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ESSAY REVIEW

Limits on plasticity

Nelson & Luciana (Eds.) *Handbook of developmental cognitive neuroscience* (2001)

Anderson, Northam, Hendy & Wrennall, *Developmental Neuropsychology: A Clinical Approach* (2001)

Bradshaw, *Developmental Disorders of the Frontostriatal System: Neuropsychological, neuropsychiatric, and evolutionary perspectives* (2001)

Huttenlocher, *Neural Plasticity: The effects of environment on the development of the cerebral cortex* (2002)

Michael S. C. Thomas
Neurocognitive Development Unit, Institute of Child Health, London

The more scientists learn about the brain, the more we learn about its infinite plasticity. … If there is a challenge to one part of the brain, we can learn to revive other parts to make all sections of the mind work together. (Elaine Colliar, "mind-mapping" champion, on how a visualization technique might be used to overcome reading impairments in developmental dyslexia; Kenyon, 2002)

Requests for reprints should be sent to Michael Thomas, School of Psychology, Birkbeck College, University of London, Malet St., London WC1E 7HX UK. E-mail: m.thomas@psychology.bbk.ac.uk

> We should encourage the public's interest in developmental brain science and applaud attempts to base early childhood policy and practice on a scientific basis. However, in some instances, public enthusiasm far outstrips our scientific understanding. Too often the messages broadcast by advocates and the media do not accurately reflect what scientists currently know about synapses, critical periods, neural plasticity, and how experience affects the brain. (Bruer, 2001, p. XXX)

HOW INFINITE IS THE PLASTICITY?

So what do scientists currently know about the plasticity of the brain? Four recent books provide a timely window on the current state of scientific knowledge. *The handbook of developmental cognitive neuroscience* (HDCN; edited by Nelson & Luciana, 2001) reports back from the frontier between biological and behavioral science and comprises 41 chapters from contributors on topics covering basic neurobiology, systems neuroscience, imaging, and cognitive science. *Neural plasticity* (Huttenlocher, 2002) provides an overview of the effects of the environment on brain development and brain function from embryo to adulthood, focusing on mechanisms underlying plasticity at various ages and across various domains, including sensory, motor, language, and executive systems. *Developmental neuropsychology* (Anderson, Northam, Hendy, & Wrennall, 2001), on the other hand, provides a clinical perspective on disorders resulting from acquired brain damage in children, and on the subsequent prospects for recovery. Lastly, *Developmental disorders of the frontostriatal system* (Bradshaw, 2001) focuses on the relation among six major neurodevelopmental disorders, each of which is characterized (at least in part) by executive dysfunction in the failure to initiate or control behavior. Together, these excellent books sketch a picture of how the normal cognitive system develops and how it responds to acquired damage (in childhood or adulthood) or to developmental damage.

Plasticity can be defined as the adjustment of the nervous system to changes in the external milieu (through sensory inputs) or internal milieu (through the effects of damage to the system) and appears to be mainly a property of the cerebral cortex rather than subcortical structures (Huttenlocher, 2002). Before I picked up these books, I was aware of two quite different stories sometimes told about plasticity. One story runs as follows. The cognitive systems in the brain are specified by an innate blueprint, which unfolds over developmental time. There are critical periods in which these cognitive structures must be established, and once the components are fixed in place, flexibility is over. Damage one component at that point, and your only option is to adopt compensatory behavioral strategies to work around the deficit. This is the type of story that is often associated with evolutionary psychologists, where evolution specifies the cognitive components in the blueprint (see,

e.g., Cosmides & Tooby, 1994; Pinker, 1997). One of its main claims is that plasticity is limited (see Lenneberg, 1967).

In contrast, the other story views the brain as permanently plastic. Functionally specialized structures emerge across development through an interaction of the individual with the environment. Specialization is "seeded" via weak initial biases of connectivity and computational preference of the underlying substrate. However, even when specialization is complete, the system retains hidden reserves available at any age to learn new skills or to compensate for damage, so long as the correct behavioral interventions are used. This type of story, with its spare capacity and lifelong plasticity, would offer more hope to those who have experienced brain damage, or those wanting to acquire new skills later in life.

Which story is true? Let us start by considering the development of the normal cognitive system.

ASSESSING LIMITS TO PLASTICITY IN NORMAL DEVELOPMENT

All four books accept the consensus that there is significant functional specialization in the brains of healthy adults, as evidenced by patterns of specific deficits found in some adults with acquired brain damage. Moreover, at a gross level, there appears to be a fair degree of consistency across individuals about which areas of the brain end up subserving which functions. For instance, Bates and Roe (2001 [HDCN]) tell us that the privileged status of the left hemisphere for some aspects of language processing is now beyond dispute, citing estimates of left hemisphere specialization at between 95% and 98% of healthy adults, irrespective of handedness.

The question is, where does the adult structure come from? In the context of language processing, Bates and Roe (2001 [HDCN]) review three possible theoretical accounts. *Equipotentiality* argues that both sides of the brain are the same at birth, and each is able to acquire the relevant computations to subserve the domain of language. *Irreversible determinism* argues that the left hemisphere is innately and irreversibly specialized for language. The *emergentist* view proposes that the two hemispheres are characterized at birth by innate but "soft" biases in information processing, biases that are relevant but not specific to language processing. Specialization of one or other hemisphere to language is then an emergent process. Bates and Roe conclude that evidence increasingly favors this third option (see also Elman et al., 1996; Johnson, 1997, 1999).

Two examples illustrate the type of empirical evidence accumulating in favor of the emergentist view. Casey, Thomas, and McCandliss (2001 [HDCN]) describe recent work using MRI. In one study, the prefrontal activity generated by a go/no-go task was compared in children and adults. Differences were found in

dorsolateral prefrontal cortex, with children activating larger volumes of this region than adults. Children with the largest areas of activation were not those who found the task hardest, suggesting that task difficulty was not responsible for the differences. Casey et al. conclude that the results are most consistent with increased selectivity of function in prefrontal cortex with age. De Haan (2001 [HDCN]) reviews electrophysiological imaging work on the development of face recognition in infants. Based on patterns of lateralization (present in adults but not infants) and specificity (present in adults, e.g., human faces vs. monkey faces, but not present in infants), De Haan concludes that cortical specialization for face processing observed in adults is achieved through a gradual specialization of an initially more general-purpose system.

It should be noted, however, that the emergentist story will not be a simple one. For instance, the emergence of functional structure is not necessarily uniform across domains. Different functions may emerge or come online at different times. For instance, frontal areas, although offering some early limited function in infants (Johnson, 1997), are nevertheless much later in maturing to their adult function than many other cognitive abilities (e.g., Benes, 2001 [HDCN]; Bradshaw, 2001; Diamond, 2001 [HDCN]; Huttenlocher, 2002; Johnson, 1997). As a consequence of developing at different rates, different brain systems may then vary in the extent to which their structure is open to shaping by the environment. Moreover, if we accept the emergentist view as the correct one, further questions need to be answered. Just how different is this process from a preprogrammed unfolding? Once it has emerged, is the structure fixed and resistant to further influence from the environment?

