

## CHAPTER 15

# *Memory and Developmental Psychopathology*

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How does children's memory operate and what are the critical developmental shifts during childhood that change memory from a less mature to a more mature system? How are these processes altered, if at all, in children experiencing trauma or psychopathology? In this chapter, we address these questions in the context of child maltreatment and the chronic stress and psychiatric sequelae associated with child abuse and neglect. Very solid theoretical grounds encourage us to anticipate that stress, particularly the stress associated with early aversive childhood experiences such as maltreatment, produces alterations in basic memory processes. Throughout this review, however, we show that the evidence does not substantiate such a claim.

Drawing on a developmental psychopathology perspective, we begin by discussing how childhood trauma might affect the normal course of memory development. Although there are scant data to date, there are a number of theories that lead us to believe that fundamental processes may be altered as a function of childhood trauma. In what follows,

we outline these theories and then evaluate them in the context of specific memory data related to children's basic memory processes and autobiographical memory. We conclude with a discussion of future research directions that should be undertaken to affirm or challenge our contention that, based on extant empirical findings, memory operates similarly in traumatized and nontraumatized individuals.

### **HOW DOES TRAUMA AFFECT MEMORY AND MEMORY DEVELOPMENT?**

Trauma and the stress associated with it are thought to have a variety of effects on basic memory processes as well as on memory for traumatic events themselves. In an early description of the field of developmental psychopathology, Sroufe and Rutter (1984) stated ". . . the focus is on the ontogenetic process whereby early patterns of individual adaptation evolve to later patterns of adaptation. . . . At times, studying the course of adaptation in selected nondisordered individuals also is of great interest . . ." (p. 25). In a further elaboration of the parameters of the field, Cicchetti and Cohen (1995a, 1995b) highlighted the interplay between normal and atypical development and argued that investigations with both populations are

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necessary to truly understand developmental mechanisms and processes. Therefore, in this chapter, we build on one of the central tenets of developmental psychopathology, namely, that investigations with normal populations can serve to inform approaches to atypical populations. Knowledge of the development of memory in normal populations of children provides a baseline with which similar or divergent memory processes in children who have been traumatized can be compared.

Because a number of reviews of the scientific and theoretical literature on the overall impact of stress on biological functioning (including the consequences of chronic Posttraumatic Stress Disorder [PTSD]) have recently appeared, we do not reiterate them here (see Bremner & Narayan, 1998; Bremner & Vermetten, 2001; Brewin, 2003; Dalgleish, 2004; Dickerson & Kemeny, 2004; Engleberg & Christianson, 2002; Kemeny, 2003; McEwen & Schmeck, 1994; Sapolsky, Romero, & Munck, 2000). Instead, we focus on specific aspects of stress responses that affect basic memory processes as well as those that are purported to affect memory for traumatic experiences themselves. We begin by providing an overview of physiological reactivity and stress, including a description of the measures most commonly used to index stress reactivity. This is followed by a synthesis of what is known about the effects of stress on the neural substrates of memory. In the penultimate section, we discuss theories and research concerned with acute versus chronic stress and memory development in childhood. We conclude by discussing clinical implications of this work and proposing directions for future research.

### Physiological Reactivity and Stress

Stressful events frequently lead to the release of adrenal stress hormones, including catecholamines and glucocorticoids (McGaugh, 2000; Sapolsky et al., 2000). Studies with animals and humans have shown that the release of these hormones is correlated with dramatic changes in memory and cognition (Cahill & McGaugh, 1998; Lupien & McEwen, 1997; McEwen & Sapolsky, 1995; McGaugh, Cahill, & Roozendaal, 1996). Although exposure to acute stressors can enhance memory storage and consolidation (for a review, see Howe, 1998), impairment of memory and cognitive processes has been associated with chronic exposure to stressors (McEwen & Sapolsky, 1995). Clearly, however, the presence of either acute or chronic stressors can both enhance and impair memory and cognition depending on a number of factors, including the intensity of

the stressor (see Howe, 1998) as well as individual differences in stress reactivity (see Quas, Bauer, & Boyce, 2004). In what follows, we provide a brief overview of the basic mechanisms involved in biopsychological reactivity to stressors and show how these reactions enhance or compromise memory and cognition. We consider research with animals and humans and, in the latter case, with both adults and children.

### *Catecholamines and Glucocorticoids*

It has been known for some time that physiological and psychological stress provoke an integrated response involving neural (including sympathetic and parasympathetic responses) and neuroendocrine (including the hypothalamic-pituitary-adrenocortical [HPA] axis) systems. The neural (sympathetic) contribution includes the secretion of the catecholamines epinephrine and norepinephrine, and the neuroendocrine contribution includes the secretion of glucocorticoids by the adrenal gland. A key similarity between these systems is that both catecholamines and glucocorticoids exhibit an inverted U-shaped relationship between the amount secreted and memory: Small amounts have little effect on memory, moderate amounts can enhance memory, and extreme amounts can impair memory (Gold & McCarty, 1995; Izquierdo & Medina, 1997; Korneyev, 1997; McGaugh, 1995). Although there is considerable debate as to whether such extreme levels exist in children's real-world experiences (see Chen, Zeltzer, Craske, & Katz, 2000), there are experimental studies that clearly show high levels to be deleterious to memory (see Sapolsky et al., 2000). Thus, stress can have seemingly opposite effects on memory depending on its intensity and chronicity. Further, these different effects can be either specific and experience-dependent (e.g., affecting only the specific event memory) or general and more globally relevant to basic memory processes (e.g., exhibiting long-lasting structural and functional consequences such as hippocampal cell loss or elevated levels of circulating catecholamines). In this chapter, we are concerned with both of these effects—the former because it addresses the issue of what can be remembered about traumatic experiences and the latter because it goes directly to the issue of how basic memory processes may be affected more generally by trauma and stress.

We should note that there are also differences in how catecholamines and glucocorticoids function. Catecholamines do not enter the brain directly but exert their effects indirectly (i.e., through secondary-messenger cascades at postsynaptic sites). Glucocorticoids directly af-

fect receptors especially in the hippocampus, a structure well-known to play an important role in learning and memory (particularly in the consolidation of memories). If this latter structure is permanently altered by glucocorticoid activity in response to stress and trauma, then questions arise concerning the longevity of memories from the traumatic experiences.

Two other distinctions between catecholamines and glucocorticoids are important. The first of these concerns the time course: The indirect effects of catecholamines develop over a relatively short interval (within seconds), whereas the glucocorticoids are secreted over a matter of minutes and their effects can take hours to emerge. Because memories are not formed instantaneously, taking a rather protracted time course to emerge (from hours to days depending on which model of consolidation one adheres to), both of these effects will be important but at different points in the time course of memory formation. The second difference concerns the site of their effects: Stress-induced catecholamine effects are localized primarily in the amygdaloid complex, whereas stress-induced glucocorticoid effects are localized primarily in the hippocampal complex (McEwen & Sapolsky, 1995).

Although different sites are affected, there is good reason to believe that both catecholamines and glucocorticoids can enhance or inhibit memory formation. That is, considerable evidence exists showing that the amygdaloid complex plays a central role in modulating (either enhancing or impairing) memories for stressful experiences (Cahill, 2000; Cahill & McGaugh, 1996; McGaugh, 1995, 2000, 2003; Pelletier & Pare, 2004). Similar memory-enhancing and memory-impairing effects have been observed with the stress-induced release of the endogenous glucocorticoids corticosterone (in rats) and cortisol (in humans), and these effects are believed to influence the neural processes linked to consolidation of memories (Diamond, Fleshner, Ingersoll, & Rose, 1996; Newcomer, Craft, Hershey, Askins, & Bardgett, 1994; Oades, 1979; Pugh, Tremblay, Fleshner, & Rudy, 1997).

Regardless of the locus of these effects, both catecholamines and glucocorticoids modulate what gets stored in memory and may do so by altering processes involved in consolidation (Abel et al., 1995; Cahill & McGaugh, 1996; Izquierdo & Medina, 1997). Consolidation is that process through which initially encoded information is transformed from its initial labile, transient form to a more stable, less volatile form (for a recent overview, see Dash, Hebert, & Runyan, 2004). More formally, consolidation refers to a phase of memory formation in which, among other things,

synaptic connections are strengthened, a process that is similar in duration at both the cellular and behavioral levels (for a more complete description, see Abel et al., 1995; Dash et al., 2004; McGaugh, 2003). Because it is during this consolidation interval that memories are most susceptible to interference and distortion, it is important to determine the length of this interval. Originally, consolidation referred to the stabilization of memory within the first few hours following encoding. Subsequently, it has been used to refer to a more protracted interval (months to years) that involves greater transformations, ones that may involve transfer of memory traces from one region of the brain (e.g., the hippocampus) to another (e.g., neocortex; Squire, Cohen, & Nadel, 1984). The modulatory effects of stress are thought to be relevant to the original sense of consolidation as most of the biochemical cascade effects occur over a briefer time course involving hours, not weeks, months, or years (Abel et al., 1995; Izquierdo & Medina, 1997). Thus, stress-induced release of catecholamines and glucocorticoids most likely modulate memories for traumatic events by altering (enhancing or inhibiting) the course of consolidation (also see Richter-Levin & Akirav, 2003).

However, there is some evidence that although glucocorticoids may act to enhance consolidation of memories, the same dose levels can act to inhibit subsequent retrieval of those memories. That is, stress-induced or exogenously administered glucocorticoids can have contradictory effects on memory, enhancing storage (consolidation) but inhibiting later retrieval. This is true even with acute doses of glucocorticoids. For example, de Quervain, Roozendaal, and McGaugh (1998) found that glucocorticoid doses sufficient to enhance consolidation profoundly impaired later retrieval. It should be noted that these effects have been obtained in spatial memory tasks with rats and may not generalize to humans.

