DEVELOPMENTAL PLASTICITY IN CHILDREN: THE ROLE OF BIOLOGICAL RISK, DEVELOPMENT, TIME, AND RESERVE

MAUREEN DENNIS

Department of Psychology, The Hospital for Sick Children, and Department of Surgery, University of Toronto, Toronto, Ontario, Canada

Older views of the functional developmental plasticity of the developing central nervous system (CNS) focused on the protective effect of a young age at the time of insult. In these views, a younger rather than an older age at onset was thought to produce fewer and/or less severe symptoms and a more rapid recovery. More recently, neurobehavioral outcome has been studied in a variety of medical conditions that affect the developing CNS; at the same time, new investigative techniques, such as brain imaging, have elucidated the biological basis of structural and functional brain plasticity. In consequence of a better understanding of the structural and functional consequences of developmental CNS insults, a body of research has emerged that is shaping a new view of functional developmental plasticity, in which neurobehavioral outcome is set by the biological risk associated with a medical condition and moderated by age and development, the time since onset of the condition, and the reserve available within the child, family, school, and community.   © 2000 by Elsevier Science Inc.

Educational Objectives: The reader will be introduced to newer views of functional developmental plasticity occurring after biological insults to the developing CNS, including concepts of: (1) *neurobehavioral outcome* and ways to measure it; (2) *biological risk*, the cumulative severity of primary and secondary CNS insult; (3) *age and development at onset* of CNS insult, which are markers for physical, brain, and cognitive development; (4) *time since onset* of CNS insult; and (5) *reserve*, the set of factors available in the child, the family, or the broader social context that buffer or enhance the effect of biological risk.

KEY WORDS: Plasticity; Biological risk; Neurobehavioral outcome; Development; Reserve

INTRODUCTION

Plasticity may be considered in terms of structure (brain microstructure or macrostructure) or function (brain function, or behavior, which is the expression of brain function). Structural synaptic plasticity within a neural network

Address correspondence to Maureen Dennis, Ph.D., Psychology Research, The Hospital for Sick Children, 555 University Avenue, Toronto, Ontario M5G 1X8, Canada. Tel: (416) 813-6658; Fax: (416) 813-8839; E-mail: <mdennis@sickkids.on.ca>.

is responsible for the alterations in CNS function that foster learning and memory (Teyler et al., 1995). Functional plasticity is the behavioral mechanism for emerging competencies and skill acquisition (Greenough et al., 1993).

Because the CNS is designed to solve functional problems in both children and adults, it exhibits functional plasticity at all ages; that is, it has a significant degree of age independence. Historically, however, it was believed that plasticity was age dependent, and the concept of *age-based plasticity* implied that the adaptive functional response of an immature central nervous system was greater than that of the mature nervous system. To the extent that the brain was fully plastic at birth, and plasticity was lost with age and development, then: (a) CNS lesions in children would produce fewer deficits, less severe deficits, and more rapid recovery of function than CNS lesions in adults; (b) brain immaturity would convey a functional advantage to an individual sustaining a brain lesion, so that children with early CNS lesions would experience no functional consequences, and the degree of CNS immaturity would be related to the degree of functional lesion-induced deficit.

Evidence for the idea that functional plasticity is age-based is equivocal. On the one hand, immature neural connections are relatively transient, and recruitment of brain regions with transient rather than stable connections may be one viable mechanism of functional reorganization for parts of the immature cortex. For example, it has been suggested that early lesions may stabilize normally transient connections in neighboring brain regions, which may then provide a substrate for function (Webster, Ungerleider, & Bachevalier, 1991). On the other hand, age itself may not be the basis of age-based functional plasticity, because factors other than age distinguish child and adult CNS insults, making equivocal the comparison between child and adult lesions that is the basis of the idea that children can experience CNS insult with functional impunity by virtue of their young age.

OLDER VIEWS OF AGE-BASED FUNCTIONAL PLASTICITY

The Equivalent Cause Problem

Early evidence for age-based plasticity compared aphasia in adults with strokes and children with diffuse, infectious, or traumatic brain injury (e.g., Basser, 1962). However, regardless of age, aphasic symptoms abate more quickly in individuals with traumatic causes and less quickly, if at all, in those with vascular cause (Dennis, 1996). Because strokes from arteritis are common in adults but relatively rare in children, it is often difficult to study the functional effects of the same pathologic condition. To assess age-based plasticity accurately, the cause of brain insult should remain constant across the age range studied.