The first question can be assessed by the impact of variations in the environment on cognitive development. If the emergence of cognitive structures can be significantly disrupted by atypical environments, the process represents more than a simple unfolding. Several authors explore differences in neurophysiological environments, for instance, the effect of nutrition on cognitive development (Georgieff & Rao, 2001 [HDCN]); or the case of prenatal substance abuse in mothers (e.g., alcohol, see Streissguth & Connor, 2001 [HDCN]; cocaine, see Stanwood & Levitt, 2001 [HDCN]). Huttenlocher (2002) argues that the early stages of brain development are closer to the unfolding of a program, whereby environmental influences during embryonic and fetal development can only have negative effects on this process, as in the case of substance abuse during pregnancy. This initial program is attempting to put the right number of neurons in the right places and establish the approximate initial input and output connections for different regions.

However, from the point of view of cognition, we are more interested in environments that are atypical in the information that they provide. In cerebral cortex, Huttenlocher (2002) suggests that the function of a given cortical region appears to be largely determined by its inputs and outputs, and, therefore, changes

in environmental input might well have major effects on the functioning of a system. This hypothesis can be assessed by the case of deprivation in infancy and early childhood.

Children born with bilateral visual deprivation due to congenital cataracts can develop normal levels of visual acuity and eye alignment so long as the cataracts are removed prior to the age of 8 weeks. Huttenlocher (2002) takes these data to suggest that during the first 8 weeks of life, the development of the visual cortex progresses almost normally in the absence of exposure to formed visual images. Moreover, evidence from premature infants indicates that additional early input has little effect on the development of visual cortex. However, subsequent to this initial early period, visual input becomes essential to the development and maintenance of normal function—environmental effects are essential even for very basic functions such as stereopsis and the processing of shape and movement. Evidence from deprivation studies with cats suggests that once environmental input becomes important, different functional circuits may then be more or less environmentally regulated. For example, the parvocellular system for object recognition appears to be more affected by deprivation of visual forms and have more extended plasticity than the magnocellular system for object location (Horton & Hocking, 1996; LeVay, Wiesel, & Hubel, 1980).

A slightly different picture emerges of the motor system. Huttenlocher (2002) reviews research on monkeys raised in apparatus that prevented them from walking or seeing their limbs (Held & Bauer, 1967). When removed from constraints at the age of 65 days, the monkeys quickly began to walk and were doing so normally after a week. Walking, then, emerges without specific training, consistent with a fixed developmental program. A similar result was found in cats with regard to the development of the normal foot-placing reaction on approach to a horizontal surface (Hein & Held, 1967). This motor response also appears to develop independently of prior experience. However, in this study, more complex behaviors, such as reaching and avoidance of a discontinuity in a surface, developed slowly after removal of constraints and required experience and practice. And the newly walking monkeys exhibited transient deficits in visually guided motor behaviors.

The implications here are twofold. First, the effects of deprivation differ between systems. Second, the greater the complexity of processing, the more important the environmental input appears to be.

Another set of studies sheds light on the effects of environmental deprivation on higher cognitive functions, this time in the context of orphanage-reared children in stimulation-poor environments, as reviewed by Gunnar (2001 [HDCN]). Interestingly, even when adequate nutrition is provided, human infants raised in environments with severely limited opportunities for active engagement and productive interaction subsequently experience massive delays in both physical and behavioral development (see also Kolb & Gibb, 2001 [HDCN], and Nelson, 1999, for

discussion of equivalent animal studies). Normal brain development is evidently reliant on activity-dependent processes, where active engagement of the infant with the environment is essential for some aspects of neural development to occur. However, studies of orphanage-reared children also indicate a wide window during which transition to an enriched environment can produce catch-up growth in these children. Despite marked delays in cognitive development at adoption, the majority of children in one follow-up study were found to be functioning intellectually within the normal range after several years in "enriched" middle-class family environments (Rutter, 1998). Almost all aspects of cognitive development could be caught up after early deprivation. There was one notable exception, which we will leave for later.

The second question provoked by the emergentist view is the degree to which adult structures, once emerged, are fixed and inflexible. If there was plasticity in building the structures, does it remain? The received wisdom here is that (a) with a lot of practice, the adult system can be tinkered with, but (b) its overall structure is reasonably fixed. Elbert, Heim, and Rockstroh (2001 [HDCN]) review the evidence for use-dependent plasticity on adult cortical structures. In particular, they examine the reorganization of cortical sensory maps under various atypical circumstances. These include intensive limb use (leading to expansion of the cortical area dedicated to this limb), prolonged disuse after limb loss (resulting in invasion of the relevant area from nearby sites on the map), synchronous use of neighboring sites, such as adjacent fingers (fusion of areas), and subsequent asynchronous use (segregation of areas). It is important to note that these plastic changes were sometimes maladaptive, for instance leading to phantom limb pain in amputees and loss of digit function in violinists.

These results are consistent with continuing plasticity in the somatosensory system at least. There is evidence of continuing plasticity elsewhere. Indeed studies have suggested that even those abilities commonly thought to be fixed past a certain critical period in development can nevertheless be modified in adults with intense practice. For instance, the ability of adults to discriminate phoneme contrasts in foreign languages, lost at 12 months of age, can be regained, as shown by Japanese speakers who can learn to produce the English /l/–/r/ distinction after perceptual training (Bradlow, Pisoni, Akahane-Yamada, & Tohkura, 1997). And Elbert et al. (2001) comment that accents thought to be fixed past adolescence can also be altered, as demonstrated by actors who lose their accents in the course of their intensive training in drama school, or individuals who acquire a second language after adolescence without a foreign accent (Bongaerts, 1999).

Despite these indications of adult plasticity, Elbert et al. (2001) conclude that the capacity for reorganization is greater in children than in adults, evidenced by the extended practice that adults require to produce changes. Huttenlocher (2002) con-

cludes from his review that whenever developmental and adult plasticity have been compared on a given task, adult plasticity has usually been found to be less and never greater than in the developing brain. However, Elbert et al. also stress the importance of positive motivation in the individual, and that experience must be behaviorally relevant to the individual to generate use-dependent adaptations of cortical representations. Passive stimulation alone does not trigger reorganization—the stimulation must be relevant to the production of some behavior of interest to the organism. Such a factor may itself differ between the developmental and the adult case. In sum, the received wisdom on adult plasticity does not seem too far wrong.