### *Glucose and Oxygenation*

Additional biological mechanisms can mediate the effects of stress on memory. In particular, catecholamine effects due to stress may arise because of changes in the delivery of oxygen and glucose to the brain. The catecholamine epinephrine, for example, may affect memory through its well-established effects on blood glucose. The modulatory effects of glucose on memory are, of course, dependent on a central cholinergic mechanism (Kopf & Baratti, 1995) and may be specific to the learning situation itself (Cahill & McGaugh, 1996). Specifically, when epinephrine release is experimentally blocked during an emotional episode (and glucose levels do not change), it is likely that  $\beta$ -adrenergic

receptor activation (by norepinephrine) may produce memory enhancement. In fact, the neurotransmitter norepinephrine, also important to memory, has been thought to enhance firing in neurons that participate in the encoding of environmentally significant information (Kety, 1970). Of particular importance, novelty is directly linked to norepinephrine release and subsequently enhanced retention in some animals (Kitchigina, Vankov, Harley, & Sara, 1997). In humans, blocking norepinephrine reduces the normal memory-enhancing effects of surprise (Cahill, Prins, Weber, & McGaugh, 1994; Nielson & Jensen, 1994). Relevant to this chapter, Cahill et al. found that the  $\beta$ -adrenergic blocking drug propranolol selectively impaired memory for an emotionally arousing story in healthy adults, supporting the claim that memory modulation due to emotional arousal is contingent on the activation of these  $\beta$ -adrenergic receptors even though they are not required for the formation of non-emotional (neutral) memories.

### *Effects of Prolonged Stress*

Chronic stress (specifically, the release of glucocorticoids) in animals can lead to the atrophy of dendritic branches in the pyramidal neurons of the CA3 region of the hippocampus (Watanabe, Gould, & McEwen, 1992; Woolley, Gould, & McEwen, 1990). Although some of this loss is reversible, irreversible neuronal loss in the hippocampus does occur with prolonged exposure to glucocorticoids (Kerr, Campbell, Applegate, Brodish, & Landfield, 1991; Mizoguchi, Kunishita, Chui, & Tabira, 1992; Sapolsky, Krey, & McEwen, 1985; Uno, Ross, Else, Suleman, & Sapolsky, 1989). The fact that chronic exposure to stress-induced glucocorticoids affects dendritic branching first followed by neuron loss does not appear to be species-specific because such findings have been obtained in rats, monkeys, and tree shrews (McEwen & Sapolsky, 1995). Although studies with humans are less well controlled, they, too, suggest that hippocampal atrophy is associated with prolonged glucocorticoid exposure (Axelson et al., 1993; Starkman, Gebarski, Berent, & Scheingart, 1992). Indeed, it has been known for some time that prolonged exposure to elevated levels of glucocorticoids is accompanied by cognitive impairment, including impairment of declarative memory and visual episodic memory (Nasrallah, Coffman, & Olson, 1989). Although some of these findings with human participants are compromised because of coincident disease, studies with healthy adults have also shown that a decline in declarative (explicit) measures of memory, but not in implicit measures, can be associated with sustained increases in glucocorticoids (Lupien et al., 1994; Newcomer et al.,

1994; Wolkowitz et al., 1990). Functional magnetic resonance imaging (fMRI) studies with adults who have been exposed to prolonged stress, for example, abused women (Schacter, Koustaal, & Norman, 1996) and individuals who have been diagnosed with combat-related PTSD (Bremner, Krystal, Southwick, & Charney, 1995), as well as primates (Sapolsky, Uno, Rebert, & Finch, 1990) and humans (Keenan, Jacobson, Soleymani, & Newcomer, 1995) who have been exposed to elevated glucocorticoids over prolonged treatment intervals, show decreased hippocampal volume. Although this decrease may be subject to a variety of interpretations, it is possible that toxic levels of glucocorticoids were reached given the prolonged exposure to stress or to steroid (e.g., prednisone) therapy.

Although few studies have been conducted with children, it is known that high-dose prednisone treatment of asthmatic children leads to poorer verbal memory than low-dose treatment (Bender, Lerner, & Poland, 1991). In addition, maltreated children whose abuse is tantamount to chronic stress tend to exhibit altered patterns of diurnal cortisol activity. Specifically, depressed maltreated children, relative to nonmaltreated depressed children, exhibit lower concentrations of cortisol in the morning and show an increase rather than the normal decrease in cortisol from the morning to the afternoon (Cicchetti & Rogosch, 2001; Hart, Gunnar, & Cicchetti, 1996). There is additional evidence that children exposed to prolonged stress, including sexually abused girls (DeBellis et al., 1994), experience some deficits on explicit (declarative) memory tasks, although it is not clear that these problems are directly attributable to prolonged stress (Schacter et al., 1996). Although it is apparent that such individuals can have problems remembering (e.g., transient amnesia) aspects of the precipitating event (e.g., sexual abuse), it is also clear that they can have related deficits associated with the recall of autobiographical events (Kuyken & Brewin, 1995; Parks & Balon, 1995).

### **Individual and Developmental Differences in Stress Reactivity**

As we have seen, traumatic events usually elicit high levels of stress hormones that are often maintained for a long period of time following the event (Sapolsky et al., 2000). This means that recently traumatized individuals should exhibit an exaggerated tonic level of arousal (e.g., elevated heart rate, hypersensitive startle response), something that leads to changes in information processing (e.g., attention may become narrowed and focused on a critical aspect of

the traumatic event—so-called weapon focus; Loftus, Loftus, & Messo, 1987). Information that gets encoded will be determined to some degree by this narrowing of attention, and what can be retrieved later is constrained by what was initially encoded (see Howe, 1997, 1998).

Thus, in general, there is a negative relationship between physiological arousal and attention, particularly in children (Richards & Casey, 1991; Suess, Porges, & Plude, 1994). In terms of children's memory and stress reactivity, reactive children have difficulty attending to external information when they are experiencing stress. This, in turn, limits information that gets encoded and stored; hence, memory traces may be more impoverished and less durable. Moreover, reactive children's arousal level can impact not only encoding, but also retrieval processes. That is, arousal levels may be too high for children to adequately search memory or utilize cues effectively (e.g., interviewer's questions or other available prompts). This is especially true when reactive children are being queried about stressful events or when the retrieval situation itself is stressful (see Quas et al., 2004).

But exactly how are these variations in stress and stress reactivity measured? Physiological reactivity has been measured in a variety of ways that reflect activation of the (1) autonomic nervous system (e.g., heart rate; respiratory sinus arrhythmia, an index of parasympathetic activity on the cardiac cycle; preejection period, an index of sympathetic activity on the cardiac cycle) or (2) HPA axis, most frequently operationalized in terms of salivary cortisol. As it turns out, measures of increased heart rate during the to-be-encoded event are negatively related to children's subsequent memory for those events (Bugental, Blue, Cortez, Fleck, & Rodriguez, 1992; N. Stein & Boyce, 1995). It appears that increased heart rate interferes with encoding, leading to impoverished representations of the event in memory. Although there are a number of limitations to studies such as these with children (see Quas et al., 2004), additional work using autonomic measures is clearly warranted, particularly investigations that also measure changes in heart rate during retrieval.

Considerably more research has used cortisol rather than variation in autonomic reactivity as an index of stress. For example, Merritt, Ornstein, and Spicker (1994) examined the relationship between salivary cortisol and a stressful medical procedure and found no relationship between cortisol levels and children's memory for the stressful event. Similarly, Chen et al. (2000) found no relationship between cortisol levels and children's memory for lumbar punctures. Unfortunately, the results of both of these stud-

ies are difficult to interpret given the relatively small sample sizes and the manner in which cortisol levels were calculated (Merritt et al., 1994) and because additional, potentially memory-relevant medications were present prior to the stressful procedure (Chen et al., 2000).

More recently, Quas et al. (2004) examined the interaction between physiological reactivity (autonomic responses and salivary cortisol) and social support (supportive versus nonsupportive interviewer style) on children's initial and long-term recollection of a stressful event (memory for an event involving a fire alarm). Although few significant relationships emerged at initial recollection, cortisol reactivity was associated with poorer long-term retention of the event. Moreover, autonomic reactivity was related to higher accuracy during a supportive interview and lower accuracy during a nonsupportive interview. It would seem that, at least in this study, autonomic and cortisol measures tap different aspects of memory processing for stressful materials (overall retention versus interview style), although both appear to be related to retrieval components of long-term retention.

Overall, the research on stress and memory with human and nonhuman participants suggests that both storage and retrieval processes are affected by stress. Although the results are often clearer in nonhuman animal studies, the results of the study by Quas et al. (2004) does suggest that retrieval of stressful memories is influenced by the type of interview being conducted and that overall retention levels may be influenced by children's stress reactivity. In terms of storage, although often difficult to demonstrate empirically with human child samples, "it seems reasonable that the storage of memories should be moderated in some way to enhance the contrast between memories of important events from those of less relevant memories—to enhance the distinction between signal and noise" (Nielson & Jensen, 1994, p. 190).

But why should it be so hard to demonstrate this connection between stress and enhanced storage or consolidation of information in children? As we have already pointed out, one reason is that a number of measurement issues exist when children are the sample of choice. In fact, with children, there is no consensus on the best index of stress. As previously discussed, a variety of techniques have been used to measure stress in children, including subjective self-ratings (where children rate their own level of stress), behavioral and objective rating scales (where parents or objective observers adduce the child's level of stress), physiological indices (heart rate, blood pressure, and the other indices listed earlier), and neuroendocrine measures (sali-

vary cortisol). Unfortunately, all of these measures have their weaknesses and have fared poorly with respect to predicting children's event memory. In addition to those studies already mentioned, Howe, Courage, and Peterson (1994, 1995) failed to find a relationship between self- or parent ratings of a child's stress and children's memory for emergency room treatment, even up to a year after the experience. Goodman and Quas (1997) also failed to find a relationship between subjective and behavioral measures of stress and memory for a stressful medical procedure, the voiding cystourethrogram (VCUG). Of course, the inability to find relationships between memory and stress as measured by subjective and behavioral scales may not be a surprise as such indices may be insensitive to the underlying physiological changes that are associated with stress (also see Howe, 1997, 1998). Thus, similar to the autonomic and neuroendocrine measures of stress reactivity, studies using subjective, objective, and behavioral scales have been unable to unambiguously link changes in children's stress with subsequent memory for those stressful events.

