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The traditional approach to the study of cognition has been to focus on normative age-related changes in intelligence across the life span. In old age, the focus has been on age-related decline. This approach views intelligence as a set of stable traits within the individual, thus allowing long-term predictions of normative gain and decline across the life span (Cattell, 1963; Schaie, 2005; Terman, 1916; Thurstone, 1938). Most of the empirical studies have focused on describing normative or average change in intellectual abilities rather than on examining the full range of interindividual or intraindividual variability that might occur as a function of experimental manipulations, interventions, nonnormative life

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events, or sociocultural change. Likewise, earlier neuroscience approaches assume that neural development came to completion in early life with little plasticity occurring in adulthood (Buonomano & Merzenich, 1998). Currently, it has been recognized that cognitive change in adulthood can be multidirectional, including gain and maintenance as well as decline.

This chapter focuses on issues of plasticity in adult cognition that are of current interest for both neuroscience and psychological theories of adult development and aging (Kramer, Bherer, Colcombe, Dong, & Greenough, 2004). We begin with a consideration of variations in the definition of plasticity in current use in both developmental psychology and neuropsychology and of related concepts such as neural plasticity, neural reserve, and cognitive reserve (Baltes, 1987; Stern, 2007). We embed our discussion of the concept of plasticity within a life span developmental theoretical perspective. Several key questions are then identified that should be addressed by a theory of cognitive plasticity in adulthood, and we discuss constructs and issues related to each question. We also briefly discuss the adequacy of current cognitive theories in addressing these questions.

## Cognitive Plasticity: Construct and Theory

### Life Span Developmental Theory and Cognitive Plasticity

The concept of plasticity is closely linked to life span theory's conception of development as a process of lifelong adaptation (Baltes, Lindenberger, & Staudinger, 2006; Thomae, 1979). Life span theory maintains that development is modifiable or plastic at all phases of development; however, there are constraints and limits on developmental plasticity, and these constraints and limits vary by period of development. A major goal of life span developmental research has been to examine the range and limits of plasticity at various phases of the life span. With respect to cognition, adaptation involves the interplay between assimilating the environment to existing forms of thought and accommodating one's thought to the environment. Development is conceptualized as multidimensional, multidirectional, and multifunctional (Baltes, 1987). Cognition would then be expected to involve multiple dimensions or levels rather than a single global approach to general intelligence (i.e., *g*). The multidirectionality of development implies that there will be both growth (gains) and decrement (losses) at any developmental stage, although with advanced aging the losses may outweigh the gains. Various abilities or cognitive processes would then be expected to vary in their developmental trajectories with some exhibiting positive or negative linear trends and others exhibiting nonlinear trends.

If development in general and cognition in particular are to be studied in terms of adaptivity (rather than static traits), then intraindividual processes that foster cognitive adaptation must be explained. That is, it needs to be specified why there are interindividual differences in intraindividual adaptation and plasticity. Theories of developmental regulation or adaptation may be one way to approach this issue. For instance, the SOC theory advocated by focuses on three mechanisms (selection, optimization, and compensation) for adaptation

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or the regulation of one's resources (including cognitive capacity) throughout development. *Selection* involves the individual's choice or election to focus on adaptation and optimization in certain areas and as a result to limit one's focus in other areas. Selection may involve an emphasis on cognition or the selection of specific cognitive domains (e.g., expertise) as a focus of adaptation. The theory posits that in old age there may be increasing restriction in the selection of domains because of age-related loss in the range of adaptive potential. *Optimization* involves maximization (e.g., gaining expertise) of one's performance and potential in selected areas; optimization may be an important mechanism for the building of reserve for future use. *Compensation* becomes operative when specific behavioral capacities are lost or one's capacity in an area drops below the threshold for adequate functioning. Compensation may result in acquisition of new behaviors or bodies of knowledge or reallocation of resources. Thus, the SOC model suggests specific behaviors to explain plasticity across the life span.