The current view of normal cognitive development, then, suggests that specialized cognitive functions emerge through development, but this process depends on active engagement with a rich environment and produces a structure that is, to a lesser extent, still plastic in the end state. Once one introduces flexibility into the developmental process, however, it seems necessary to explain how adults could end up showing apparent uniformity in their areas of brain specialization (of which the left lateralization of some aspects of language is but one example). There are two possible sources for this uniformity. First, it may arise if development is experience-dependent, but everybody has very similar early experiences. Second, it may arise if there are reasonably strong neurocomputational constraints that guide the developing system (what Bates & Roe, 2001 [HDCN] call the "relevance" of certain neural areas to certain cognitive functions). The first of these possibilities emphasizes plasticity in the system (because a radically different environment would presumably lead to a quite different functional structures), whereas the second possibility plays down plasticity (because experience drives the system along developmental pathways that have been channeled by the system's neurocomputational parameters). (See Greenough, Black, & Wallace, 1987, for an equivalent distinction between "experience-expectant" and "experience-dependent" systems.)

To get an idea of which of these two possibilities is the more likely, we need to look beyond normal development to how the system responds to disruption. Examining the impact on brain specialization of exposure to radically different environments is one possible approach. Alternatively, we can look at systems where the initial neurocomputational parameters are different, as in the case of developmental disorders (Thomas & Karmiloff-Smith, 2002). Or we can look at normal systems damaged early in development and examine their ability to recover through reorganization. This will allow us to gauge the relevance of the neurocomputational parameters in different brain areas to their normal cognitive function. If an area can learn to subserve Function A normally, but Function B (or perhaps A and B) after early damage, then its computational properties cannot have been solely relevant to A. In this case, we would move closer to a position of weaker constraints, greater plasticity, and equipotentiality.

ASSESSING LIMITS TO PLASTICITY VIA RECOVERY FROM EARLY BRAIN DAMAGE

In *Developmental neuropsychology,* Anderson et al. (2001) review the impact of several forms of brain damage in infants and children. These include traumatic brain injury, hydrocephalus, spina bifida, cerebral infections such as meningitis, endocrine and metabolic disorders, and childhood epilepsy. The authors also discuss a theoretical debate of particular relevance to our concerns. This debate contrasts two views of the prognosis for recovery after early brain damage: plasticity versus early vulnerability.

As we have seen, there is a general view that the child's brain has greater plasticity than the adult brain. One might assume, therefore, that the younger brain is more able to recover from early damage and, in particular, if the emergentism view is correct, is able to offer compensatory reorganization and transfer of function to the remaining parts of the system. There is evidence to support this idea. Children with early left hemisphere damage can go on to acquire many age-appropriate language abilities, whereas similar lesions in adults produce obvious patterns of aphasia (see Bates & Roe, 2001 [HDCN] for review). Even if an entire hemisphere is removed (for instance in the treatment of severe epilepsy), children can nevertheless develop relatively normal cognitive function (Basser, 1962). This apparent early plasticity led some of the first researchers in the nascent field of child neuropsychology (Kennard, 1936, 1940; Teuber, 1962) to propose the Kennard Principle, interpreted by Teuber to suggest that "if you're going to have brain damage, have it early" (Schneider, 1979).

However, Anderson et al. (2001) suggest that this view may have been excessively optimistic. Pediatric research has indicated that the same dose-response relation found in adult populations also holds in cases of child brain damage. The more severe the cerebral pathology, the greater the resulting neuropsychological impairment. Indeed, children with generalized cerebral insult (e.g., from traumatic brain injury) exhibit both slower recovery and poorer outcome than do adults who suffer similar insults. This is quite inconsistent with notions of greater early plasticity. From the "early vulnerability" perspective, short-term recovery from cerebral insult may be the same across age, whereas long-term recovery favors the mature brain. Across time, a child who has seemed initially to recover well from the insult may start to increasingly lag behind age-matched peers and fail to show the expected emergence of new cognitive skills. The child thus appears to "grow into" his or her cognitive deficit as the brain matures. From this perspective, the development of attention, memory, and learning skills is seen as vulnerable to early disruption. Without these capacities, the child does not have the tools to efficiently acquire new abilities and cannot progress along the normal pathway of cognitive development. An adult experiencing the same damage, however, already has the relevant abilities in place and only loses those directly associated with the area of damage.

In their book, Anderson et al. (2001) show how these apparently conflicting positions can be reconciled by considering the nature and severity of insult, age at time of insult, and other factors such as gender and the psychosocial context. Broadly, plasticity is associated with focal lesions. Small focal lesions are associated with good recovery. Large focal lesions may also be associated with good recovery, so long as they are unilateral, for then interhemispheric transfer of function can occur (although often with a general reduction in performance as additional functions are crowded into a single hemisphere). For moderately sized or bilateral lesions or both, functional plasticity is generally not evident and outcome is typically poor. Finally, when there is diffuse or general damage, plasticity is not evident, and the results are almost always poor, suggesting global dysfunction. The dose-response relation is evident, with greater damage leading to worse outcome.

In terms of age, the exact picture is still unclear. Bates and Roe (2001 [HDCN]), for instance, admit that "the shape of the [age-related] function governing loss of plasticity in humans is still entirely unknown, and it may not even be monotonic" (p. 297). Thus Anderson et al. (2001) suggest that prenatal injury may actually have the poorest outcome, with no evidence of transfer of function from the damaged site to intact tissue. On the other hand, plasticity may be greater in early childhood, leading to abnormal cortical organization and greater sparing of function. By late adolescence and adulthood, as patterns of adult deficits reveal, such plasticity becomes increasingly less evident. Both Anderson et al. and Bates and Roe discuss this U-shaped effect of damage in detail with regard to the impact of left hemisphere damage on language development. Reflecting the lack of consensus in the field, we find the former supporting the notion and the latter expressing skepticism.

Animal models suggest that the relation between age and damage will indeed be a complex one. For instance, Kolb and Gibb (2001 [HDCN]) report a study looking at reaching behavior in rats after frontal lesions were performed at different developmental ages. Rats with lesions in adulthood or on Postnatal Day 1 were severely impaired in the reaching task. Rats with lesions on Postnatal Days 5 and 10 were progressively better, but rats experiencing a prenatal lesion on Embryonic Day 18 performed as well as normal control animals. Kolb and Gibb conclude that the effects of damage vary qualitatively with the developmental events occurring at the time of injury. Recovery in rats was poor if the cortex was damaged immediately following the completion of neurogenesis, but good if injury occurred during the time of maximum synapse formation. Based on their findings with rats, Kolb and Gibb predict that the worst time for injury in the human brain would be the third trimester, with relatively good compensation for injury during the second trimester. This prediction remains to be evaluated for healthy human development. But as we shall see later, it does not seem to be supported by current views on the causes of autism and schizophrenia.