The Symptom Base Rate Problem

The base rate of certain behavioral symptoms is different in children and adults. For example, aphasic neologisms, which provide evidence about the functional plasticity of the phonologic system, occur less frequently in children than in adults (Dennis, 1996).

The Age at Lesion Versus Time Since Lesion Problem

In older studies, acute phase adults were compared with chronic phase children (for discussion, see Dennis, 1980). Understanding functional plasticity requires separation of *age at insult* and *time since insult* variables, each of which may be associated with different effects on cognitive function (Dennis & Barnes, 1994a).

The Functional Variability Problem

Some parts of the CNS exhibit a predictable relation between structure and function; for example, the frontal eye fields reliably control eye movements. For other parts, the relation is less clear. Where lesion effects are not fully predictable for either children or adults, questions about age-based plasticity are difficult to address.

The Dearth of Information on the Subcortical and Subtentorial Brain Regions Problem

Historically, information about age-based functional plasticity was based on cortical lesion data. Many developmental CNS insults involve subcortical or subtentorial (below the tentorium cerebelli or tent) brain regions, which may differ from cortical insults in the nature and extent to which they exhibit age-based functional plasticity (Dennis, Hetherington, Spiegler, & Barnes, 1999).

The Recovery of Old Functions Versus Acquisition of New Functions Problem

Earlier views did not recognize that age-based functional plasticity involves both recovery of old skills and acquisition of new skills. For the mature organism with brain insult, many skills are fully developed; for the immature organism, the maturational challenge is to move from one developmental level to the next. Understanding the acquisition of new skills requires consideration of multiple developmental indices, including the growth curves relating functional competence and age.

NEWER VIEWS OF AGE-BASED FUNCTIONAL PLASTICITY

Recent research is shaping a newer view of functional developmental plasticity. In this view (Dennis, 2000), neurobehavioral outcome is set by the *biological risk* associated with a medical condition and is moderated by *age and development*, the *time since onset* of the condition, and the *reserve* available within the child, family, school, and community.

Neurobehavioral Outcome

Neurobehavioral outcome is the observed pattern of cognitive and behavioral skills. To understand outcome, five concepts are important.

Modal profile. The modal profile is the most typical set of cognitive strengths and weaknesses associated with a condition. For example, the modal profile for children with hydrocephalus involves cognitive strengths in the processing of objects, facts, references, and words, and cognitive weaknesses in the cognitive processing of locations, procedures, inferences, and texts (Dennis, Barnes, & Hetherington, 1999).

Variability. Variability is part of neurobehavioral outcome. Newer approaches to plasticity have related variability around a modal profile to biological features of the condition. For example, variability around the modal cognitive profile of children with hydrocephalus is related to physical symptoms, such as eye movement disorders, to medical severity indices, such as level of spinal cord lesion, and to specific brain dysmorphologies in the cerebellum, midbrain, corpus callosum, and posterior cortex (Fletcher, Dennis, & Northrup, 2000).

Core deficits. Core deficits, an important part of neurobehavioral outcome, are cognitive impairments defined in terms of underlying processes that are robust across various levels of disorder severity and mental ability. For example, impairment in deriving contextual meaning is a core language deficit for children with hydrocephalus because: it is relatively independent of general cognitive level; it does not reduce to deficits in other, more basic language functions; and it is evident on context-dependent but not context-independent language tasks (Dennis, Barnes, & Hetherington, 1999).

Challenge level. The ability to function under challenge is an important component of neurobehavioral outcome relevant to successful function in the real world. Children with CNS insults may manage routine tasks, but fail with multitasking, distraction, the need for metacognitive monitoring, or a combination thereof.

Phenocopies. Phenocopies are outcome profiles that are superficially alike but that arise from different cognitive processes. For example, children with hydrocephalus and children with closed head injury both exhibit poor reading comprehension, but this skill is related to a *processing speed bottle-*

neck in head injury and to *impairment in deriving contextual meaning* in hydrocephalus (Barnes, Dennis, & Wilkinson, 1999; Barnes, Faulkner, & Dennis, 1999). Understanding of phenocopies in neurobehavioral outcome prompts exploration of the constituent processes of cognitive outcome.

