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Neurobiological Effects of Emotional and Sexual Child Abuse
as Contributors to Learning Disabilities

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Introduction

There are more than three million allegations of child abuse and neglect each year in the United States; of these allegations, about one million are substantiated (Teicher, 2002). In 1998 in Canada, there were about 135,000 child abuse investigations, or about 22 investigations for every 1,000 children (Trocme et al., 2001); abuse was substantiated in almost half of these cases.

It is not surprising that children who have been abused are at significantly increased risk for a host of developmental and psychiatric problems, including internalising problems such as anxiety, depression, suicidal ideation, and posttraumatic stress disorder, and externalising problems such as aggression, impulsiveness, delinquency, hyperactivity, and substance abuse (Becker et al., 1995; Teicher et al., 2006). It has been assumed that many of these problems are the extreme end result of psychological attempts to cope with abuse. Recently, however, there has been growing evidence that these problems may be a direct result of physical damage to the brain (Teicher, 2002; Weber & Reynolds, 2004; Streeck-Fischer & van der Kolk, 2000).

It has been assumed that this damage is caused by physical abuse (i.e., blows to the head). However, in a fascinating synthesis of the neurobiological and psychiatric research on child abuse, developmental neuropsychiatrist Martin Teicher makes a convincing case that the other all-too-common forms of child abuse—verbal, emotional, and sexual—can also lead to physical brain damage, and that this brain damage can lead directly to many of the psychiatric problems associated with child abuse (Teicher, 2000, 2002; Teicher et al., 2002, 2003; Teicher, Tomoda, & Andersen, 2006). This research shows that child abuse contributes directly to brain damage, not that brain damage increases a child's risk of being abused, or that a common brain pathology

running in families increases parents' likelihood of being abusive (Teicher, Tomoda, & Andersen, 2006).

Learning disabilities (LD) are among the conditions for which abused children are at increased risk. Some 30% of abused children have severe learning problems (Streeck-Fischer & van der Kolk 2000; Hughes & Di Brezzo, 1987), and almost as many suffer from attention-deficit/hyperactivity disorder (Ackerman, 1998). It is not surprising, therefore, that many abused children show poor academic achievement (Kinard, 2005; Navalta et al., 2006). Although few studies have shown direct links between emotional or sexual abuse and the development of LD, the brain changes caused by such abuse are similar to those that cause (or are at least implicated in) LD. In this paper, I will look at some of the abuse-related neurobiological changes that Teicher demonstrates and attempt to show how these changes can lead to learning disabilities.

Child abuse, limbic abnormality, and LD

In a study of 253 adult psychiatric inpatients, Teicher (2000) found that more than half of them reported having been physically and/or sexually abused. The patients who were physically abused reported a greater than average incidence of symptoms suggestive of temporolimbic disturbance and temporal lobe epilepsy (e.g., seizures, numbing, tingling, illusions and hallucinations, dissociation, depersonalisation, and intense bouts anger or sadness). The number of symptoms was even greater for those who had been sexually, but not physically, abused. Patients who had been abused before the age of 18, a time when the brain is still developing, had more symptoms than those who were abused after the age of 18 (although those who were both physically *and* sexually abused displayed more symptoms regardless of the time of abuse). These

findings were mirrored by EEG evidence showing abnormal brain wave patterns in 43% of patients with a history of emotional abuse, but only 27% of patients without a history of abuse.

The implications of temporolimbic disturbance for the development of LD may be significant. The temporolimbic system includes structures that are critical for learning: the hippocampus and mammillary bodies, which are involved in the storage of episodic memories (e.g., remembering a story); the thalamus, which is a key “relay station” for incoming sensory stimuli; and various regions of the temporal lobe, which are responsible for language reception and comprehension. Not surprisingly, children with a wide range of LD have problems with memory, language, and “sensory overload” (Roth Smith, 2004). Abnormalities in the brain regions involved Galante et al. (1972) and Smith et al. (1972) have shown a correlation between EEG abnormalities and reading disability, and Spreen, Risser, and Edgell (1995) have shown that EEG abnormalities occur more frequently in LD children than in normal learners, but tend to be nonspecific both in terms of type of LD and the locus of EEG abnormality. Because memory, language, and reading problems are common in abused children (Teicher, 2000; Navalta et al., 2006; Nolan et al., 2004; Oates, Peacock, & Forrest, 1984), and because these children have experienced brain damage in the temporolimbic structures that are key to learning, it is not implausible that child abuse leads to abnormalities in temporolimbic regions, which in turn lead to various learning problems.