Nevertheless, the importance of timing in early brain development in humans is emphasized by the existence of the multiple "vulnerable periods" for different neg-

ative environmental events (Huttenlocher, 2002). Thus Huttenlocher details how a deficiency in folic acid occurring in the first weeks of pregnancy can lead to spina bifida (a defect in the development of the neural tube, the structure that normally differentiates into brain and spinal cord). By contrast, exposure to ionizing radiation can disrupt neurogenesis if it occurs between 7 and 20 weeks postconception, causing a decrease in the number of neurons produced. And maternal carbon monoxide poisoning can disrupt neuronal migration if it occurs between 17 and 20 weeks postconception. In short, during early brain development, the type of disruption caused by a negative environment clearly depends on the time that the event occurs.

Beyond timing, two further factors predict recovery from acquired damage in childhood. One is gender. Although Anderson et al. (2001) admit that the evidence is controversial, hormonal factors appear to affect brain development, with the female brain developing more rapidly, perhaps with more diffuse organization allowing greater potential for plasticity and reorganization of function (Kolb, 1995; Strauss, Wada, & Hunter, 1992). Animal studies also support the idea that hormonal factors can affect plasticity and recovery after injury, but here results indicated that effects were domain dependent. Thus, Kolb and Gibb (2001 [HDCN]) comment that after early medial frontal lesions, male rats showed enhanced recovery of spatial-navigation behaviors, whereas females showed enhanced recovery of skilled forelimb reaching.

The final factor is perhaps the most interesting. This is the effect that the environment itself has on recovery. Anderson et al. (2001) conclude that family function, socioeconomic status, access to rehabilitation, and response to disability all play a major role in recovery. So powerful is this effect that, although severity of damage is of primary importance immediately postinsult, in the long term it is environmental factors that are more important in predicting recovery from acquired brain damage. Once more, this finding is supported by animal studies, which show that manipulation of postinjury environment influences subsequent learning capacities (e.g., Kolb, 1995).

Anderson et al.'s (2001) analysis of the factors predicting recovery suggests a modification of the Kennard Principle. Instead of "If you're going to have brain damage, have it early," one might propose the following: "If you're going to have brain damage, have as little of it as possible. Have it early, and have it on just one side. Be a girl, and come from a supportive family which lives near a good hospital."

How Strong Are Constraints on Plasticity?

Let us consider three different cognitive domains where children have experienced early damage and subsequently shown some degree of recovery across development. First, however, it is important to clarify what we mean by "recovery." Again,

Anderson et al. (2001) provide some relevant distinctions. Two types of mechanism of recovery have been proposed, restitution and substitution. In restitution, spontaneous recovery occurs after brain injury, as the damaged tissue heals and pathways are reactivated. One such process, which also affects undamaged tissue, involves diaschisis. In diaschisis, functions far from the site of the insult are temporarily closed down after injury. Recovery occurs when these remote functions open up again. Subsequently, restitution may come in the form of the regeneration of damaged neurons and their axons; in terms of sprouting, where remaining nerve fibers develop branches that occupy sites left empty by damaged neurons; and in terms of denervation supersensitivity, where postsynaptic processes may become supersensitive to residual neurotransmitter signals. In substitution, behavioral functions are restored either by internal anatomical reorganization, where different neural areas take over the function of the lost tissue, or by behavioral compensation, where new external behavioral strategies are adopted to work around the functions impaired by damage. It is important to keep these distinctions in mind, because only some types of recovery will bear on the inherent plasticity and equipotentiality of the brain.

Our three domains are language processing, face processing, and spatial cognition. Bates and Roe (2001 [HDCN]) review evidence of language development in children with unilateral brain injury. Bearing in mind that at least 19 out of 20 adults show left hemisphere specialization, several studies are reviewed that assess the continuing impact on language performance of initial damage either to the left or the right hemisphere. Broadly, these studies demonstrate that plastic reorganization takes place when damage occurs prior to 5 to 7 years of age, such that when tested later, these children show little if any language impairment. More important, there are no effects of side of damage (or if such effects exist, they are subtle—see Huttenlocher, 2002, p. 135). Subsequent work is described that examines the first stages of language development in children who have experienced early unilateral damage. Almost all the brain-injured children exhibited delays in first-word production, regardless of lesion side or site. However, such delays tended to be more severe in children with left posterior (and particularly temporal) damage. Bates and Roe interpret these data as suggesting that it is hard to get language development "off the ground" after significant damage to either hemisphere. However, they puzzle over why left posterior damage should be associated with additional expressive deficits in very young children, when expressive deficits in adults are associated with left frontal damage. Their solution is that the early computational constraint that pulls language toward the left hemisphere is the ability of left temporal areas to provide fine perceptual analysis aiding word recognition. This ability subsequently drives both word comprehension and production. However, Bates and Roe maintain that this fine-detail bias is not necessarily specific to language, or even to audition. And the computational bias represents no more than a "soft" constraint, such that plasticity after

damage permits the function to be subserved by other circuits in the right hemisphere, although perhaps not quite as efficiently.

De Haan (2001 [HDCN]) examines the case of face processing. In adults, face processing is specialized to the right hemisphere and can show dissociations from object processing after acquired damage (e.g., McNeil & Warrington, 1993). De Haan reviews the work of Mancini and colleagues, who examined the effects of perinatal unilateral lesions on later face-processing abilities in children ranging in age from 5 to 14 years. The results indicated that the effects of the lesions were mild. Less than half the children exhibited impairments in face or object recognition compared with controls. Moreover, following a right hemisphere lesion, face-processing deficits were no more common than object-processing deficits, and a face-processing deficit never occurred in the absence of an object-processing deficit. Once more, plasticity appears to allow early damage to be overcome, but note here that the specialization of face processing from object processing appears to be purely a product of development. The dissociation of face and object recognition in the adult cannot be replicated by early damage to the normal system.

Stiles (2001 [HDCN]) explores the development of spatial cognition. In the adult case, left posterior and right posterior focal brain damage produce different deficits in spatial analytic function. Left posterior damage disrupts local processing and results in disorders involving difficulty defining the parts of a spatial array (e.g., in a drawing task, omitting the details when copying a picture). Right posterior damage causes problems with global processing, disrupting the configural aspects of spatial analysis (e.g., in the copying task, including details but failing to maintain coherent organization among the parts). A review of studies of children with pre- and perinatal focal brain injury to left and right posterior areas reveals that in construction and perception tasks, the adult profiles were reproduced in very young children. Those with right hemisphere injury displayed difficulty with spatial integration. Those with left hemisphere injury oversimplified complex spatial forms and failed to encode details. However, deficits shown in these children were milder than those found in adults, and longitudinal studies suggested that both groups of children eventually reached ceiling on most spatial construction tasks. Of particular interest here, however, is that even when performance was comparable to healthy peers (i.e., at ceiling on the tasks), the processing strategies of children with early lesions continued to reflect a persistent underlying deficit. Moreover, there were signs that the deficit depended on the initial side of lesion. For example, in a copying task using the Rey–Osterrieth Complex Figure, both left and right lesion groups continued to use an immature and piecemeal approach to drawing the figure. In a subsequent draw-from-memory condition, children from the left hemisphere damage group organized their figure according to global form, whereas those from the right hemisphere damage group organized it according to details. Stiles views the results as consistent with early specification in the development of spatial cognition, followed by plastic adaptation. Recovery takes place,

but it appears that "normal" behavior is being achieved by different underlying processes, and perhaps even compensatory behavioral strategies.