Another reason these measures, salivary cortisol in particular, may not predict event memory in studies with children is that the extent of these reactions can vary both developmentally and across individuals. For example, there are marked developmental and individual differences in cortisol responses to stress (for a review, see Gunnar, Tout, de Haan, Pierce, & Standbury, 1997). That is, neuroendocrine reactivity can fluctuate with other variables, including age, attachment styles (Nachmias, Gunnar, Mangelsdorf, Parritz, & Buss, 1996), temperament (Boyce, Barr, & Zeltzer, 1992; Kagan, 1994), stress reactivity (Gunnar et al., 1997), and knowledge (N. Stein & Liwag, 1997; for a review, see Howe, 1998). Salivary cortisol is a marker worth studying, especially because of its sensitivity to cognitive and social factors, as long as when it is examined, these other covariates are considered simultaneously. To do so, and to link cortisol and memory, Goodman and Quas (1997) pointed out that it is important to consider not only these individual differences, but also individual differences in the quality and quantity of memories for traumatic events. Frequently, such differences can be related to individual differences in the rememberer's reactions to and consequences from traumatic experiences (Brewin, Andrews, & Gotlib, 1993; Malinowsky-Rummell & Hansen, 1993). Thus, although a number of promising measures that may lead to a better understanding of the link between stress and memory in children exist, few studies have incorporated these measures into a single design. It is only when all of these individual differences factors are considered in concert that

relationships between neuroendocrine measures of stress reactivity and memory for stressful experiences will be discovered.

### **Effects of Stress and Trauma on Neural Development**

Now that we have examined the extant research on physiological reactivity and stress in memory, we direct our attention to neural development. Specifically, what are the long-term consequences of prolonged stress, especially that associated with maltreatment, on neurological development? Although research on this question has really flourished only in the past decade or so, there is evidence that childhood physical and sexual abuse is associated with decreased cortical integration (Schiffer, Teicher, & Papanicolaou, 1995; Teicher, 1994), increased electroencephalogram abnormalities (Ito et al., 1993), and diminished size of the corpus callosum (DeBellis et al., 1999; Teicher et al., 1997). There is also evidence of similar, attenuated maturation of the corpus callosum in neglected children (Teicher et al., 2004). Moreover, studies have found that early maltreatment (neglect, physical and sexual abuse) exerts a larger negative impact on the developing corpus callosum in boys than in girls (DeBellis & Keshaven, 2003; Teicher et al., 2004). This gender difference was more important (accounting for more of the variance) than whether children developed PTSD as a function of the occurrence maltreatment. Thomas and DeBellis (2004) also found that the prolonged exposure to stress in traumatized children who develop PTSD is related to those children having greater differences in pituitary volume with age than that of control children.

Although it is not clear what these differences in neurological development might mean for the functioning of basic memory processes in neglected and abused children, some of the results concerning changes in hippocampal development are suggestive. For example, a number of researchers have found that maltreatment during childhood (physical and sexual abuse) is associated with diminished left hippocampal development in adulthood (Bremner et al., 1997; Ito, Teicher, Glod, & Ackerman, 1998; M. B. Stein, 1997) especially when it is comorbid with PTSD (Bremner, Vythilingam, Vermetten, Southwick, McGlashan, Nazeer, et al., 2003; Bremner, Vythilingam, Vermetten, Southwick, McGlashan, Staib, et al., 2003). Other investigators have not found changes in hippocampal volume as a function of trauma, even in those who develop PTSD (Bonne et al., 2001), and still other researchers have not found any support for the premise that hippocampal

development differs in maltreated and nonmaltreated populations (DeBellis, Hall, Boring, Frustaci, & Moritz, 2001; Pederson et al., 2004). Part of the reason for this discrepancy in outcome may have to do with the age of the maltreated subjects when examined; for example, in the Bremner et al. (1997) study, the participants were adults who retrospectively reported childhood abuse, whereas in the DeBellis et al. study, the participants were children whose abuse was documented. Caution is also needed when interpreting these outcomes because the sample sizes are often somewhat small (e.g.,  $n_s = 9$  maltreated and 9 control in DeBellis et al., 2001, and  $n_s = 17$  maltreated and 17 control in Bremner et al., 1997).

Finally, some reports suggest an abnormal connectivity among brain regions involved in the recall of traumatic events in individuals who develop PTSD, but not in those who do not develop PTSD (Lanius et al., 2001, 2003, 2004). It may be that such dysregulation results in changes in how traumatic experiences are retrieved for people who develop PTSD, not in how or that such experiences are represented (encoded, stored, retained) in memory. Overall, then, although there is good reason to believe that the neurological substrates that subserve basic memory functioning should be altered as a consequence of early stressful experiences (e.g., child maltreatment), the empirical evidence is at best mixed.

## EXTANT THEORIES OF MEMORY AND TRAUMA

This review of the cognitive neuroscience literature has shown that there are any number of reasons we should suspect that memories of trauma are somehow different from memories of ordinary events. As we have also seen in this review, although there is some evidence that individuals who develop PTSD may retrieve traumatic memories somewhat differently from those who do not, there is little evidence to suggest that the basic memory processes of traumatized individuals are somehow fundamentally different from that of other individuals. These facts notwithstanding, there exists theoretical speculation that victims of trauma may have fundamentally altered basic memory processes. In this section, we consider a number of these theories.

### Terr's Theory

Contrary to what is known from the vast literature on memory, where amount remembered is directly related to the number of times one has experienced an event, Terr (1988, 1994) has suggested that memory for trauma is inversely related to the number of traumatic experiences a

person has encountered. That is, Terr believes that children often forget repeated traumas (what she calls Type II syndromes) but remember single traumatic experiences (what she calls Type I syndromes). Type I events revolve around a single incident, such as a kidnapping or schoolyard sniper attack, and tend to be memorable because they are distinctive inasmuch as they are unexpected and potentially life threatening. Type II events involve multiple repeated incidents of physical or sexual abuse and are not as memorable as Type I events because they are predictable and repetitive and become anticipated. Because such events become expected, children will employ defensive techniques such as denial and psychic numbing to reduce the emotional impact of these events, practices that in turn alter the encoding of these experiences such that later retention is difficult and such experiences become easily forgotten.

As has been shown in this chapter, the sorts of experiences Terr labels as Type I are, in fact, well remembered (for a review, see Howe, 2000). However, there is very little (if any) evidence that Terr's Type II events are forgotten or more poorly remembered. In fact, the only evidence that Type II experiences are not well remembered comes from Terr's (1988) own examination of 20 children's recollection of trauma they experienced prior to the age of 5 years. Of the seven who had Type II, repeated experiences, three were unable to verbally recall the events 5 months to 12 years later and four retained what were called "spotty" memories.

As others have pointed out, there are any number of reasons these conclusions about Type I and II event memories are flawed. First, Type I and II events were confounded with the child's age at the time of the trauma (also see McNally, 2003). That is, the children who could not produce verbal recall of Type II traumas were on average younger when their traumas ended than children who had single-incident Type I traumas. In fact, they were so young (6 months, 24 months, and 28 months of age) that many of these findings can be accounted for more parsimoniously by noting that this is exactly the time frame that is coincident with childhood amnesia (see Howe, 2000; Howe & Courage, 1993, 1997). Second, as Roediger and Bergman (1998) noted, the memory literature is replete with studies showing that repeated events are better remembered than one-time events. Although event repetition may lead to schematization of the memory, making some of the details concerning individual episodes difficult to discriminate, that such events are remembered and are remembered better than events that occurred only once is not in question. The extant literature on memory and repetition shows that the greater the repetition, the better the memory. Although

some might argue that such findings pertain only to non-traumatic event memories and that somehow the nature of traumatic events violates the general laws of memory, even in the context of traumatic experiences, most clinicians and researchers have found excellent recollection of repeated abuse (Goleman, 1992; Yapko, 1994), including physical (Pelcovitz et al., 1994) and sexual abuse (Archdiocesan Commission, 1990), community violence (Fitzpatrick & Boldizar, 1993), and horrific events such as the Cambodian holocaust (Kinzie, Sack, Angell, Manson, & Rath, 1986). Thus, contrary to Terr's notion that Type II repeated traumas are more poorly remembered than one-time events, there is abundant evidence that repeated abusive experiences are well remembered. Moreover, studies of intrusive memories of victims of abuse have clearly shown that people remember their abuse all too well (Ehlers et al., 2002; Hackmann, Ehlers, Speckens, & Clark, 2004; Holmes, Grey, & Young, 2005). In fact, it has been proposed that these involuntary memories occur in response to stimuli that bear a resemblance to stimuli experienced immediately prior to the traumatic event(s) and may serve as a "warning signal" (Charney, Deutch, Krystal, Southwick, & Davis, 1993; Ehlers et al., 2002; Foa & Rothbaum, 1998). Thus, Terr's idea contradicts what is known empirically. Indeed, all of the scientific evidence runs counter to Terr's assertions that the more children are abused, the more likely it is that they will forget having been abused.

### **Freyd's Betrayal Trauma Theory**

Freyd (1996; Freyd, DePrince, & Zurbriggen, 2001) proposed that children are more likely to experience amnesia for abuse inflicted by parents or caretakers than by strangers because of evolutionary pressures to maintain attachments to those who are vital to one's survival. That is, it is adaptive to forget abuse that involves such betrayals of trust to ensure one's own survival. Although she agrees with Terr that repeated traumas are more likely to be forgotten, she does so because she believes that such abuse is more likely to have occurred at the hands of a caretaker. Thus, it is the status of the abuser (caretaker or stranger) that determines forgetting, not the number of abuse incidents.

As we have already seen and will see again later when all of the evidence is discussed, abuse tends not to be forgotten but rather to be remembered (Herman, 1981). In fact, Russell's (1999) epidemiologic study found that not one incest survivor had ever forgotten his or her molestation experience. Goodman et al. (2003) found no relationship between abuser status (parent/caregiver versus

stranger) and failure to report abuse years after the abuse. Thus, as for most, if not all, survivors of trauma, there is little or no support for the idea that incest survivors experience amnesia for their abuse.

### **Van der Kolk's Theory**

According to van der Kolk (1994; van der Kolk & Fisler, 1995), trauma interferes with explicit (declarative), but not implicit, memory. This is because trauma leads to the release of stress hormones, hormones that create a sort of state-dependent memory for the traumatic experience. Further, van der Kolk argues that state-dependent memories are inaccessible to conscious recollection until the same state is induced again, and that until that time such memories remain intact and unchanged. That is, unlike other memories, van der Kolk maintains that traumatic memories are not subject to the normal deterioration processes that other memories are subject to; rather, they remain indelible. However, unlike explicit memories, implicit memories are said to appear spontaneously in the guise of flashbacks, dreams, body memories, avoidant behaviors, and so forth (Brown, Schefflin, & Hammond, 1998). Moreover, it is through these implicit memories that those who have been traumatized can "recover" an explicit memory of the traumatic experience.