In addition to the study of plasticity at the level of the individual, the biocultural constructive perspective within the life span approach emphasizes that developmental adaptivity and plasticity may also be observed on a number of different and interacting levels (i.e., from the neurobiological to the behavioral and the sociocultural) and on different time scales ranging from momentary microgenesis over life span development to phylogenetic evolution (Baltes, 1997; Li, 2003; see also Willis & Schaie, 2006). This approach situates plasticity within a larger evolutionary framework of development. Three propositions in this perspective specify the interaction between neurobiological and sociocultural influences on adaptivity and plasticity: evolutionary selection processes most strongly influence development in early life; with increasing age, behavioral plasticity depends on ever-increasing cultural resources because of a smaller degree of neuronal plasticity; however, the beneficial effect of increasing cultural resources is diminished in old age because of a decline in neurobiological functions.

### Cognitive Plasticity

Cognitive plasticity has often been defined in terms of the individual's latent cognitive potential under specific contextual conditions. Specifically, plasticity has been defined in terms of the capacity to acquire cognitive skills (Jones et al., 2006; Mercado, 2008). Cognitive skills are here defined as the abilities that an organism can improve through practice or observational learning and that involve judgment or processing beyond perceptual motor skills. The definition of cognitive plasticity usually involves a contrast between the individual's current average level of performance under normative conditions and one's latent potential.

Several aspects of the definition of cognitive plasticity should be noted. First, cognitive plasticity deals with *intraindividual* potential, the range of plasticity within an individual (Baltes & Lindenberger, 1988). While interindividual differences in intraindividual plasticity are often studied, plasticity focuses on intraindividual change or potential. Second, the *context* within which cognitive plasticity is studied needs to be specified. In most studies, cognitive plasticity has been examined within an experimental or intervention context.

The individual's average level of cognitive functioning in normative, everyday experience is then contrasted with the range of plasticity exhibited under experimental or training conditions. Specification of the contextual conditions under which plasticity is studied is critical since the range of plasticity manifested will vary on the basis of such factors as the duration, intensity, or instructional procedures used in the intervention. Third, cognitive plasticity has generally been studied within a short *time frame*. Most training studies range from one session to, at most, several months in length. Hence, the range of plasticity exhibited may also be constrained by the temporal length or intensity of the intervention. It should be noted that early in the study of cognitive plasticity within the field of cognitive aging, plasticity was assessed almost exclusively with behavioral measures. However, recently, there is increasing interest both in the conceptual relationship between cognitive plasticity and neural plasticity and in experimental studies that examine cortical changes occurring concurrently with the behavioral training or intervention efforts (Jaeggi, Buschkuhl, Jonides, & Ferrig, 2008; Westerberg & Klingberg, 2007).

### Cognitive Reserve

In the study of cognitive aging within neuropsychology, there has been considerable interest in the capability of the individual to continue to function at an adequate cognitive level when there have been neural deficits or pathology. Stern and colleagues (Stern, 2002, 2007) have proposed applying the concept of cognitive reserve to the study of this phenomenon. A distinction is made between passive and active reserve. *Passive reserve* is defined in terms of the amount of neuropathology that can be sustained before reaching a threshold for clinical expression. This model presupposes that there is a critical threshold of brain reserve capacity such that clinical or functional deficits become evident once brain reserve capacity is diminished beyond this threshold.

*Active cognitive reserve*, by contrast, is based on the premise that the brain may actively attempt to cope with and compensate for deficits by using alternative preexisting cognitive processes or by enlisting compensatory approaches (Stern, 2007). Rather than positing that brains of individuals with high levels of cognitive reserve are anatomically different than those of individuals with less reserve, the cognitive reserve hypothesis proposes that the high-functioning individuals process tasks in a more efficient manner. Individuals with higher cognitive reserve, therefore, are believed to be more successful at coping with the same amount of neuropathology. Two types of neural mechanisms underlie cognitive reserve: (a) *neural reserve* involves using brain networks or cognitive paradigms that are more efficient and flexible. Neural reserve is a normal process used by both healthy individuals coping with task demands and by individuals with brain damage, (b) *neural compensation* refers to adopting new compensatory brain networks because pathology has impacted those that are normally used.