Variations in Plasticity

In Huttenlocher's (2002) book, there is a careful consideration of the way that functional plasticity may alter over time and across cognitive domains, and so provide the means to recovery from acquired damage. Huttenlocher lists several potential mechanisms of plasticity, some of which are available throughout life span (increase in synaptic strength, decrease in local inhibition, dendritic sprouting, formation of new synapses, and possibly formation of new neurons), and other mechanisms that are available only in the immature brain (utilization of unspecified or labile synapses, including silent synapses, competition for synaptic sites, persistence of normally transient connections).

Throughout the book there is particular emphasis on synaptogenesis, which Huttenlocher (2002) views as playing a particularly prominent role in developmental plasticity. In the developing infant, there is a tremendous burst in synapse formation (roughly 100,000/sec in the visual cortex; Huttenlocher, 2002, p. 47) at the end of which the total number of synapses is nearly twice that seen in the adult. Thereafter, there is a much longer period of pruning of excess synaptic connections, which is not completed until late adolescence in many cortical areas. This overproduction appears to reflect a general principle governing perinatal and postnatal brain development, whereby many structures including dendritic trees, axons, and synapses go through a period of "exuberant growth followed by elimination of trimming away of the excess" (Huttenlocher, 2002, p. 29). Essentially, the system is overproducing potential connections between neurons (a process that appears to be relatively independent of environmental input), and then allowing the environment to specify which of these connections are useful in the formation of functional circuits. Over time, the surplus unused synapses are then eliminated. This pattern of change over time is encountered in several contexts within Huttenlocher's book, including age-related changes in synaptic density, in the amplitude of event-related potentials, and in the level of neural activity (as assessed by positron-emission tomography [PET]).

The idea here is that during the period in which there are surplus unused connections in the cortex, a brain region will be better able to produce adaptive changes in response to internal damage or to the external environment. However, once spare connections are eliminated, with the remaining connections already committed to functions, then subsequent plasticity must rely on other (perhaps less efficient) mechanisms.

Although Huttenlocher (2002) views synaptogenesis as important in developmental plasticity and the capacity to overcome early damage, he is careful to add qualifications: The number of synapses does not equate to the level of cognitive

functioning. A 3-year-old has many more synapses than an adult but much less sophisticated cognitive function. Individuals with learning disabilities may nevertheless have normal levels of synaptic density (Cragg, 1975). Moreover, there is no direct relation between the development of complex cognitive skills and the profile of synaptic density, with skills such as abstract thought, judgment, planning, and reasoning emerging slowly throughout late childhood and into adolescence, at a time when synaptic density is gently declining to adult levels.

Two crucial points emerge from Huttenlocher's (2002) review of plasticity. The first is that, as assessed by changes in synaptic density, changes in plasticity are region specific. Thus the plasticity of the visual system appears to reduce before the plasticity of the language systems. The plasticity of prefrontal cortex and posterior parietal cortex, which underlie complex cognitive skills, reduces more slowly again. Somatosensory cortex appears to retain plasticity throughout life, evidenced by the reorganization of sensory maps in response to limb loss. The motor cortex, on the other hand, has a lower initial synaptic density, less synaptic pruning, and demonstrates less ability to reorganize after damage.

Huttenlocher (2002) argues that the burst of synapse formation in each area can be associated with the onset of simple functions. For instance, he notes that synaptogenesis in the auditory cortex precedes that in receptive language areas (Wernicke's area), which precedes that in productive language areas (Broca's area; Huttenlocher, 2000). This is the same order in which the functions emerge during development: Response to speech sounds precedes language comprehension, which precedes language production. The region-specific pattern of synaptogenesis may even be a particularly human characteristic. In rhesus monkeys, no such regional differences were found. Changes in synaptogenesis were found to be concurrent across all regions. Moreover, humans showed a much longer time course of synaptogenesis and synapse elimination than monkeys (Huttenlocher, 2002, p. 56).

The second crucial point is that the neuroanatomic and neurobiological findings provide no evidence for the strict notion of an early critical period for learning that ends in late infancy (Bruer, 1999). Huttenlocher (2002) suggests that optimal time windows for learning may well exist, but these windows are wide and close slowly. They vary according to the anatomical development of specific cortical regions (see Table 10.1, p. 212). In Huttenlocher's view, periods of enhanced effect of environmental input and recovery from damage coincide with the plateau of high synaptic number and density present from late infancy to late childhood in most cortical areas.

However, the ability of the cognitive system to reorganize in response to damage reminds us that we must be aware of the range of mechanisms that are available (at different times) for functional changes. For example, evidence from event-related potential studies suggests that the comprehension of single words moves from bilateral processing between 13 and 17 months to left-lateralized processing

at 20 months (Mills, Coffey-Corina, & Neville, 1993, 1997). As we have seen, this is part of a general developmental pattern of emergentism, where processing is initially diffuse but is then increasingly restricted into more specific regions dedicated to single functions. Such modularization improves the efficiency of processing but may do so at the expense of decreased plasticity (Huttenlocher, 2002, p. 142). However, if the word-recognition system has organized itself into the left hemisphere and begun to eliminate unused synapses at 20 months, how is it that language function can be shifted into the right hemisphere following left hemisphere damage up until 7 years of age? Is a new language system really able to form from scratch in the right hemisphere at any age up to 7? Huttenlocher suggests not, and that instead, the potential mechanism of plasticity in this case is residual functional circuits in the right hemisphere, which have persisted from an earlier phase of bilateral language processing.

A second example of different mechanisms of plasticity comes from focal damage to the motor cortex. In humans, unilateral perinatal damage to areas responsible for facial movement can nevertheless be followed by normal functional development. Such recovery is not possible if damage is postnatal. Similarly, damage to areas responsible for the movement of lower extremities (e.g., those responsible for gait) can show reasonable recovery provided they occur in childhood. However, after early damage to areas responsible for voluntary hand and finger movements, there is very little recovery. Even in the motor areas, levels of plasticity following damage appear to differ.

Huttenlocher (2002) argues that the first two of these motor behaviors are recoverable due to the existence of transient axonal connections. In normal development, these connections are subsequently lost. However, following damage to one motor cortex, the motor cortex in the undamaged hemisphere can retain and use the transient connections to take over the function of the damaged motor cortex. One motor cortex is sufficient to control both sides of the body in these two cases because face movements and limb movements in walking are relatively symmetrical. However, voluntary hand movements are more complex and often asymmetrical. A single motor cortex does not have sufficient developmental plasticity to take on such a complex function. And the reason for limitation in plasticity is that motor cortex has a special structural organization to reflects its function. Thus Huttenlocher comments, "Plasticity that depends on the availability of large numbers of unspecified synapses may be diminished in favor of a relatively fixed, very efficient, rapidly conducting system that allows for remarkable speed and precision of voluntary movements" (2002, p. 126). Plasticity after damage can clearly vary both in its absolute level and in the mechanisms that it can exploit, not only across cortical regions but also across different functions within those regions.