This theory, too, is plagued by empirical and conceptual problems. First, as we have already discussed and will explicate more completely later, trauma does not prevent the formation of explicit, declarative memories. In fact, more often than not, stress aids the consolidation of memories for traumatic events. Although extreme levels of stress can impair consolidation, this does not result in memories that last forever, unchanged. Rather, it means that these memories do not get stored, so they cannot be recovered later. However, there is little evidence that such extreme levels of stress occur naturally. It is more likely, therefore, that such memories do get stored and that stress experiences enhance and promote the formation of explicit, declarative memory for traumatic events.

Second, even if trauma resulted in state-dependent memories that could not be accessed consciously, the evidence concerning state-dependent memories does not support the idea that such memories would be dissociated from consciousness. Specifically, research on state-dependent memories shows that although people tend to have less extensive access to those memories, they are not amnesic for the experience (Eich, 1995; Eich, Macaulay, Loewenstein, & Dihle, 1997). Of course, state depen-

dency cannot explain why memories of trauma would be recovered in a therapeutic environment that was presumably devoid of such trauma. Even individuals who report dissociative alterations in consciousness (e.g., slowing of time, out of body experiences) during traumatic events such as near-death experiences (for a review, see Greyson, 2000) or first-time skydiving (Sterlini & Bryant, 2002) do not become amnesic for these experiences, but are capable of providing detailed declarative recollections of such experiences.

Third, implicit memory does not contain an unchanging, veridical record of experience, traumatic or otherwise (see Howe, 2000). In fact, implicit memory is as subject to change and distortion as is explicit memory (Howe, 2000; Lustig & Hasher, 2001). Even if implicit memories did return as bodily sensations, unexplained feelings, and flashbacks, there is nothing in implicit memory traces that reveal their source. That is, such traces do not contain information that indexes their origin, and hence it is impossible to use them to accurately re-create the original experience. As Roediger and Bergman (1998) point out, sparsely encoded traces do not become more accurate with time. Instead, such traces undergo greater reconstruction, and hence are potentially more inaccurate, than more completely encoded traces. Moreover, such memories are not stored in muscle tissue—the idea of “body memories” is inconsistent with the cognitive neuroscience of memory, even the neuroscience of traumatic memories (see earlier section). In fact, animal research has shown that there is no such thing as indelibly etched emotional memories. Even well-established memories based on fear conditioning are labile and subject to alteration (Morrison, Allardyce, & McKane, 2002; Nader, Schafe, & LeDoux, 2000; Zola, 1997).

### Foa's Fear Networks

It is well-known that many traumatized individuals develop a heightened sensitivity to trauma-related information (Field et al., 2001; J. M. G. Williams, Mathews, & MacLeod, 1996) as well as heightened memory for, and less forgetting of, trauma-related information (Amir, McNally, & Wiegartz, 1996; Cloitre, Cancienne, Brodsky, Dulit, & Perry, 1996; McNally, Metzger, Lasko, Clancy, & Pitman, 1998; Paunovic, Lundh, & Oest, 2002). Foa (Foa & Kozak, 1991; Foa & Rothbaum, 1998) has attempted to explain these phenomena by arguing that traumatized individuals, especially those who develop PTSD, develop semantic “fear” networks that serve to organize trauma-relevant information. These networks, like other semantic networks,

are developed to integrate information about a specific theme, in this case trauma, and may be used to alert their users to the warning signs of impending threat of trauma. Not only do these networks create states of hypervigilance, but they can also serve to preserve information about trauma (e.g., through rehearsal of information) and link similar experiences together in memory (making for stronger traces). Repeated traumatic experiences should, contrary to Terr, increase the likelihood of creating these networks, hence increasing memory for traumatic experiences. These networks are also said to be linked to intrusive memories for the traumatic experience(s).

There is some evidence supporting this idea. For example, it might be predicted that individuals who have been abused (e.g., childhood sexual abuse, or CSA) might have particularly robust CSA memories as PTSD symptomatology increases, with more severe abuse resulting in better CSA memory. This is exactly what Alexander et al. (2005) found: Individuals with documented CSA and individuals with more PTSD symptomatology had particularly accurate CSA memories. Thus, emotional events such as CSA may be better preserved in trauma-related semantic networks resulting in better, not worse, recollection later in adulthood.

Conversely, traumatized individuals who have subsequently developed PTSD should exhibit anxiety responses that are specific to trauma-relevant materials. Alternatively, PTSD may be the result of pathological memory formation (Keane, Fairbank, & Caddell, 1985) in which associative learning coalesces trauma-specific cues with neuroendocrine responses in what has been called a “fear network” (Foa & Kozak, 1991; Foa & Rothbaum, 1998; Foa, Steketee, & Rothbaum, 1989). The more an individual reexperiences the traumatic event, the greater the potential increase in tonic levels of sympathetic activation. This means that victims of recent trauma should exhibit fear responses to specific trauma-related cues (as the fear network continues to emerge) and that only individuals with chronic PTSD will exhibit generally increased levels of reactivity to all stimuli.

Recent reviews of physiological research with chronic PTSD patients have routinely concluded that these individuals have heightened responses to stimuli that are related to the traumatic event (Orr, Metzger, & Pittman, 2002). In fact, this pattern seems to hold for recently traumatized individuals as well (Elsesser, Sartory, & Tackenberg, 2004). That is, both recently traumatized individuals and those with chronic PTSD exhibited similar elevated levels of activation to trauma-related materials, but the groups did not differ with respect to levels of generalized activation.

In summary, although the theories that exist regarding the effects of trauma on memory have proffered some intriguing ideas and speculations, extant empirical studies do not substantiate the theoretical claims that traumatized individuals utilize fundamentally different memory processes than nontraumatized persons. These investigations are reviewed in a subsequent section of this chapter.

## CHILDREN'S MEMORY DEVELOPMENT AND TRAUMA

Given all of the neurophysiological evidence and theoretical speculation we have just reviewed, there is good reason to expect that early trauma may result in changes so fundamental to the neural machinery necessary to encode, store, retain, and retrieve memories that the development of these basic processes will be compromised sufficiently to effect performance on everyday memory tasks. Indeed, a number of researchers have suggested that prolonged physiological reactivity to the effects of stress results in significant global changes in functioning during childhood, including increased risk of physical and mental disorders, emotional dysregulation, and cognitive dysfunction (Johnston-Brooks, Lewis, Evans, & Whalen, 1998; Porges, 1997; Raine, Venables, & Mednick, 1997; Rieder & Cicchetti, 1989; Scarpa, 1997). Despite the seeming obviousness of this linkage, there have been a number of failures to provide direct evidence of such a one-to-one mapping. Indeed, it seems more prudent to conclude that the mapping is one-to-many at best and, more realistically, many-to-many and that, rather than seeing reactivity as always being a risk factor, it is more appropriately conceived of as a form of biological sensitivity to environmental and contextual influences (also see Quas et al., 2004).

Despite these complexities, theoretical accounts of how basic memory processes might be compromised by early trauma do exist. Of the theories of memory and trauma we have just reviewed, only Foa's has received empirical support, but even here, this support has been limited. Although this theory, like the others just reviewed, is applicable to children as well as adults, none of the theories have explicitly addressed questions related to the potential changes to memory development that trauma and stress might produce. To address this question, we must first know something about the normal course of memory development. Because a complete exegesis would require numerous tomes, some of which have appeared recently (e.g., Brainerd & Reyna, 2005; Howe, 2000; Rovee-Collier, Hayne, & Columbo, 2001; Schneider & Pressley, 1997), we restrict

our presentation to those aspects of children's memory development most likely to be affected by stress, the sequelae of child maltreatment, and psychiatric disorder.

## Development of Basic Memory Processes

To begin, a number of investigators have theorized that there is considerable continuity in basic memory processes across childhood and into adulthood (Bauer, 1996; Howe, 2000). That is, infants', toddlers', and children's memory, like that of adults, is subject to misleading information and false memories (Brainerd & Reyna, 2005; Rovee-Collier et al., 2001) and interfering information (Howe, 1995; Rovee-Collier et al., 2001), can be recoded once in storage (Howe, 2004b; Rovee-Collier et al., 2001), can be selectively "forgotten" when directed to do so (Howe, 2002), benefits from distinctiveness (Howe, in press; Howe, Courage, Vernescu, & Hunt, 2000; Rovee-Collier et al., 2001), and is subject to spacing effects (Dempster, 1988; Rovee-Collier et al., 2001) and to many other factors that are well-known in the adult memory literature. Although there is continuity in basic memory processes across development, there is also considerable growth. For example, memory improves as children's knowledge base increases (Bjorklund, 1987), as their ability to encode source information (the context in which information was acquired) improves (Drumme & Newcombe, 2002), as effective strategies (e.g., rehearsal, organization) improve (Schneider & Pressley, 1997), and as metamemory improves (Howe & O'Sullivan, 1990). This growth results in increases in the *complexity* of the information that can be retained, the *duration* of the interval over which information can be retained, and the *representational flexibility* of information in memory, as well as a reduction in the *context-specificity* of what is retained, to name a few outcomes (see Howe, 2000; Rovee-Collier et al., 2001).

Despite our ability to scientifically document these and other remarkable achievements in children's memory, the question remains: What is it that develops? That is, given the striking processing continuity that exists in the face of such prodigious growth, does there exist a common set of basic processes that underlie this dramatic growth in memory functioning and capacity? There is a singular dearth of theories concerning memory development, but those that do exist either emphasize neuroscience (i.e., advances in brain maturation) or cognitive science (i.e., advances in organizational efficiency) in their explanation of growth in memory throughout childhood, although these two approaches are not mutually exclusive. We next consider the

neuroscience explanations, followed by the cognitive science explanations.