Biological Risk

Biological risk is the cumulative effect of primary and secondary CNS insults. This may involve effects relating to gene, metabolism, physiology, congenital brain dysmorphologies, or primary and secondary acquired brain insult.

Primary severity involves the CNS compromise associated with the basic insult. For example, head injury severity is assessed using metrics such as depth of coma, duration of impaired consciousness, and length of posttraumatic amnesia (Yeates, 1999). Insult to the CNS is a process of events, not a single event. For example, traumatic brain injury involves a cascade of interrelated processes (direct damage caused by calcium influx into cells, free radical-mediated damage, receptor-mediated damage, and inflammation) that contribute to delayed cellular damage (Gennarelli & Graham, 1998). Secondary effects of CNS insults may magnify the effects of the basic insult. For example, raised intracranial pressure, sometimes accompanied by hydrocephalus, occurs secondary to a variety of medical conditions: lysosomal storage diseases such as mucopolysaccharidosis (Whitley et al., 1993); bacterial meningitis (Stovring & Snyder, 1980); head injury (Bruce, 1995); and subtentorial brain tumors (Raimondi & Tadanori, 1981). Cognitive outcome is generally poorer when secondary complications are added to primary insult. For example, in identical twins with low birth weight and intraventricular hemorrhage, secondary hydrocephalus in one twin magnifies neuropsychological deficit (Dennis & Barnes, 1994b).

Age and Development

Age is a marker for level of physical, brain, and cognitive development. Neurobehavioral outcome must be referenced to the child's skill level at disease onset, the subsequent course of skill development, and the status of long-term skill maintenance (Dennis, 1988; Dennis & Barnes, 1994a). Because skills are volatile in the child, cognitive deficits may emerge only slowly. Delayed or latent (Taylor & Alden, 1997) effects, which are identified from longitudinal study of function into adulthood, are critical for understanding age-based functional plasticity.

A younger age at onset of CNS insult is associated with greater vulnerability to a variety of medical conditions and to the cognitive morbidity that follows them. Immature organisms are more likely than mature ones to experience certain disorders, and, once they experience a disorder, younger cohorts are more vulnerable to cognitive deficit.

Fetal onset disorders are devastating to physical and cognitive development. For example, adult mercury exposure produces restricted lesions in the parietal–occipital and cerebellar regions, whereas fetal exposure produces widespread brain compromise (Takeuchi, 1968). Fetal origin insults are associated with autism and Joubert syndrome, both profound neurodevelopmental disorders with CNS dysmorphologies involving the midbrain and cerebellum (Rodier, Ingram, Tisdale, Nelson, & Romano, 1996; Maria et al., 1997).

Newer evidence now supports the view that earlier onset of CNS insult during childhood produces poorer neurobehavioral outcome. For conditions involving diffuse or multifocal disease processes, a young age at onset is associated with greater cognitive morbidity (Taylor & Alden, 1997) for conditions that include: congenital and infantile hydrocephalus (Hetherington & Dennis, 1999); childhood diabetes (Holmes & Richman, 1985); bacterial meningitis (Anderson et al., 1997); closed head injury (Barnes, Dennis, & Wilkinson, 1999; Ewing-Cobbs, Levin, Eisenberg, & Fletcher, 1987; Kriel, Krach, & Panser, 1989; Wrightson, McGinn, & Gronwall, 1995); heart disease (Wright & Nolan, 1994); and cranial irradiation (Anderson, Smibert, Ekert, & Godber, 1994). An older age at onset may even lower the risk of poor cognitive outcome. For example, the younger the age at diagnosis of malignant tumors of the posterior fossa, the greater the neuropsychological dysfunction (Allen & Epstein, 1982; Duffner, Cohen, & Thomas, 1983; Kun, Mulhern, & Crisco, 1983). Finally, early onset of some forms of CNS insult may result in more rapid deterioration and dementia in childhood and adolescence than later onset (Shapiro, Lockman, Balthazor, & Krivit, 1995).