In earlier writing, Stern (2002) differentiated between reserve as the ability to optimize or maximize normal performance and compensation, an attempt to maximize performance in the face of brain damage by using brain structures or networks not engaged when the brain is not damaged. There is variability in cognitive reserve—both variability between individuals and variability within

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individuals (intraindividual) across time. Variability in cognitive reserve can stem from genetic differences and also from life experiences, such as education, occupation, or leisure activities. Cognitive reserve is implicated not only in the emergence of a clinical condition but also in the rate and magnitude of recovery of function from brain injury. Cognitive reserve is a malleable entity the level of which at any point in time is dependent on summation of life experiences and exposures; thus, cognitive reserve can be enhanced by relevant purposeful activities.

These perspectives of cognitive plasticity within the life span approach and cognitive reserve within neuropsychology share a number of similarities. First, both approaches emphasize that the individual is an *active* agent of the development of reserve and in compensatory efforts. Stern differentiates between passive and active reserve, while the life span perspective sees the individual as a codirector of one's development and adaptation. Both acknowledge individual differences and variability in reserve. Likewise, multiple antecedents or correlates of reserve are enumerated in both perspectives. Both approaches acknowledge that there are limits or constraints to reserve or plasticity. At the same time, both approaches view reserve as malleable and suggest that there are opportunities for the enhancement of reserve. While the life span approach has utilized experimental and intervention techniques to study the range of plasticity and reserve at various developmental periods, the Stern approach has focused more on descriptive examples of cognitive reserve. Developmental differences in the range of plasticity across the life span are a major concern of life span theory, while Stern's cognitive reserve focuses primarily on old age and/or response to brain injury or neuropathology.

## Conceptualization of Cognitive Plasticity:

### Key Questions

In this chapter, we discuss five broad issues or questions that we consider to be central to any theory or conceptual framework for the study of cognitive plasticity. The first question focuses on the various levels (brain, behavior, and society/culture) at which plasticity has been or needs to be examined. We consider the conceptualization of plasticity at each level, the types and range of plasticity at each level, the key concepts and issues driving research at each level, and the relationship or interplay between plasticity at various levels. The second question focuses on the temporal durability of cognitive plasticity. Both short-term and long-term conceptions of plasticity are discussed, and the adaptivity of plasticity at short-term versus long-term intervals is considered. The third question focuses on the processes or mechanisms for achieving plasticity. Processes associated with the three levels of plasticity identified in question one are discussed. Of interest are commonalities in processes or mechanisms among the levels of plasticity. The fourth question briefly considers some issues related to developmental differences in plasticity. We consider issues such as sensitive periods for plasticity, developmental differences in asymptote and range of plasticity, utilization of different abilities or processes at different developmental periods, and developmental differences in environmental demands. A number of methodological issues arise in relation to the previous four

questions. In a final question, then, we address issues of the appropriate design and methods for study of various levels of plasticity, short-term versus long-term plasticity, and developmental differences in plasticity.

## Levels of Plasticity

In this section, we consider three different levels at which cognitive plasticity occurs and that need to be considered in any comprehensive conceptualization of plasticity. We begin with discussion of plasticity in the brain, and then proceed to discuss cognitive plasticity at the behavioral and finally at the socio-cultural levels.

### Brain

#### Neural Plasticity

Both brain structure (morphology) and brain function have been studied in relation to cognitive plasticity (Kramer et al., 2004; Mercado, 2008; Raz, 2000). While the concept of neural plasticity has been closely related to the concept of cognitive plasticity, the exact relation (or even directionality of the relation) between the two concepts has not been fully explicated. It has been assumed that neural plasticity contributes to or underlies cognitive plasticity; however, Stern's concept of cognitive reserve would suggest that cognitive reserve can exist even when neural plasticity has been compromised. Neural plasticity refers to the capacity of neural circuits to change in response to fluctuations in neural or glial activity (Kemperman, Gast, & Gage, 2002) and is associated with changes in synaptic connections between neurons, addition of new neurons (neurogenesis), increased myelination of axons, or change in the size or shape of a neuron. Neural organization and hence plasticity can occur at multiple levels—from molecules and synapses to cortical maps and large-scale neural networks (Buonomano & Merzenich, 1998; Garlick, 2002).