### The Neuroscience of Memory Development

According to C. A. Nelson (1995, 1997, 2000), improvements in memory during childhood are mediated by the extensive and rapid growth of structures in the brain. Specifically, Nelson argues that there are multiple memory systems (e.g., procedural versus declarative) that have different developmental trajectories. Some early developing neurological structures (e.g., the hippocampus, striatum, cerebellum, and olivary-cerebellar complex) are sufficiently well developed early in life to subservise a procedural, or what he calls a "preexplicit," memory system. This is the system that makes recognition memory possible, and it is the system that he argues is necessary for the types of memory performance seen in early infancy (e.g., novelty preference, habituation, operant and classical conditioning, visual expectancy). By contrast, performance on explicit, declarative memory tasks depends on the addition of later developing structures of the medial temporal lobe (e.g., the amygdala), inferior temporal cortical regions, and some aspects of the prefrontal cortex. These memory-relevant structures are not available until some time in the second half of the 1st postnatal year.

In addition, early life brings with it a number of ongoing neural developments, including proliferation of synapses, dendrites, fiber bundles, neurotransmitters, and myelin (see Johnson, 1997, 2000). In fact, the process of synaptogenesis peaks in infancy and toddlerhood and, following a period of overproduction, connections are pruned back and decline thereafter in early childhood (Huttenlocher, 1999). A key point for the purposes of this chapter is that experience is critical to this neural development. Specifically, the selective survival or loss of certain synaptic connections as well as the growth of new ones hinges on experience and interaction with the environment (Greenough & Black, 1999; Greenough, Black, & Wallace, 1987). Experience-based synaptogenesis and related changes in connectivity contribute to some of the neural infrastructure or hardware that supports advances in memory software (e.g., encoding, storage, retrieval). The extent to which and the manner in which different types of early experience affect neural development and hence the development of basic memory hardware and software is not well documented. Although this important area of investigation remains young, it is clear that a complete understanding of the development and malleability of basic memory processes early in life is contingent on

mapping the interaction of experience with the structural and functional plasticity of early neural development.

### Cognitive Science of Memory Development

The ideas discussed in the previous section are not without their detractors due to some rather contentious assumptions. For example, some of the work has been conducted in brain-damaged adult populations or with lesioned animals. It is not clear that such findings generalize to intact, human children. Moreover, it is not at all clear that multiple memory systems exist. Indeed, there is evidence against this hypothesis (for a review, see Howe, 2000; Rovee-Collier et al., 2001). Even if there are multiple memory systems, it is not clear whether they develop sequentially or in parallel.

A good example of this debate concerns the idea that there exist separate implicit (i.e., nondeclarative memories that are said to be outside of conscious awareness) and explicit (i.e., declarative memories that are available to consciousness) memory systems. It has been argued that even though we may not have a conscious recollection of a traumatic experience, its residue nonetheless persists in our implicit, nondeclarative system and can still influence our behaviors, unbeknownst to us (for a brief overview, see Howe & Courage, 2004). The problem here is that extreme caution must be exercised when interpreting nonverbal behaviors (the quintessential index of implicit memory) as indices of memories of past traumatic events. This is because there is no direct mapping of specific traumatic experiences onto specific behaviors (e.g., a child's fear of loud noises cannot be inextricably linked to an earlier experience of hearing gunshots while witnessing the murder of his or her parents). Although it is important to remain open to the possible nonverbal behaviors that may signal memories for traumatic events, it is nonetheless prudent to be wary of the circularity of such observations (Howe & Courage, 2004). In any event, the available evidence suggests that even if there exist separate declarative and nondeclarative memory systems, they are both available and functioning very early in life and most likely develop in parallel (Howe, 2000; Rovee-Collier et al., 2001).

Because of these and other concerns about a pure neuroscience approach to memory development, cognitive science models of memory development (e.g., Brainerd & Reyna's, 2005, fuzzy-trace theory; Howe's, 2000, trace-integrity theory) have been constructed. Although these models were constructed independent of the ones just reviewed, they are consistent with findings in the neuroscience literature. For example, in fuzzy-trace theory, memory consists of two traces: a verbatim trace (one that

contains surface features of the experience as well as item-specific information) and a gist trace (one that contains the meaning of the experience as well as other relational, elaborative information). Although both types of traces develop early and in parallel, verbatim processing tends to dominate early in memory development, with gist processing becoming more dominant (depending on the task at hand) as development proceeds.

In contrast, trace-integrity theory views memory as consisting of unitary traces and memory development as a continuous process (Howe, 2000). This means that both verbatim and gist, implicit and explicit, procedural and declarative aspects of memory are contained within the same unified trace in memory. Differences in the retrievability of these different components of memory traces are controlled by trace integration and access features of the retrieval task and not by differences in the availability of different memory traces.

Regardless of which theory one adopts, the key to memory development is corresponding changes in organizational capacity. In fuzzy-trace theory, this may seem obvious inasmuch as gist processing begins to take on more and more importance as memory development unfolds. Perhaps the role of organization is equally obvious in the trace-integrity theory. Here, memory traces are viewed as collections of basic elements (e.g., features, nodes) that come to be integrated through processes linking the current functional information (that subset of features extracted and encoded from the current environment) with knowledge already stored in memory. Traces lie on a continuum from little or no integration to high integration, in which the elements within the trace have great cohesion and the trace itself is distinct from other traces in memory, making it highly discriminable for retrieval. The relationships among items or elements within the trace provides the “glue” for these traces. As children’s ability to organize and extract features from the environment grows, their ability to store and retrieve more durable traces increases. Because children’s ability to extract more and more abstract information from displays increases with conceptual development, as does their knowledge base, their ability to store and retrieve more conceptually sophisticated information also burgeons.

As an example, consider the onset of autobiographical memory or personal memories for events that happened to “me” (a more complete account is provided in the later section on autobiographical memory). It is well-known that before 18 to 24 months of age, children can remember events, but those events are not organized as events that happened to “me,” nor are they remembered in later childhood or adulthood (so-called infantile amnesia). Event memory can

become autobiographical only when there is a cognitive self (a me with recognizable features) that provides an organizational framework for storing and retrieving event information (Howe, 2004a).

Most theories of memory development concur that traumatic experiences should be well remembered due to their distinctive (e.g., emotional, unique) character (better discrimination of traces). The effects of chronic stress may be outweighed by effects due to differences in the semantic organization of experiences. That is, because maltreated children are often those who come from the lowest socioeconomic strata of our society, and because it is well-known that socioeconomic status (SES) is linked to differences in memory performance due to variation in knowledge base, it may well be that maltreated children’s basic memory processes are the same as those of nonmaltreated children (unless there is demonstrable brain injury) and differ only in the manner in which information is organized semantically (see Howe, Cicchetti, Toth, & Cerrito, 2004, for further discussion).

## MEMORY DEVELOPMENT AND CHILDHOOD TRAUMA

The question as to whether childhood trauma alters the course of normal memory development is a difficult one to answer and is the subject of a number of ongoing research programs. In this section, we review what is known about this topic by addressing questions concerning the nature of children’s memory for stressful and traumatic experiences and whether there is any evidence that traumatized children’s basic memory processes have been altered by their trauma experience(s).

### Children’s Memory for Stressful and Traumatic Experiences

A number of recent reviews have appeared on the topic of children’s recollection of stressful and traumatic experiences (e.g., Cordon, Pipe, Sayfan, Melinder, & Goodman, 2004; Howe, 1997, 1998, 2000), and all of them have concluded that, barring accompanying cerebral assault, children can and do remember many of the central elements of a traumatic event. For example, Howe and colleagues (1994, 1995) reported a series of studies in which young (18 months to 5 years) children’s memory for an accident and subsequent emergency room treatment was examined immediately (within 2 to 3 days of treatment), after 6 months,

and following a 1-year delay. Using free recall as well as open-ended cued recall, Howe et al. found that although details peripheral to the event were forgotten over time, the gist of the event was well remembered even after a 1-year interval. Interestingly, a subset of children who experienced additional emergency room experiences during the retention interval often blended these experiences into a single report (Howe et al., 1995). Although these intrusions did not retroactively interfere with recall of the original experience, an interviewer naïve to the original experience would not have been able to separate the information from the different experiences. Similar findings have been obtained in studies examining children's recall of medical examinations (Bruck, Ceci, Francoeur, & Barr, 1995; Ornstein, Shapiro, Clubb, Follmer, & Baker-Ward, 1997) and memories for anticipated painful medical procedures such as bone marrow transplants (Stuber, Nader, Yasuda, Pynoos, & Cohen, 1991), lumbar punctures (Chen et al., 2000), and the VCUg (Goodman, Quas, Batterman-Faunce, Riddlesberger, & Kuhn, 1994; Merritt et al., 1994). Overall, the findings from studies such as these converge on the conclusion that even recall of traumatic experiences is reconstructive and prone to the same type of errors commonly found in the recall of more mundane events.

Similar conclusions have been reached concerning children's memory for other, naturally occurring traumatic events. For example, children's memory for sniper attacks (Pynoos & Nader, 1989; Schwartz & Kowalski, 1991), hurricanes (Ackil, van Abbema, & Bauer, 2003; Bahrick, Parker, Fivush, & Levitt, 1998; Shaw, Applegate, & Schorr, 1996), earthquakes (Najarian, Goenjian, Pelcovitz, Mandel, & Najarian, 1996), a fatal bus-train collision in Israel (Tyano et al., 1996), attacks during the Gulf War (Dyregov, Gjestad, & Raundalen, 2002; Laor et al., 1997), and imprisonment in Cambodia (Kinzie et al., 1986), although accurate concerning the gist of the traumatic experience, is also subject to significant reconstruction. Not only do children tend to remember traumatic experiences reconstructively, but, like adults, they also experience involuntary, intrusive memories of these events (Malmquist, 1986; Pynoos & Eth, 1984). Such intrusive memories may serve as a warning signal that similar traumatic experiences are about to occur (see Ehlers et al., 2002; Hackmann et al., 2004).

Finally, children's and adults' recollections of childhood abuse (sexual and physical) are not only well remembered, but, as already noted, the probability of remembering tends to increase with the number of abusive incidents (Archdiocesan Commission, 1990; Goleman, 1992; Yapko, 1994). Like memories of natural disasters (Green et al., 1994), recollections of abusive experiences tend to be fairly durable,

being recallable many years later (see Alexander et al., 2005; Femina, Yeager, & Lewis, 1990; Loftus, Polonsky, & Fullilove, 1994; L. M. Williams, 1994). However, such recollections, like all recollections, are subject to reconstructive memorial processes.