Time

Time since onset of CNS insult is an important moderator of outcome, and time does not always entail recovery. For example, in childhood survivors of meningitis, behavior problems increase and some cognitive functions decrease with time (Taylor et al., 1990; Taylor, Schatschneider, & Minich, 1999). Time may bring no change in cognitive status; alternatively, time may reveal new deficits, exacerbate old deficits, or change the rate of development. For example, younger head-injured children show a slower rate of improvement over time and more significant residual deficits than do older children with equally severe injuries (Anderson & Moore, 1995; Ewing-Cobbs et al., 1987).

Longitudinal studies of cognitive function in adult long-term survivors of fetal CNS insults provide a long developmental perspective. For example, children with early onset hydrocephalus and poor math skills develop into adults with limited academic competence in math and functional innumeracy (Dennis et al., 1998), which identifies neurobehavioral outcome as a deficit, not a lag.

Reserve

Insults to the CNS occur in children in the context of a reserve, which refers to preinjury and postinsult factors that buffer or exacerbate neurobehavioral outcome (Satz, 1993). Preinjury reserves may involve demographic, cognitive (e.g., high intelligence), physical, or socioeconomic resources. Social disadvantage increases the probability of incurring particular medical conditions (Broome, 1987). Preinjury behavior may also affect outcome. For example, preinjury behavior problems, which are more common in children with mild head injury than in noninjured children, may have increased the risk of incurring the head injury (Asarnow et al., 1995).

After insult, physical health is often compromised by sensory loss, cranial nerve dysfunction, seizure disorders, hemiplegia, or ataxia, as well as difficulties in growth, metabolism, and sleep. Health status affects school attendance, attentiveness, and performance on neuropsychological tests. Mental health also moderates neurobehavioral outcome. For example, the outcome of childhood traumatic brain injury depends on both injury severity and postinjury psychiatric disorders (Max et al., 1999). Socioeconomic resources are also important and predict postinsult intelligence better than the best biological moderators for some conditions overrepresented in socially disadvantaged groups, such as sickle cell disease (Brown et al., 1993). Both socioeconomic status and family stress predict outcome after childhood brain tumors (Carlson-Green, Morris, & Krawiecki, 1995). Effective academic and neuropsychological rehabilitation moderates cognitive outcome after CNS insults.

CONCLUSIONS

Issues related to biological risk, age and development, time, and reserve are relevant to newer views about functional plasticity in childhood. These views have practical implications for diagnosis and management, as well as theoretical relevance to the search for a developmental perspective on childhood CNS insults.

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CONTINUING EDUCATION

Developmental Plasticity in Children: The Role of Biological Risk, Development, Time, and Reserve

QUESTIONS

1. Current understanding of the mechanisms of plasticity has been shaped by which of the following?
   a. Evidence that young children experience full recovery from early lesions
   b. Evidence that young children with early lesions can continue to show functional problems into adulthood
   c. Evidence that the pattern or extent of recovery is independent of the nature of the insult to the brain
   d. Evidence that fetal brain lesions have virtually no cognitive sequelae
   e. All of the above
2. The modal profile of a disorder:
   a. Must account for all variants seen in the disorder
   b. Can only be assessed during the acute phase of a disorder
   c. Is the same concept as the core deficits of a disorder
   d. Must include the most common signs that characterize the disorder
   e. Will explain patterns of individual variability seen with the disorder
3. Direct comparisons of plasticity in adults and children is complicated by which of the following?
   a. The prevalence of specific etiologies in child and adult populations that lead to behavioral deficits
   b. The tendency of studies to compare children in chronic phases with adults in acute phases of disease states
   c. The difficulty in comparing prelesion skill levels for adults and children
   d. Failure to consider that additional deficits may emerge with time in children who experienced early brain damage
   e. All of the above
4. Neurobehavioral outcome:
   a. Is relatively unaffected by factors like socioeconomic status
   b. Is independent of the time after onset of brain insult
   c. Is dependent on a child's developmental stage
   d. Is best predicted from knowledge of the child's "reserve"
   e. a and c
5. The concept of "reserve" could include which of the following factors?
   a. Premorbid intelligence
   b. Parental economic status
   c. Educational resources
   d. Personality traits
   e. All of the above