Neural plasticity has usually been studied through the neuroimaging methods of positron emission tomography (PET) and functional magnetic resonance imaging (fMRI). As Poldrack (2000) suggests, our conception and current understanding of neural plasticity may be at least partly a function of these methods used to study the concept. In fact, PET and fMRI techniques provide indirect measures of synaptic activity by examining brain function via measurement of blood oxygenation and blood flow. Hence, our current knowledge of neural plasticity is focused primarily at the level of changes in synaptic activity and cortical maps. Thus, our understanding of the interrelation or interaction between various levels of neural organization and cognitive plasticity is limited. Or, as Kramer et al. (2004) stated, "There is a gap in our knowledge with regard to identifying global changes in the brain" (p. 941). For example, there is limited understanding at present of the relation between synaptic activity as represented in functional imaging signals and the lower levels (e.g., biophysical and molecular) of neural organization. Likewise, current understanding of the relation between brain function as represented in synaptic activity and brain structure or morphology is limited (Poldrack, 2000). While much of the research on neural plasticity has

come from functional imaging techniques, there is also some research based on size of brain structure. For example, significant relations between white matter lesions, cerebral atrophy, and low levels of education were found in a sample of nondemented community-dwelling elders (Koga et al., 2002).

With regard to brain function, PET and fMRI research has resulted in at least two tentative general findings (Kramer et al., 2004); alternative explanations are now offered for each finding. First, findings indicate that older adults show lower levels of activation in a wide variety of tasks and brain regions. While one explanation of reduced activation holds that aging is associated with loss of neural resources, another explanation is that neural resources are available but not adequately recruited. Instruction in strategy use, for example, has been shown to reduce underrecruitment (Logan, Sanders, Snyder, Morris, & Buckner, 2002).

A second general finding is that older adults exhibit nonselective recruitment of brain regions (Kramer et al., 2004). Older adults, compared to young adults, show recruitment of different brain regions in addition to those activated in younger adults. This observation of bilateral activation has led to the hemispheric asymmetry reduction in older adults (HAROLD) model of neurocognitive aging (Cabeza, 2002; Hayes & Cabeza, 2008). The model suggests that cortical activity tends to be less lateralized in older than younger adults. There is current debate as to whether additional activity observed in older adults is compensatory or a marker of cortical decline. There is contradictory evidence whether older adults who perform better on a task show bilateral recruitment; some studies report this finding, while other studies do not. A limitation in examining this phenomenon has been that MRI studies use indirect measures of activation that depend on the relative activation compared to activation in surrounding areas. Thus, smaller brain structures would suggest different activation patterns even when the active structures are similar. Another limitation is that most studies have been comparisons across different subjects; future research needs to examine intraindividual changes in performance-brain activation patterns across various tasks, across longer developmental periods, and within training conditions (Kramer et al., 2004).

#### **Neural Plasticity: Availability, Reconfigurability, and Customizability**

To integrate the existing conceptual approaches and to explain the empirical findings, Mercado (2008) has recently argued that three key processes impact cortical modules and thus indirectly neural and cognitive plasticity: availability, reconfigurability, and customizability. Each process may vary between and within individuals, and the interplay between these processes may explain why there is no simple relation between any single indicator of plasticity and plasticity in complex abilities and intellectual functioning. In addition, Mercado attempts to integrate three factors previously associated with intellectual capacity (brain size, prefrontal cortex, and neural speed or efficiency; Deary, 2000).