What can we conclude about the plasticity of cognitive systems based on recovery from early brain damage? Here are four points: (a) Functional plasticity appears to reduce with age, but the exact profile of this reduction is not clear. There

are differences in plasticity between brain regions and, indeed, between different functions within brain regions. (b) The brain is not equipotential. Specialization is channeled by preexisting computational biases in different neural circuits. (c) These biases appear to vary in strength across cognitive domains—weaker for language, stronger for spatial cognition—and appear not to be particularly precise regarding content, as illustrated by the failure of face recognition and object recognition to dissociate developmentally after early damage. (d) The process of plasticity is greatly influenced by the richness of the environment and is not, therefore, a purely intrinsic property of the system.

In terms of our initial theories on the origin of adult brain structures, then, we can conclude that the brain does not possess equipotentiality, nor are its structures irreversibly determined by an innate plan. Instead, the intermediate position of emergentism dictates plasticity and limits on plasticity. These limits may change over time, and crucially, they are not uniform across the brain. There is likely no single thing as the brain's plasticity.

ASSESSING LIMITS TO PLASTICITY VIA DEVELOPMENTAL DISORDERS

How tightly do neurocomputational constraints or biases guide the emergence of the adult functional structure across development? This question can be addressed by examining cases where those neurocomputational constraints have been altered by events early in brain development, that is, in developmental disorders (see Thomas & Karmiloff-Smith, 2002, in press-a, in press-b).

Bradshaw's recent book (2001), *Developmental disorders of the frontostriatal system,* compares six different developmental disorders that share deficits relating to executive function. These are Tourette's syndrome, obsessive–compulsive disorder, attention deficit hyperactivity disorder (ADHD), schizophrenia, autism, and depression. Several of these disorders are also covered in separate chapters of HDCN (Tourette's syndrome: Leckman, Peterson, Schultz, & Cohen, 2001, chap. 35; ADHD: Karatekin, 2001, chap. 36; schizophrenia: Elvevåg & Weinberger, 2001, chap. 37; autism: Ozonoff, 2001, chap. 34). But here the disorders are situated and related within a single coherent framework from the perspective of executive dysfunction.

Bradshaw (2001) begins by outlining the structure and function of the frontostriatal system. These are the circuits that enable us to decide what to do, when to do it, and how to achieve it, influenced by additional biases from limbic mechanisms. Modulatory neurotransmitters help drive the system, and the system combines with both the cerebellum and basal ganglia in selecting, inhibiting, releasing, filtering, modulating, and automating behaviors. The six disorders are then related to possible malfunctions of this system. Atypical behaviors are viewed

as lying on a continuum with normal performance, both in that disorders often represent the persistence of apparently juvenile behavioral patterns found in children, and in that the behaviors can also be found on occasion in normal healthy adults (e.g., when we make "doubly" sure the oven is turned off, when we sometimes feel "blue," and so on). Bradshaw discusses genetic heritability and comorbidity, as well as neuropsychological, neuroscientific, and treatment aspects of each disorder. There is even a discussion of possible evolutionary advantage of the exaggerated traits (apparently sufferers of ADHD may have made superb warriors in the past and schizophrenics rather good shamans).

For our purposes, developmental disorders are interesting because—by definition—these disorders represent the limits of plasticity. For them to be classified as disorders, whatever initial damage was caused to the brain, reorganization and compensation are not sufficient to ameliorate deficits during the developmental process.

Some researchers have attempted to extend the methodology of adult cognitive neuropsychology to developmental disorders, attempting to identify specific cognitive deficits and draw parallels with deficits shown by adults with brain damage (e.g., in disorders such as dyslexia, dyscalculia, Specific Language Impairment [SLI], and so on; see, e.g., Temple, 1997). Given what we have seen about plasticity, this search seems questionable. Once early damage has been filtered through the process of development, specific cognitive deficits are unlikely (Karmiloff-Smith, 1998). Indeed such deficits would only emerge under a very particular kind of developmental account that is rarely argued for (an account that we term *residual normality;* see Thomas & Karmiloff-Smith, in press-a, for discussion).

This seems particularly true in developmental disorders with a genetic cause. Thus Pennington (2001 [HDCN]) argues that the correspondence between genes and complex behavioral phenotypes is many-to-many, not one-to-one. As a result, he casts doubt on the nativist notion that there might be a gene (or specific set of genes) that affects a very specific aspect of cognition, such as a particular gene for grammar. Indeed, where behavior does look normal in developmental disorders, the plasticity and compensation that do exist within these systems may mean that atypical cognitive processes underlie apparently normal behavior (Karmiloff-Smith, 1998; Karmiloff-Smith, Scerif, & Thomas, 2002).

Specific cognitive deficits will be unlikely in genetic developmental disorders because damage is typically thought to be quite widespread in the brain. Although in previous decades, disorders such as autism and schizophrenia were sometimes blamed on methods of parenting, the pendulum has now swung back toward viewing them in terms of disruptions to early stages of brain development. Pennington (2001 [HDCN]) suggests three broad classes of genetic effects on brain development that might be open to disruption. These are effects on (a) brain size, in terms of altering the number of neurons or synapses; (b) neuronal migration, sometimes

in a regionally specific fashion; and (c) neurotransmission, either by changing levels of neurotransmitter or changing the binding properties of receptor proteins. Consistent with these predictions, Bradshaw (2001) concludes that all developmental disorders show abnormal brain morphology of some kind, including deficits or alterations in neural migration, altered asymmetries, microgyria, lissencephaly, microcephaly or hydrocephaly, abnormalities of apoptotic pruning, and specific histological or biochemical abnormalities. In contrast, he argues that none of the pervasive developmental disorders have been unequivocally associated with discrete focal lesions. In sum, differences in the structural and neurocomputational properties of developmentally disordered systems are widespread, and it is these altered properties that will determine the limits on plasticity.

An indication of the types of plasticity and compensation that are available in these disorders can be illustrated by three cases: reading skills in developmental dyslexia, speech recognition in SLI, and face processing in autism.