Thus, the evidence indicates that, similar to memory for more mundane events, children's memory for traumatic experiences is good for central or gist information even though it is reconstructive in nature. One important difference may be that traumatic experiences are more durable than nontraumatic ones. The reason for this might be that traumatic or stressful memories carry with them some adaptive significance. That is, they can signal impending threat when the appropriate warning stimuli are present in the environment (e.g., Ehlers et al., 2002; Foa & Rothbaum, 1998). The preservation of such memories then helps to assure the organism's survival by assisting in the avoidance of similar threatening events (also see Wiedenmayer, 2004). In addition, such experiences are distinctive and personally significant, qualities that are well-known in autobiographical memory research (Brewer, 1986; Conway, 1996; Howe, 1997) for promoting longevity. More generally, naturally occurring distinctive experiences, as well as those created in the lab, are well-known to be better remembered than less distinct experiences (Howe, *in press*; Howe et al., 2000). Thus, there is considerable continuity in the memory processes governing recollection of traumatic experiences and those governing more traditionally studied memory experiences. The question that remains, however, is whether these general processes that govern memory and memory development are somehow altered by stressful and traumatic experiences, causing them to differ over time, particularly in children who have experienced chronic stress.

### Traumatized Children's Basic Memory Processes

As previously discussed, autobiographical memory can begin as early as 2 years of age, the time at which the cognitive self emerges (Howe, 2004a). Although we will defer discussion of autobiographical memory until the next section, it is of interest to note that the clinical literature is replete with examples of nonverbal indices of traumatic memories from before the age of 2 years in traumatized infants (Gaensbauer, 1995, 2002; Paley & Alpert, 2003; Terr, 1988). Most of these infant memories are revealed during play therapy and, as cautioned earlier, need to be interpreted carefully. In fact, it is not clear that these nonverbal play behaviors represent actual memories as they require considerable prompting to be elicited, are heavily dependent on

context (events, places, people, etc.), and may be elicited because of the demand characteristics of the props and context (Cordon et al., 2004; see Howe et al., 1994, for a case study). Generally speaking, then, there is little evidence to substantiate the claim that traumatic experiences are retained, implicitly or explicitly, before the age of 18 to 24 months, and it is very clear that they do not become part of our adult memories for events (L. M. Williams, 1994).

By approximately 2 years of age, children are capable of remembering distinctive and traumatic events. Traumatized children are also capable of remembering events at this age (see the section on autobiographical memory), but the relevant question here is whether these experiences subvert the normal course of memory development. That is, do traumatized and maltreated children's basic memory processes begin to work in ways that are fundamentally different from those of nonmaltreated children?

This question has been addressed in only a handful of studies, most of which have been published only recently. For example, Eisen, Qin, Goodman, and Davis (2002) examined maltreated children's memory and suggestibility about an anogenital examination and clinical assessment. The children, ages 3 to 17 years, had all been referred to an inpatient hospital unit specializing in the assessment of child abuse and neglect. Children were partitioned into three groups: (1) the abuse group that included children who were physically abused, sexually abused, or both; (2) the neglect group that included neglected children and children whose parent had a documented addiction but where there was no indication of abuse; and (3) a nonabused group consisting of children who had been referred to the clinic but where claims of abuse or neglect were not substantiated. Children were administered a series of standard tests (including scales measuring dissociation, memory, and general psychological functioning), measures of stress, and a questionnaire concerning memory for the psychological consultation, including a photo identification task concerning the clinician who conducted the interview. Of particular interest were the children's responses to a questionnaire designed specifically to examine suggestibility in the context of the child's anogenital examination. This contained questions about events that happened (e.g., "Was there a nurse in the doctor's room with you?") as well as misleading questions (e.g., "The nurse took her clothes off, didn't she?").

The results showed the typical age effects in memory, where older children remembered more than younger children and younger children were more susceptible to misleading suggestions than were older children. Of particular importance, maltreated children were no more likely than

nonmaltreated children to confuse details about the medical examination. In other words, maltreated children were no more susceptible to suggestion than nonmaltreated control children.

Although maltreatment may not affect children's ability to correctly remember a medical examination or their ability to resist suggestions about the examination, they may be more susceptible to false memories, particularly those that are relatively automatically generated. In particular, if stress and maltreatment result in lowered cognitive inhibition (e.g., it is more difficult to "screen" intrusive memories or selectively attend to only certain aspects of a stimulus display), then maltreated children may be more susceptible to falsely remembering strong semantic associations when memorizing lists of interrelated concepts. Previous research with nonmaltreated children has shown that, unlike suggestibility effects, false memories such as the ones just described (i.e., those obtained using the Deese-Roediger-McDermott or DRM procedure) increase rather than decrease with age in childhood (for a review, see Brainerd & Reyna, 2005). This is because, although such false memories occur relatively automatically, they do depend on children's being able to extract the meaning of not only each individual concept, but also the common meaning across all of the list members. For example, upon hearing (and trying to remember) the list *bed, rest, awake, tired, dream, wake, snooze, blanket, doze, slumber, snore, nap*, older children are more likely to falsely remember *sleep* than younger children.

To see whether maltreated children were more (or less) susceptible to these types of false memories, Howe et al. (2004) examined three groups of children's performance on the DRM task. Specifically, 60 middle-SES children (the children most frequently sampled when studying memory development and false memories in particular), 48 maltreated low-SES children (all of whom had documented physical, sexual, or emotional abuse or were neglected), and 51 nonmaltreated low-SES comparison children (none of whom were abused or neglected) ages 5 to 12 years studied a series of DRM lists. Recall measures were taken following the presentation of each list and a final recognition test was administered after all of the lists had been presented.

The results showed that all of the children exhibited age increases in both true and false memories, consistent with previous research, and that these patterns were the same regardless of maltreatment status. That is, both maltreated and nonmaltreated children exhibited false memories, and the number of false memories increased with age at the same rate. The only differences that emerged were associ-

ated with SES. That is, consistent with prior studies, low-SES children exhibited poorer overall performance (lower true and false memories) than did middle-SES children.

To summarize, there is considerable reason to believe that maltreated (abused and neglected) children's basic memory processes should be adversely affected if for no other reason than the chronic stress such children have experienced. However, like the conclusion concerning similar subversive effects on neural development, particularly neural developments relevant to memory performance, empirical findings to date do not substantiate such claims. Although admittedly few studies have been published and there are clearly other basic memory processes that need to be examined, those that have been conducted do not reveal differences in basic memory performance between maltreated children and demographically similar nonmaltreated control children.

Perhaps these differences are more apt to show up in maltreated children's recollections of traumatic experiences themselves. Although we have already seen that children tend to be reasonably accurate at remembering traumatic experiences, due perhaps to their distinctiveness (Howe, *in press*), there exists a strong theoretical rationale for the belief that these memories, indeed all of autobiographical memory, may be fundamentally different in children who have been maltreated compared to those who have not (Foa & Rothbaum, 1998; Terr, 1988). We examine this belief next in the context of research on children's (maltreated and nonmaltreated) autobiographical memory.

## AUTOBIOGRAPHICAL MEMORY

The stress and trauma of child maltreatment (acute or chronic) can be associated with subsequent psychiatric impairment (MacMillan et al., 2001). In particular, child maltreatment is a risk factor for Major Depressive Disorders (Heim & Nemeroff, 2001) and PTSD (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995). The interaction of individual differences characteristics in stress reactivity, cognitive as well as psychobiological, and child maltreatment are complex and lead to considerable variation in predicting which children will come to have psychopathologies (Delahanty, Nugent, Christopher, & Walsh, 2005; Shea, Walsh, MacMillan, & Steiner, 2005). In fact, diversity in process and outcome is a hallmark of a developmental psychopathology perspective (Cicchetti & Rogosch, 1996). These concepts are important to consider when examining memory in victims of trauma. Rather than focusing on main effect models that look broadly at the effects of

trauma on memory, it is necessary to recognize that not all trauma victims are affected similarly (Cicchetti, 2002; Cicchetti & Toth, 1995). The principle of multifinality suggests that similar experiences of trauma may not affect memory in the same way in different individuals. Thus, for example, it is unlikely that all physically or sexually abused children will evidence similar memory changes. This principle is also useful in addressing seemingly discrepant findings in the literature regarding enhanced versus impaired memory in traumatized populations. Conversely, equifinality suggests that there are multiple pathways to the same outcome. Accordingly, various types of traumatic experiences may eventuate in similar effects on memory.

Despite diversity in process and outcome with respect to the emergence of psychopathology, there are some important implications for memory functioning when these pathologies are a consequence of child maltreatment. In this section, we concentrate on those consequences that are associated with autobiographical memory.

Autobiographical memory begins as early as 18 to 24 months of age (see Howe, 2000, 2004a; Howe & Courage, 1993, 1997; Howe, Courage, & Edison, 2003). As noted in trace-integrity theory, this time corresponds to the point at which children acquire a cognitive sense of self, an important ingredient to organizing memories as autobiographical. That is, although as we have already noted that children have both implicit and explicit memory from very early in life, such memories are fragmentary and somewhat disorganized and are not well retained over time. With the advent of the cognitive self, such memories become organized not just as events but as events that happened to "me." Like all other advances in early memory development, information is better retained, becoming more durable, as it becomes better organized. Once children can use the cognitive self to organize events, autobiographical memory emerges and children's memories of events that happened to them become more stable and durable.