Child abuse, left hemisphere deficiencies, and LD

Children who have been emotionally or sexually abused show marked abnormalities in their left brain hemispheres. In these children, left-sided EEG abnormalities are more than twice as common as right-sided EEG abnormalities. Among those who were emotionally abused, all of

the EEG abnormalities were on the left. Neuropsychological tests showed that in those with a history of psychological abuse, left-hemisphere neurological deficits—such as deficiencies in verbal performance—were six times as prevalent as right-hemisphere deficits (such as poor visual-spatial ability). Among those who were not abused, the prevalence of left-sided and right-sided neurological deficits were roughly equal.

EEG coherence studies—which measure not just brain function, but also brain structure—have shown similar patterns. In one group of psychiatric patients with a history of intense physical or sexual abuse (and no prior history of neurological disorders or abnormal intelligence), the left cerebral cortex was less developed than that of healthy controls (Teicher 2000; Teicher et al., 2002, 2003). More interestingly, the right hemisphere of the abused patients was more developed than the left. Cortical damage was present throughout the left hemisphere of the abused patients, but was more pronounced in the temporal regions, including the superior temporal gyrus and the hippocampus (Teicher, Tomoda, & Andersen, 2006). The hippocampus is particularly susceptible to damage because it is one of the few to continue growing after birth (Bremne & Vermetten, 2001). In particular, it is sensitive to damage by stress hormones such as cortisol, which are released in great quantity during times of extreme stress. Teicher (2000) cites studies showing reduced hippocampal volumes (sometimes as much as 15%) in people with a history of abuse, proportionate to the severity of abuse.

The implications for LD are significant, considering that many left hemisphere functions—including verbal memory, language reception and expression, and analytical functions—are depressed when the left hemisphere (especially the left temporal region) is damaged. This dovetails with the finding that the left cerebral hemisphere of LD children is

smaller than their right hemisphere (the opposite of that seen in children without LD) and that one pattern common to many LD children is over-use of the right hemisphere and under-use of the left (Roth Smith, 2004). Children exhibiting this pattern have problems reading, spelling, paying attention to and memorising facts, doing math calculations, writing essays, sequencing thoughts, and engaging in social conversation (Roth Smith, 2004). Furthermore, as Budson and Price (2005) remind us, damage to the left hippocampus can also lead to problems with episodic memory, and damage to other temporal regions can lead to problems with semantic (factual) memory. It is not surprising that all of the abused patients who showed abnormalities in the EEG coherence studies had lower verbal memory scores than non-abused controls (Teicher 2000).

A review by Shaywitz et al. (2000) shows that part of the neurological signature of dyslexia is depressed activation of numerous left-hemisphere regions, including the left occipital, left inferior parietal, and left middle temporal regions. Simos et al. (2005) report similar left-hemisphere deficits in dyslexia, and Brosnan (2002) further implicates under-activation of the left frontal cortex. This is consistent with the findings of Bakker (1979), who used visually evoked potentials to show that one type of dyslexia—P-type—involves an over-development and over-use of the right hemisphere and an under-development and under-use of the left hemisphere. P-type dyslexics read slowly because they focus too much on the perceptual features of words and not enough on the sound and meaning of words—an ability mediated by the left hemisphere.

Regardless of the type of LD, it is believed that over-reliance on the relatively well-developed right-hemisphere causes children to focus too much on wholes (the way words look, main ideas) and not on individual, fine-grained analysis. As Roth Smith (1994) says, these children “can’t see the trees for the forest” (p. 66). To the extent that dyslexia and other learning

problems appear to be related to brain abnormalities similar to those caused by emotional and sexual abuse, it may well be that child abuse can directly contribute to the development of LD (see Anyanwu & Campbell, 2001; Oates, Peacock, & Forrest, 1984).