Casey et al. (2001 [HDCN]) report functional MRI studies in adults with a history of developmental dyslexia. These studies show that compared to controls, people with dyslexia exhibited reduced activity in left posterior temporal-parietal areas during phonologically demanding reading and listening tasks. However, several studies have demonstrated increased activity in occipital regions. These differences are consistent with attempts to compensate for deficits in phonological processing by employing additional visual strategies. Bedi (2001 [HDCN]) evaluates training programs employed with children with SLI. She concludes that such programs, involving intensive practice on speech discrimination tasks, promote experience-dependent neural plasticity and are effective in improving auditory processing and language comprehension. However, no imaging data are available to indicate whether plasticity was operating on areas typically involved in auditory discrimination in healthy participants or elsewhere. De Haan (2001 [HDCN]) describes how certain characteristics of face processing are abnormal in autism, including the absence of categorical perception of facial expressions. However, when individuals with autism were tested on an expression-recognition task, only those with lower IQs showed impairments—despite the fact that all individuals showed deficits in categorical perception. De Haan concludes that the individuals with higher IQs were using compensatory strategies to achieve good recognition by other means. She suggests that there is "a degree of plasticity in the developing system that allows for development of alternative strategies/mechanisms in face processing" (p. 393; see also Karmiloff-Smith, 1997, for related findings of atypical underlying processes in proficient face recognition in Williams syndrome).

Interestingly, new evidence has emerged from the PET imaging of neurotransmitter systems that the alteration in the plasticity of brain areas (as indexed by the numbers of particular types of synapses) may not follow the normal course in developmental disorders (see Huttenlocher, 2002, p. 73). D. C. Chugani et al. (1999) found that in controls, serotonin synthesis capacity (which depends in part on the

number of serotonergic synapses) in 5-year-old children was twice the adult value, subsequently decreasing back to the adult value following synaptic pruning. Children with autism, on the other hand, had a lower serotonin synthesis capacity than controls at age 5, but the level steadily increased to 1.5 times the normal level by age 15, implying both delayed early synaptogenesis and then decreased synaptic pruning. Intriguingly, Huttenlocher notes that this abnormal pattern has been found in the primary visual cortex of animals deprived of normally formed visual images during the system's early sensitive period.

In short, plastic compensatory changes can take place in developmental disorders. However, the evidence is suggestive of the idea that different processes have to be used to achieve normal-looking behavior; and that, whatever anomalies have been built into the relevant neural structures, they cannot be overcome by experience-dependent plasticity (which may in any case differ from normal in atypical systems).

Returning to the disorders considered by Bradshaw (2001), of particular interest is how late in development symptoms can appear—especially given the claim for such early neurodevelopmental causes. For instance, Bradshaw gives the age of onset of Tourette's syndrome as between 5 and 10 years, obsessive–compulsive disorder as after 7 years and typically shortly after puberty, ADHD as peaking between 3 and 4 years, autism as between 2 and 5 years, and schizophrenia as a disorder of young adulthood. Now, in some cases, the late onset of these disorders is because their symptoms overlap with normal behavior in young children. However, this is not the whole story.

Take two cases, autism and schizophrenia, both disorders in which initial development often appears to progress normally. Here are the underlying causes that Bradshaw (2001) proposes for each (lists are illustrative rather than complete). Autism: increased brain size but no gross brain abnormalities; widespread low-level anomalies, including reduced numbers of Purkinje cells in the cerebellum; increased cell density but reduced dendritic arborization and neuronal size in the hippocampus, subiculum, entorhinal cortex, amygdala, and mammillary bodies; less distinct laminar structure in the anterior cingulate—the overall pattern consistent with a neurodevelopmental disorder of reduced programmed cell death (pruning) in the second trimester of prenatal development. Schizophrenia: reductions in whole-brain volume; ventricular enlargement, reduced hippocampal volume, hypofrontality, with temporal and frontal lobes, and related limbic structures especially affected; abnormalities in neuron density, number, and size in several areas, including substantia nigra and thalamus; indications of abnormal midline brain development—the overall pattern consistent with a neurodevelopmental disturbance in early or midgestation, perhaps involving abnormal neuronal migration due to improper functioning of proteins that regulate such migration, pruning, and synaptogenesis.

The point here is not in the exact details of these lists, but in the question they prompt: If there are so many differences in these disorders, how come initial devel-

opment looks normal? There are two kinds of answers here. First, retrospective analyses of individuals who later experience these disorders often demonstrate the presence of subtle behavioral precursors, for instance as revealed by the inspection of home movies of children who later develop schizophrenia as adults. The second answer is more relevant to our concerns. Bradshaw (2001) argues that the consequences of atypical development may not manifest until considerably after the relevant processes have acted, because deficits cannot emerge until the appropriate structures have developed to the extent that their normal effect should now impinge on behavior. In the case of frontal systems, for example, it may be that only when competing cognitive skills need to be controlled will deficits in the control system emerge. Previous, stimulus-driven behavior may have appeared to have developed normally because there was no need to mediate between goals at this earlier stage of development. Bradshaw supports this argument with evidence from primate studies, where prenatal prefrontal injury only manifests symptoms much later in development and often when such individuals are exposed to environmental stressors. In other words, the neural machinery present in these atypical brains is sufficient to support behavioral development in the normal range only up to a certain point. Only when the next behavior to emerge requires structures that have been particularly affected by abnormal neurodevelopment, or when the system has been placed under stress, do the symptoms emerge. However, the underlying differences have been present from birth.

We can now draw some conclusions about developmental disorders and plasticity. Some researchers have looked for specific cognitive deficits in these disorders, seeking analogues with deficits found in adult brain damage. On the face of it, this type of developmental-acquired comparison is odd because, as we saw earlier, the combination of focal deficits + development in healthy children tends to produce recovery through plasticity in the cognitive system. The fact that deficits persist in developmental disorders suggests that these disorders involve atypical limitations on plasticity, rather than focal damage. These limitations arise because disruption to early brain development leads to (among other things) the alteration of low-level neurocomputational constraints. This alteration in turn modifies, to a greater or lesser extent, the relevance of the computational properties of various brain areas to the cognitive functions they normally come to acquire. In some brain areas, the constraints have been altered in a way that makes them unable to acquire their normal function (see Thomas & Karmiloff-Smith, 2002, in press-a, in press-b, for further discussion of this idea). Although compensation can occur during cognitive development, it does not appear to reflect equipotentiality in the system. Rather, behaviors are often achieved by qualitatively different underlying processes. One clear example was the case of people with developmental dyslexia for whom imaging data suggested recruitment of additional visual processing to compensate for deficits in phonological processing. In short, the existence of superficially specific deficits in behavior in the outcome of developmental disorders does not imply that

the underlying cause is specific and localized damage, as it sometimes does in cases of adult brain damage.

If there are analogies to be drawn between developmental disorders and cases of acquired brain damage, the relevant comparison group appears to be healthy children who have suffered generalized and diffuse damage. Recall that with general damage, recovery over the long term is poor, with children "growing into" their deficits and failing to show the normal emergence of new behaviors. In developmental disorders, differences in the brain can also be widespread, as illustrated by the cases of autism and schizophrenia. And as we saw previously, individuals can also demonstrate an analogue of growing into their disorder: Initial development can appear normal, but when certain skills are due to emerge that rely on particularly anomalous brain structures, it is then that overt behavioral markers show marked divergence from the normal pathway.