This theory not only is logically appealing but also has received empirical support (see Howe, 2004a; Howe et al., 2003). An interesting feature of this theory is that the onset of the cognitive self, and hence autobiographical memory, appears to be controlled more by maturational factors than by environmental factors. For example, infants who have delayed maturation (e.g., due to Down syndrome, familial mental retardation, Autism) show marked delays in the onset of the cognitive self (Cicchetti, 1991; Hill & Tomlin, 1981; Loveland, 1987, 1993; Mans, Cicchetti, & Sroufe, 1978; Schneider-Rosen & Cicchetti, 1984, 1991; Spiker & Ricks, 1984). These children do acquire a cognitive self if and when they achieve a mental age comparable to that of

nondelayed infants who do have a cognitive sense of self. Other recent data have shown a link between the onset of the cognitive self and constitutional factors, such as stress reactivity and temperament (DiBiase & Lewis, 1997; Lewis & Ramsay, 1997). DiBiase and Lewis found that infants with a difficult temperament at 5 months of age were more likely to show earlier acquisition of the cognitive self than were infants with an easy temperament. Lewis and Ramsay found that children with higher stress reactivity (measured in terms of cortisol levels and behavioral responses to inoculations at 2, 4, 6, and 18 months) also had earlier onset of the cognitive self than did those with lower stress reactivity. In contrast, Lewis and Brooks-Gunn (1979) have shown that the onset of the cognitive self is not related to a child's sex, maternal education, family SES, birth order, or number of siblings. Likewise, maltreated infants whose aberrant caretaking environments are associated with delays or deviations in emotional development as it relates to the self are not delayed in the onset of the cognitive self (Cicchetti, 1991; Cicchetti & Beeghly, 1987; Schneider-Rosen & Cicchetti, 1984, 1991), although they do exhibit a more negative reaction to their mirror image (Cicchetti, Beeghly, Carlson, & Toth, 1990; Schneider-Rosen & Cicchetti, 1984, 1991). Thus, the onset of the cognitive self, the critical achievement necessary to kick-start autobiographical memory, is linked more to maturation-constitutional factors than to social-experiential factors. That is, the emergence of the cognitive self and autobiographical memory is associated with maturational achievements and does not appear to be influenced by variations in social or child-care experiences in any obvious way.

Autobiographical memory is, of course, memory for our personal history. When asked to generate detailed personal memories for events, most people can generate fairly specific responses. For example, when a child (or an adult) is presented with the cue word *holiday*, he or she might respond with a fairly specific accounting of a trip to Disney World in Florida (e.g., Hammond & Fivush, 1991). The same cannot be said for people suffering from depression. Such individuals tend to provide an overly general response, such as "I remember enjoying holidays as a child," to autobiographical memory cues. This effect was first discovered in suicidal individuals (J. M. G. Williams & Broadbent, 1986; J. M. G. Williams & Dritschel, 1988) and has subsequently been replicated in other populations of depressed individuals (Brittlebank, Scott, Williams, & Ferrier, 1993; Croll & Bryant, 2000; Dalgleish, Spinks, Yiend, & Kuyken, 2001; Goddard, Dritschel, & Burton, 1996; Henderson, Hargreaves, Gregory, & Williams, 2002;

Kuyken & Dalgleish, 1995; Moore, Watts, & Williams, 1988; Puffet, Jehin-Marchot, Timsit-Berthier, & Timsit, 1991; Wessel, Meeren, Peeters, Arntz, & Merckelbach, 2001; J. M. G. Williams & Scott, 1988), those suffering from Bipolar Disorder (Scott, Stanton, Garland, & Ferrier, 2000), postpsychotic depression (Iqbal, Birchwood, Hemsley, Jackson, & Morris, 2004), adolescent mood disturbances (Swales, Williams, & Wood, 2001), and individuals remitted from Major Depression (Mackinger, Pachinger, Leibetseder, & Fartacek, 2000). Overgenerality of autobiographical memory responses has been found in other clinical populations, including persons suffering from PTSD (McNally, Lasko, Maclin, & Pitman, 1995; McNally, Litz, Prassas, Shin, & Weathers, 1994), Acute Stress Disorder (Harvey, Bryant, & Dang, 1998), Borderline Personality Disorder (Jones et al., 1999; Startup et al., 2001), Obsessive-Compulsive Disorder with comorbid depression (Wilhelm, McNally, Baer, & Florin, 1997), and persecutory delusional patients (Kaney, Bowen-Jones, & Bentall, 1999). These populations all exhibit (or exhibited) significant levels of depression, a history of trauma (particularly childhood trauma), or both. Interestingly, populations that do not have these characteristics do not tend to recall overgeneral autobiographical memories. This latter group includes populations suffering from Seasonal Affective Disorder (Dalgleish et al., 2001), Obsessive-Compulsive Disorder without comorbid depression (Wilhelm et al., 1997), and Generalized Anxiety Disorder (Burke & Mathews, 1992; Wessel et al., 2001), except for those associated with trauma giving rise to PTSD or Acute Stress Disorder.

A variety of explanations have been proposed for why overgeneral retrieval of autobiographical memories occurs in depressed or traumatized populations but not other clinical groups. Although some theories focus on the neurobiological effects of stress, the majority have to do more with cognitive explanations of changes in retrieval patterns than with neurobiologically based problems that necessarily affect the encoding and storage of information. Specifically, J. M. G. Williams (1996) has suggested that autobiographical memory is organized hierarchically (as in Conway & Pleydell-Pearce, 2000). In this model, there are two types of hierarchies in which events are stored and organized: *general categories*, in which information relating to events, people, activities, and places are stored, and *extended time periods*, in which information relating to different life periods, or periods lasting longer than a day, are stored. Within each hierarchy, more specific details are nested. Because information is organized in this top-down fashion, and because retrieval proceeds in a top-down manner, re-

trieval can be aborted at the highest level before specific information is accessed, leading to overgeneral or generic autobiographical recall. Ending a search prematurely in the general categories hierarchy leads to categoric overgeneral memory, in which participants report a summary of several episodes sharing common features (e.g., “I like theme parks”), whereas ending a search prematurely in the extended time period category leads to an extended overgeneral memory that summarizes several episodes from a common time period (e.g., “I enjoyed holidays as a child”). As it turns out, not only are depressed individuals more likely than nondepressed to produce generic memories than specific ones, but they are also more likely to produce categoric overgeneral recall than extended overgeneral memory, whereas the different generic responses when given by nondepressed individuals are equally common (J. M. G. Williams & Dritschel, 1992).

J. M. G. Williams (1996) attempted to link this model to an early sociolinguistic approach to autobiographical memory in childhood (K. Nelson & Gruendel, 1981) in which children were said to first represent and retrieve events generically.<sup>1</sup> Williams hypothesized that stressful experiences during childhood might have led depressed patients to become “arrested” in their generic retrieval of experiences. That is, they adopt a generic retrieval strategy to avoid specifics of past events and hence reduce the negative affect associated with them.

What aspects of a person’s history cause him or her to adopt a retrieval style that stops searching for specific memories and reports only generic information? Studies that have compared prior trauma and depression provide mixed results. First, as we have already indicated, numerous studies (listed earlier) have found that overgeneral autobiographical memories are correlated with depression.

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<sup>1</sup>It is now known that children’s event representations, like that of other domains of cognitive functioning, are represented quite specifically in memory and that memory development proceeds from specific, verbatim, contextually driven representations to more general, generic, gist-like representations (Brainerd & Reyna, 2005; Howe, 2000; Mandler, 2004). As well, as we have already described in this chapter, it is also known that even young children’s autobiographical memories contain very specific, verbatim details of their experience and are not represented generically (also see Howe, 2000). Of course, although the developmental arrest component of this model is not supported in theory or data, Williams’s point that people in some clinical populations report only generic rather than specific autobiographical information is still seen as feasible by some in the clinical literature today.

However, what is not clear is whether these effects represent true retrieval effects or simply a more general reluctance to report retrieved memories. That is, specific memories may have been retrieved, but depressed patients are simply less likely to report them to an interviewer than are nondepressed individuals. This is consistent with the finding that depressed individuals exhibit global cognitive and memory deficits due to lapses in attention and motivation (Burt, Zembar, & Niederehe, 1995). Although a promising idea, Wessel, Merckelbach, and Dekkers (2002) reported no such association between standardized measures of memory performance and overgeneral autobiographical memory. A further complication, and one that may suggest that overgeneral memory is not simply a reporting issue, are the findings that depressed individuals do produce specific memories as well as overgeneral ones and sometimes produce more specific memories to negatively valenced cues, sometimes produce more specific memories to positively valenced cues, and sometimes there is no difference between positive and negative cues (see Dalgleish et al., 2003).

The view that overgeneral memory may be the result of trauma, particularly trauma in childhood, is supported by Kuyken and Brewin’s (1995) reanalysis of their data set (reported in Dalgleish et al., 2003), in which more overgeneral memories were produced by a clinical sample of individuals who reported having been abused than by those not reporting abuse, even when depressed mood was controlled. The finding that self-reported abuse is linked to overgeneral memory has been replicated in a nonclinical community sample of abuse victims (Henderson et al., 2002). Taken together, these results suggest that self-reported abuse, not depression, may be responsible for overgeneral autobiographical memories. This is consistent with J. M. G. Williams’s (1996) suggestion that exposure to psychological trauma in childhood perturbs the normal development of autobiographical memory (for negative experiences primarily, but may also affect recall of positive experiences) and that children learn a specific retrieval style that minimizes recall of specific experiences. Unfortunately, other researchers have not found a similar link between childhood trauma (self-reported or verified) and overgeneral memory. For example, Wessel et al. (2001) found no relationship between early abuse and overgeneral memory but did find one between depression and overgenerality. Similarly, Wessel et al. (2002) found no relationship between overgeneral memory and childhood trauma but did find an association between depression and overgenerality.

One reason for these discrepant findings concerning the role of abuse may be that the participants in the different studies suffered from varying severities of abuse. That is, studies that have shown a link between abuse and overgeneral memory (Henderson et al., 2002; Kuyken & Brewin, 1995) have differed from those that have not found this link (Wessel et al., 2001) on a number of dimensions, including severity of abuse and/or the presence/absence of abuse. Although the clinical group in the Wessel et al. (2002) study had been interned in Japanese concentration camps during World War II as children and were no doubt exposed to traumatic events at that time, it is not clear what the overall level of trauma exposure was during the internment. It may be, then, that the latter studies did not evince sufficient levels of abuse to detect a relationship with overgeneral memory. Indeed, when a clinical sample is selected such that levels of reported abuse are likely elevated relative to healthy controls, and who do not suffer from mood disorders, there exists a clear association between abuse and overgeneral memory (Dalgleish et al., 2003). Of course, the limitation with these studies is that the association is between overgeneral autobiographical memory and self-reported abuse and not abuse that has been independently corroborated.