The development of left-sided cognitive problems in abused children depends in part on the time of abuse. For instance, the likelihood of damage to left hemisphere structures is increased if abuse occurs when that hemisphere is in the period of most rapid development: 2-10 years (Teicher, 2000). Considering that this is the time that verbal language is in rapid development, abuse that happens in this window is likely to increase the risk of verbal language deficits. If abuse happens earlier, the implication for language development may be less dire, because it is mainly before age 2 that a child's right hemisphere can successfully take over many of the left hemisphere's functions if that hemisphere is damaged (e.g., Lenneberg, 1967). LD children who have left-hemisphere deficits may try to compensate for poor academic performance by recruiting their frontal lobe (Roth Smith, 1994), but this may not be possible for children whose LD is caused by child abuse because, as will be discussed next, their left frontal lobe is not spared from damage (Teicher 2000; Teicher et al., 2002, 2003).

Child abuse, frontal cortical damage, and LD

The frontal lobe, particularly the prefrontal cortex, is responsible for executive functions such as planning, organising, articulating, focusing attention, and controlling impulsivity. Damage to this region can lead to deficits in any of these executive functions (Rutter, 1999), all of which are essential for learning (Duff et al., 2005). Based on converging evidence, a direct connection may exist between child abuse and the development of LD. Abused children evidence damage to the frontal lobe (at least on the left; Teicher 2000, 2002; Teicher et al., 2002,

2003) and also show deficits in executive functioning (Rutter, 1999; Navalta et al., 2006). Such deficits are also seen in LD children (Roth Smith, 2004), who similarly show decreased activation of the frontal lobe (Hechtman, 1991).

One of the key problems associated with frontal lobe damage is the inability to develop object constancy and mental maps, which means that abused children cannot form verbal and conceptual representations of their inner world and their surroundings and, hence, are unable to plan (Streeck-Fischer & van der Folk, 2000). Furthermore, because these children have grown up in unpredictable environments, they are hyper-aroused—always vigilant for signs of future abuse, and terrified of novel stimuli. Thus, they are unable to calm themselves, separate relevant from irrelevant information, explore their environments, and handle new information (Streeck-Fischer & van der Kolk, 2000; also Teicher, 2006, on how abuse-induced amygdaloid abnormalities can also lead to these problems). They also have trouble with perspective-taking (Barahal, Waterman, & Martin, 1981), another function mediated by the frontal lobe. It is obvious that deficits in any of these areas can lead to learning problems.

The frontal lobe is also responsible for encoding episodic memories (Bruce & Price, 2005) and damage to these regions can lead to distorted or false memories, in which children recall the incorrect source, context, or sequence of information or events. Furthermore, all forms of working memory are heavily reliant on the frontal lobe (e.g., Newman et al., 2004). Because abused children have lower verbal memory scores than non-abused children (Teicher, 2000) and because some children with LD have difficulties with working memory (e.g., Bayliss et al., 1995), it may be that child abuse can directly lead to memory problems and associated LD.

Child abuse, deficient hemispheric integration, and LD

In people with a history of abuse, the corpus callosum—a major structure connecting both cerebral hemispheres—is significantly smaller than in people without a history of abuse (Teicher 2000; Teicher et al., 2002, 2003). A well-functioning corpus callosum is essential for successful learning because it ensures the smooth flow of information from one hemisphere to the other. When the hemispheres communicate well, people are able to appraise ideas and situations in both global and fine-tuned ways; they can, in other words, appreciate both the forest and the trees. Furthermore, bilateral activation of the frontal lobe makes difficult tasks involving working memory much easier (Budson & Price, 2005).

Damage to the corpus callosum may explain why people with a history of abuse have problems integrating information. An interesting finding is that people with a history of abuse use predominantly their left hemisphere when recalling neutral memories and their right hemisphere when recalling negative memories (Teicher 2000; Teicher et al., 2002, 2003). Non-abused people show more bilateral activation when recalling both neutral and negative memories. This problem with integration is also evident in people with LD, in which one side of the brain is used more than the other (Roth Smith, 1994). It might be hypothesised that the unilateral brain activation seen in dyslexia (Shaywitz, 2000; Simos, 2005; Brosnan, 2002) is due, in part, to problems with the corpus callosum (Hynd et al., 1995). The same could be said for attention-deficit/hyperactivity disorder, which has been linked to decreased size of this structure (Tannock, 1998). To the extent that damage to this key structure leads to problems similar to those seen in people with LD, it might be hypothesised that abuse-related brain damage can lead to LD.