TWO CAUTIONARY TALES

Before we finish, two findings suggest we are still some way from having the full picture on plasticity and development. First, recall Bradshaw's (2001) claim that the normal pattern of brain lateralization is often disturbed in developmental disorders. From the emergentist view of adult cognitive structures, we can imagine that some aspect of disordered brain development has deflected the process of functional specialization. However, Bradshaw points out that in some disorders, medication can not only control the clinical manifestation of the disorder, but also correct the anomalous lateralization. As Bradshaw says, "The expression of many asymmetries has at least a partly neurochemical basis, which may modulate any underlying structural asymmetries" (p. 265). In a similar vein, lateralization differences due to hormone levels have been found in a functional MRI study of verbal and nonverbal memory in two groups of postmenopausal women, one on hormone replacement therapy and one off it (Shaywitz et al., 1999). This study revealed increased right cortical activation in the group on estrogen therapy, differences that were presumably not present prior to treatment. This difference might have functional significance because estrogen therapy in postmenopausal women is associated with an improvement in verbal memory performance (Sherwin, 1997; see Huttenlocher, 2002, chap. 7, for discussion). In short, although the final adult structure may be less open to changes through plasticity, this does not necessary imply that it is static in all respects.

Second, previously we examined the case of orphanage-reared children in relation to the effect of an impoverished environment on cognitive development. Despite the deleterious effect of a lack of stimulation on many aspects of cognitive and sensorimotor development, subsequent follow-up of these children after adoption into middle-class families suggested that the window for provision of this

stimulation was quite wide (Gunnar, 2001 [HDCN]). This argues against an early critical period for acquisition. There was, however, one exception where successful development may rely on early exposure to a particular kind of experience. This exception involves a cluster of skills including executive function, emotion regulation, the capacity to respond appropriately to social cues and boundaries, and the capacity to establish person-based relationships. According to Gunnar, the necessary environmental input appears to be exposure to experiences within consistent adult-infant relationships, for which the developmental window is much narrower. Gunnar comments, "Development of aspects of frontal functioning may be highly dependent on early experiences in ways that are not readily recoverable if privation continues beyond the first year or so of life" (pp. 626–627). Furthermore, a preliminary PET imaging study of children from Romanian orphanages subsequently adopted by U.S. families revealed decreased metabolism in several areas of prefrontal cortex (H. Chugani et al., 2001). On the basis of this evidence, Huttenlocher (2002, p. 72) argues that the prefrontal cortex may be an area of the brain "whose development is especially dependent on environmental stimulation."

This finding, although tentative, deserves significant follow-up, if only on humanitarian grounds. However, it is also interesting for the following theoretical reason. Many accounts of developmental plasticity have argued that plasticity diminishes in systems subserving lower level cognitive functions before it does so in systems subserving higher level functions. Indeed, the high-level frontal executive systems are thought to retain their plasticity the longest (see Johnson, 1997, for discussion). The longer the plasticity of a system, the more opportunity the environment has to determine its structure and overcome initial disruptions. Yet evidence from these unfortunately deprived children paints a different picture. If plasticity is measured by recovery after early deprivation, high-level executive systems appear to demonstrate a swifter reduction in plasticity than do the more basic sensorimotor and cognitive abilities. If we appeal to the theoretical framework of acquired brain damage in children, these data suggest that we should view the development of executive functions less in terms of plasticity and more in terms of early vulnerability.

CONCLUSIONS

We are now in a position to address the concerns that Bruer (1999) expressed in the quotation at the beginning of this essay. When people talk about brain plasticity, are "advocates and the media" prone to leap beyond the scientific facts? Take, for example, the quote from our mind-mapping champion. Has she allowed her enthusiasm to "outstrip scientific understanding" when she credits the brain with infinite plasticity and the ability to compensate for the deficits of developmental dyslexia

via special techniques? Based on the three books reviewed in this essay, the answer is yes and no.

In terms of ascribing infinite plasticity to the brain, this is clearly overstating the case. Significant plasticity, perhaps. However, this is a plasticity that changes over time in a way that is not yet completely understood, and which is not uniform across cognitive domains. Plasticity is greater in children, at least as assessed by the ability to recover from certain sorts of lesions (focal, unilateral). In contrast, the adult brain requires hard work to change its structure, and such changes are not extensive. Moreover, it is currently unclear how much the inherent plasticity of the brain is actually reducing over time, and how much the adult brain simply has many more functions established within it that are unwilling to give up their circuits and synapses while they are still being used.

It terms of compensation, it is clear that the brain is not equipotential in a strong sense. Brain areas have different neurocomputational biases that "seed" eventual adult specialization across development. These biases may be weaker or stronger for various cognitive domains. The strength of the bias determines the extent to which cognitive development is railroaded or free to wander, and how readily one part of the brain can take over the function of another. Consistent structure may still emerge with weak biases if the environment guarantees a consistent, highly structured input for a given cognitive domain.

If compensation sometimes seems to imply equipotentiality, then we need to bear in mind the different ways that the brain can recover. In particular, we must distinguish between two types of compensation when alternative tissue attempts to take over the function of damaged tissue. Compensation can either constitute an attempt by the new tissue to acquire the same cognitive processes (perhaps not as efficiently, given this replacement tissue does not have quite the right bias). Or it can constitute the new tissue, achieving the same behavioral ends by alternate means in the form of atypical cognitive processes. And it is worth noting that compensation from alternative tissue may lead to crowding effects, if this tissue is required to perform its normal function as well as its adopted function. Crowding effects imply capacity limitations.

On the other hand, the science does back up our mind-mapping champion when she claims that visualization techniques might aid people with dyslexia. Brain-imaging studies suggest that visual areas are activated to a greater extent in people with developmental dyslexia than in control groups during reading, consistent with compensation by these areas for poor phonological processing. However, other imaging studies show that intense training on phonological decoding alone can also produce plastic changes and associated behavioral improvement (see Casey et al., 2001 [HDCN]; Tallal et al., 1996). With respect to developmental disorders, it is important to realize that compensatory changes may lead to atypical cognitive processes, rather than normalization, because the limits on plasticity may have been changed during early brain development.

Are there hidden powers in the brain that can be spontaneously released in the adult? What of the legendary 90% of the brain's capacity that cannot be accessed unless one pays $10 to someone via a newspaper advertisement? No, it seems that hard work, practice, and motivation are necessary to change the adult brain. Moreover, crowding effects indicate that the brain can indeed "fill up," arguing against huge residual capacity.

Most of all, these books stress that brain plasticity existing in children and adults is greatly influenced by the environment. Cognitive development itself is crucially dependent on the right sort of interaction with the environment. Discussion of brain plasticity should not narrow our focus to neural processes alone. If people want to recover, if people want to change, then they need to rely on the world and other people as much as the properties of their own brains.

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