slone, G., Tiller, J., Treasure, J. (1993): Childhood adversity

and adult defence style in eating disorder patients a controlled study. Br J Med Psychol 66, 353-62.

Schoon, I., Montgomery, S. M. (1997): Zum Zusammenhang von frühkindlicher Lebenserfahrung und Depression im Erwachsenenalter. Z Psychosom Med Psychother 43, 319-333.

Schore, A. N. (1997): Early organization of the nonlinear right brain and development of a predisposition to psychiatric disorders. Dev Psychopathol 9, 595-631.

Schore, A. N. (2000): Attachment and the regulation of the right brain. Attach Hum Dev 2, 23-47.

Schumacher, J., Eisemann, M., Brähler, E. (1999): Rückblick auf die Eltern: Der Fragebogen zum erinnerten elterlichen Erziehungsverhalten (FEE). Diagnostica 45, 194-204.

Stein, M. B., Koverola, C., Hanna, C., Torchia, M. G., McClarty, B. (1997): Hippocampal volume in women victimized by childhood sexual abuse. Psychol Med 27, 951-9.

Straus, M. A. (1979): Measuring intrafamily conflict and violence: the Conflict Tactics (CT) Scales. Journal of Marriage and the Family 41, 75-88.

Vaillant, G. E. (1976): Natural history of male psychological health. V. The relation of choice of ego mechanisms of defense to adult adjustment. Arch Gen Psychiatry 33, 535-45.

Vaillant, G. E., Bond, M., Vaillant, C. O. (1986): An empirically validated hierarchy of defense mechanisms. Arch Gen Psychiatry 43, 786-94.

Vallee, M., Mayo, W., Maccari, S., LeMoal, M., Simon, H. (1996): Long-term effects of prenatal stress and handling on metabolic parameters: relationship to corticosterone secretion response. Brain Res 712, 287-292.

Vallee, M., Mayo, W., Dellu, F., LeMoal, M., Simon, H., Maccari, S. (1997): Prenatal stress induces high anxiety and postnatal handling induces low anxiety in adult offsprings. J Neurosci 17, 2626-2636.

van der Kolk, B. A., Fisler, R. E. (1994): Childhood abuse and neglect and loss of self-regulation. Bull Menninger Clin 58, 145-68.

van Os, J., Jones, P., Lewis, G., Wadsworth, M., Murray, R. (1997): Developmental precursors of affective illness in a general population birth cohort. Arch Gen Psychiatry 54, 625-31.

Wadsworth, M. E. (1987): Follow-up of the first national birth cohort: findings from the Medical Research Council National Survey of Health and Development. Paediatr Perinat Epidemiol 1, 95-117.

Wadsworth, M. E., Kuh, D. J. (1997): Childhood influences on adult health: a review of recent work from the British 1946 national birth cohort study, the MRC National Survey of Health and Development. Paediatr Perinat Epidemiol 11, 2-20.

Werner, E. E. (1989): High-risk children in young adulthood: a longitudinal study from birth to 32 years. Am J Orthopsychiatry 59, 72-81.

Werner, E. E. (1992): The children of Kauai: resiliency and recovery in adolescence and adulthood. J Adolesc Health 13, 262-8.

Werner, E. E. (1997): Vulnerable but invincible: high-risk children from birth to adulthood. Acta Paediatr Suppl 422, 103-5.

Wetzels, P. (1997): Gewalterfahrungen in der Kindheit: Sexueller Missbrauch, körperliche Misshandlung und deren langfristige Konsequenzen. Baden-Baden.

Widom, C. S. (1989). The cycle of violence. Science 244, 160-66.

Wyman, P. A., Cowen, E. L., Work, W. C., Parker, G. R. (1991): Developmental and family milieu correlates of resilience in urban children who have experienced major life stress. Am J Community Psychol 19, 405-26.

Yarrow, M. R., Campbell, J. D., Burton, R. V. (1970): Recollections of childhood. A study of the retrospective method. Monogr Soc Res Child Dev 35, 1-83.

Address for correspondence:

Prof. Dr. med. Ulrich T. Egle, Dep. of Psychosomatic Medicine and Psychotherapy, Untere Zahlbacherstr. 8, 55131 Mainz, e-mail: egle@psychosomatik.klinik.uni-mainz.de