Child abuse, abnormal cerebellar vermis activity, and LD

The cerebellar vermis is a strip of the cerebellum, a key region of the hindbrain. We usually think of this region as involved in motor co-ordination, but through its effects on dopamine and norepinephrine, it also seems to be implicated in limbic activation and attentional and emotional regulation (Teicher 2000; Teicher et al., 2002, 2003). In those who have been emotionally or sexually abused, the vermis appears less able to regulate emotion and attention (Teicher, 2000; Teicher et al., 2002, 2003). This may partially account for the high rate of ADHD (30%) and limbic symptoms among abused people (Teicher, 2000; Teicher, Tomoda, & Andersen, 2006) and among those with LD (Roth Smith, 2004). Like the hippocampus, the vermis develops slowly, continues growing after birth, and is extremely sensitive to the damaging effects of stress hormones like cortisol.

Cause and effect

A common criticism of the kind of neurobiological studies mentioned in this paper is that they only show correlations between abuse and cognitive deficits, not cause and effect relationships. For example, could it not be that children with cognitive deficits are at increased risk of being abused? Perhaps the impoverished social environments that children find themselves in are just as likely to contribute to cognitive deficits as is the abuse that is likely to be engendered by such environments. One could also ask whether damage to regions like the hippocampus could be due not to abuse *per se*, but, rather, to consequences of psychopathology resulting from the abuse. Furthermore, one might ask whether pre-existing brain damage can increase the risk for psychopathology and cognitive deficits, independent of the effects of abuse.

Teicher, Tomoda, & Andersen (2006) respond to all of these criticisms by referring to animal studies. To control for the effects of SES and other psychosocial factors, they exposed rats, mice, and monkeys to maternal deprivation (an analogue for emotional abuse). They found that these animals exhibited the same kind of EEG abnormalities and brain damage seen in human subjects, and the damage was closely linked to the precise time when the deprivation occurred. Thus, it was abuse *per se*, not other factors—and certainly not years of abuse-related psychopathology—that directly led to brain damage.

Another issue relates to whether the LD-related symptoms mentioned in this paper might not represent specific LD but, rather, the non-specific effects of other psychiatric conditions. For instance, people with a history of abuse may have trouble concentrating in school not because they have sustained damage to the frontal lobe, but because their abuse has triggered a clinical depression. Two observations suggest this may not be the case. First, there are reports of specific LD symptoms in students who have experienced child abuse but who are otherwise healthy and have no comorbid conditions (e.g., Navalta et al., 2006). Second, many types of LD, especially dyslexia, have a specific neurological signature (Shaywitz et al., 2000) that corresponds frequently with the specific neurological signature of child abuse (limbic dysfunction, left-sided abnormalities, and hemispheric disconnection). If the cognitive dysfunctions seen in people with a history of abuse were merely incidental, non-specific effects of a range of psychiatric disorders, we would not expect to see this frequent a correspondence.

Conclusion

Child abuse is a significant problem in North America. In addition to the devastating psychological toll it leaves, it puts children at increased risk for developing all sorts of cognitive and learning problems. It used to be thought that these problems were due mainly to the psychological impact of abuse, but new research is showing that these problems are often due specific abuse-related brain damage. More interestingly, it is not just physical abuse, but also emotional and sexual abuse, that can cause such brain damage.

This paper has tried to show a connection between child abuse, specific brain damage, and the development of learning disabilities. Although it is certainly true that LD children are at increased risk for abuse (e.g., Brown, 1999), converging evidence from human and animal studies has suggested that abuse itself—by causing brain damage—can increase the risk for developing LD. If this connection holds true, we have more reason than ever to be alarmed at the number of children who are abused each year. Furthermore, because the effects of child abuse are not just psychological, we cannot rest assured in thinking that abused children will simply be able to “get over it.” Once experience etches itself into the brain, it may be impossible to undo the damage.

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