It may be that individuals who have been abused in childhood or those who are depressed exhibit overgeneral memory because they may have something in common; that is, traumatic experiences such as physical and sexual abuse in childhood are often associated with depression in both children and adults (Allen & Tarnowski, 1989; Andrews, Valentine, & Valentine, 1995; Bemporad & Romano, 1992; Bifulco, Brown, & Harris, 1994; Carlin et al., 1994; Cicchetti & Rogosch, 2001; Downey, Feldman, Khuri, & Friedman, 1994; Fox & Gilbert, 1994; Kazdin, Moser, Colbus, & Bell, 1985; Kessler & Magee, 1994; Kuyken & Brewin, 1994, 1995; Sternberg et al., 1993; Stone, 1993; Straus & Kantor, 1994; Toth, Manly, & Cicchetti, 1992). This is consistent with J. M. G. Williams's (1996) idea that early traumatic experience leads to a failure to reduce retrieval inhibition in autobiographical memory. That is, because specific emotional information associated with traumatic events is too painful to reexperience, it is defended against by stopping the retrieval process at the overgeneral level. In the only study to examine these effects in traumatized children, Orbach, Lamb, Sternberg, Williams, and Dawud-Noursi (2001) found that children who had been exposed to family violence produced overgeneral autobiographical memories but only for that subsample who was comorbid for depression. Thus, for children, traumatic experiences alone may not be sufficient for the develop-

ment of overgeneral autobiographical recall. Rather, these traumatic experiences must be accompanied by depression.

Somewhat problematic for J. M. G. Williams's (1996) approach is that the developmental sequence for memory is incorrect. As reviewed earlier, memory development from infancy onward (including autobiographical memory) proceeds from remembering highly specific, contextually bound, verbatim information to learning about more general, categorizable gist-like meanings (Brainerd & Reyna, 2005; Howe, 2000; Rovee-Collier et al., 2001). Thus, we do not proceed from the general to the more specific but go from the specific to the more general (also see note 1). Three additional facts are at odds with Williams's account: (1) Depressed and traumatized individuals do remember specific episodes, not just generic information; in fact, the percentage of memories recalled that are specific always exceeds 50% and often exceeds 75% across studies (see Dalgleish et al., 2003), suggesting that lapses into overgeneral retrieval are the exception rather than the rule; (2) when depressed individuals recall specific information, it is more likely when they are given negative cues than positive cues; and (3) individuals with PTSD tend to have intrusive, specific, and very detailed autobiographical recollections of the event related to PTSD (although there may be a difference between involuntary and voluntary autobiographical recall). In fact, concerning this last point, the rate of memory intrusions tends to be highly correlated with overgenerality in autobiographical recall (Brewin, Reynolds, & Tata, 1999; Wessel et al., 2002). Perhaps this association emerges because some individuals use a repressive coping style to avoid consciously ruminating about traumatic memories, increasing the likelihood that they appear as intrusive recollections. Whatever the case may be, it is clear that an alternative theoretical framework is necessary to accommodate the empirical findings.

In summary, what this review has shown is that similar to more basic neurological and memory processes discussed earlier, although there is good reason to suspect that autobiographical memory processes may be perturbed in the presence of childhood maltreatment and the ensuing psychopathology, the empirical evidence is not consistent with that viewpoint. First, there appear to be no differences in the age of onset of autobiographical memory unless a maturational delay exists (e.g., due to Down syndrome, Autism). Second, although childhood trauma and maltreatment have been linked to an overgeneral reporting bias of autobiographical memories, it is not clear that these effects are related to maltreatment per se or to the comorbidity factors such as depression. Indeed, it is not clear that these effects are memory effects at all and are not simply a more

general and global characteristic of depression in which there is frequently a cognitive bias affecting the motivation to participate in tasks or to report memories of experiences. Before we can be certain, considerable research will be needed to sort through these different issues and determine whether changes in autobiographical memory occur as a result of maltreatment or comorbidity factors, and if so, whether such overgeneral reporting is simply a motivational issue consistent with other biases associated with depression or is really a memory retrieval phenomenon.

## CONCLUSION AND FUTURE DIRECTIONS

We began this chapter by asking how stress, particularly the chronic stress associated with child maltreatment, affects the development of children's basic memory processes as well as how it affects children's recollection of the stressful and traumatic experiences themselves. Although there are sound theoretical reasons to believe that stress may affect basic memory processes and memory for traumatic experiences (e.g., enhanced consolidation and storage, inhibition of retrieval), the data in hand suggest otherwise. What this review has shown is that the experience of trauma, acute or chronic, does not exert any special effects on memory that fundamentally change the operation of memory from that observed in nontraumatized populations. With that said, however, we want to underscore the fact that research in this area is still in its infancy. Because of the paucity of work in this area, we use the remainder of this chapter to highlight future directions that need to be pursued in order to have confidence that memory operates similarly in traumatized and nontraumatized individuals.

First, and perhaps foremost, we want to emphasize the importance of conducting investigations with individuals who have experienced trauma rather than trying to extrapolate from studies conducted with normal populations or from analogue studies. This recommendation is consistent with developmental psychopathology's commitment to examining atypical as well as normal populations. Unfortunately, historically much of the work on trauma and memory has been conducted with normal populations involved in simulated analogue studies. For example, in investigations of eyewitness testimony, Yuille and Cutshall (1986) reported that between 1974 and 1982, 92% of investigations purporting to examine eyewitness testimony involved college students participating in simulation studies. Much of the early work on false and suggested memories in

children similarly involved simulated studies with normative groups of children (Ceci & Bruck, 1995). Although these studies provide a valuable starting point for examining complex issues, they cannot be viewed as definitive or as the end point. Rather, such studies provide building blocks on which investigations with actual victims of trauma can be initiated. Only when a sufficient body of studies cohere can we truly have confidence about how trauma does, or does not, affect memory.

Related to this, the nature of materials utilized to investigate memory needs to be carefully considered. The normative literature on basic memory provides an excellent point for beginning to determine how memory may operate similarly or differently in traumatized children. However, the fact that memory for standard lists of words utilized with normative populations is not affected by trauma does not mean that memory for emotionally laden words will operate similarly (Rieder & Cicchetti, 1989). Therefore, although beginning with standard memory paradigms allows comparisons with a large normative literature to be made, memory for traumatic material also must be investigated. In this regard, investigations of autobiographical memory may be particularly germane. However, to conduct such investigations, accurate information must be available on the actual event that occurred and on what information an individual recalled immediately following the event. The conduct of such investigations is likely to be particularly challenging and to pose logistic as well as ethical challenges. Immediately following an event, an individual may be involved in investigative and/or court proceedings, and interviews could potentially contaminate the accuracy of the recall of the event. Moreover, over time it might prove to be troubling to an individual to be queried about past trauma. Although utilization of court testimony from the victim and witnesses might provide a window into immediate recall, sensitivity in assessing memory over time is needed. Because it is critically important to enhance our knowledge of autobiographical memory in victims of trauma, the design and conduct of investigations in this arena hold much promise for informing our understanding of trauma and memory.

A number of issues exist in the memory and trauma literature that must be addressed if the field is to move forward in attaining a comprehensive understanding of this important area. A major omission in the extant literature on trauma and memory involves the absence of studies of extreme and chronic stress. It may be that in cases of extreme stress, an event is not stored. However, such a conclusion cannot be drawn in the absence of studies with highly stressed individuals.

The presence of co-occurring risk factors in samples of traumatized individuals has also contributed to difficulty in ascertaining exactly what may be operating to affect memory. For example, in populations who have experienced PTSD, the effects of the trauma on memory per se are confounded with issues such as hospitalization, utilization of medication, the provision of psychotherapy, substance use and abuse, and comorbid mental illness (e.g., depression). Sufficiently large samples with improved control over potentially confounding factors must be conducted to better parse the effects of trauma from other factors.

With the advent of neuroimaging technology, new avenues for examining brain structure and functioning in victims of trauma have emerged. Although such technologies are appealing and may help to elucidate how trauma affects the brain, caution also is in order. Technological advances cannot compensate for the absence of sound theory, careful experimental design, meticulous execution of empirical investigations, and rigorous scientific methodology. It is crucial that these conceptual and methodological issues be placed at the forefront of research on neuroimaging and memory in traumatized populations. Otherwise, it will be unclear whether neuroimaging findings reflect genuine differential physiological processes between traumatized and nontraumatized individuals, whether these processes are merely epiphenomena, or whether these physiological differences represent compensatory or adaptive neurobiological changes (Cicchetti, 2002; Curtis & Cicchetti, 2003; Peterson, 2003). As our understanding of basic memory processes increases, the incorporation of studies involving MRI and fMRI is more likely to yield fruitful results. To date, investigations of brain structure and memory processes have rarely been conducted in the same individuals who have experienced trauma. In the future, it is essential that increasing multiple-levels-of-analysis research on memory and brain structure and function be conducted with traumatized populations.

Although we are increasingly accruing knowledge about trauma and memory, clinical, social policy, and legal implications are inherent in how the questions posed in this chapter are answered. Therefore, a significant responsibility exists to build on current knowledge with normative populations and to design increasingly methodologically rigorous and theoretically sophisticated investigations of trauma and memory. For example, understanding how various types of trauma interact with developmental status to affect memory will be critical for suggesting what type of therapeutic intervention to utilize. Because scientific evidence does not support views that memories can be stored in the body preverbally, efforts to help young trauma vic-

tims recall an event that they, in truth, have no knowledge of could prove to be countertherapeutic. Because trauma may actually alter neuronal connections in the brain (Cicchetti, 2002; DeBellis, 2001), therapies that require victims to relive traumatic events might actually consolidate the maladaptive negative neuronal pathways that may be associated with the experience of trauma. The area of “repressed” memory is particularly salient, and much has been written about the perils inherent in therapists’ suggestion of trauma to patients who may not consciously recall (or who may not have experienced) such trauma (Loftus & Ketcham, 1994). Given our current state of knowledge regarding trauma and memory, unless children or adults seek help specifically to address their reactions to trauma, caution in exploring such events is indicated.

In the policy and legal arenas, we are beginning to attain evidence that memory in victims of trauma is certainly no worse than in the general population. Conversely, we also do not have reason to believe that memories of trauma are indelible or somehow enhanced, nor are they protected from reconstructive processes common to most, if not all, memories. Again, however, we urge caution, as the seminal studies that can shed light on these issues remain to be conducted.

In closing, we are embarking on a new era of investigations of trauma and memory that will involve actual victims of trauma and that will utilize both basic memory paradigms and those that are likely to be more emotionally arousing. We are guardedly optimistic that such investigations not only will enhance our understanding of the operation of memory in victims of trauma, but also will assist in refining and enhancing developmental theories of memory.

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