*Availability* refers to the number and diversity of cortical modules that are available for differentiating stimulus representations (Mercado, 2008). Larger brain regions provide room for more complex circuitry, more dendritic expansion, more synapses, thicker myelin, more neurons, and larger neurons—all of

which increase functional capacity. Brain size limits the maximum number and diversity of cortical modules and constrains cortical organization (Jerison, 2002). Prior explanations of the fact that bigger brains are related to cognitive capacity have focused on amount of additional cortical tissue or the overall computational power as estimated by the number of neurons or synapses. In contrast, Mercado suggests that the amount of tissue and neurons is less critical than the manner in which circuits within the tissue are organized; it is the resolving power that determines capacity rather than general information processing capacity. Environmental domains, furthermore, are believed to increase the diversity of cortical modules available (Kahn & Krubitzer, 2002).

Second, *reconfigurability* refers to the brain's ability to flexibly develop new configurations of cortical modules and to switch rapidly between them as a function of task demands (Crone, Donohue, Honomichi, Wendelken, & Bunge, 2006; Mercado, 2008). This refers to the flexibility in using the same modules for different cognitive tasks and even within tasks as one progresses in learning increasingly better ways to perform the task. In fact, different brain regions serve different roles at different stages of cognitive skill learning (Crone et al., 2006; Doeller, Opitz, Krick, Mecklinger, & Reith, 2006; Pauli et al., 1994; Poldrack, Prabhakaran, Seger, & Gabrieli, 1999). The involvement of any particular subset of brain regions in cognitive skill acquisition depends on task difficulty, an individual's level of expertise, and the particular task being performed. In addition, the specific set of cortical regions that become active during acquisition may depend on the specific strategies used. In addition, there are clear indications that as people age they tend to recruit neural circuits differently than they did as younger adults (HAROLD; Cabeza, 2002) and that it might actually be adaptive to use different neural circuits across task trials to perform well in the same cognitive task (Reichle, Carpenter, & Just, 2000). Reconfigurability is associated with maintenance and control of representations; of particular relevance, then, are the frontal lobes, which are considered the seat of cognitive control, supporting capacity through flexible coordination of decision processes and memory.

Third, *customizability* refers to the brain's capacity to dynamically adjust the selectivity of cortical modules based on experience (Mercado, 2008). That is, the degree to which repeated experience in turn shapes the structure of the brain to perform well-learned tasks more efficiently or more accurately. Thus, plasticity on the level of the brain presumes that specialized cortical modules throughout the brain can be used in a variety of combinations (and somewhat interchangeably) to enable the acquisition and performance of cognitive skills. Variability in cognitive plasticity across individuals of different ages also reflects variability in their capacity to reconfigure cortical modules. Evidence indicating that individuals with greater intellectual capacity may be able to configure their neural circuits more flexibly has come primarily from electroencephalographic studies in humans (Jausovec & Jausovec, 2000; Thatcher, North, & Biver, 2005).

Mercado differentiates between the older concept of neural *efficiency* and neural *plasticity* or customizability. The assumption critical to the neural efficiency model was that differences in efficiency accounted for variability in cognitive capacity. Faster brains that could process information more efficiently should have greater capacity. Mercado (2008) argues that the focus on neural



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efficiency has receded and has been replaced by the role of neural plasticity in relation to behavioral flexibility. According to Mercado, neural plasticity involves not only making connections stronger and more efficient but also enhancing the ability to reallocate and retune circuits. Neural plasticity may determine how quickly an individual can adapt to new situations. Different abilities require different connections across circuits; neural plasticity guided by experience establishes the patterns of connection. Different levels of neural plasticity across individuals could impact the number and complexity of neural connections—this could then affect processing speed and neural efficiency. The number and diversity of cortical circuits engaged during learning can change as a function of experience.

A major question regarding neural plasticity is the issue of how experience or cognitive stimulations impacts the brain and enhances brain functioning. Kramer et al. (2004) have suggested two alternative hypotheses regarding the relationship between cognitive stimulation including training and plasticity in neural structure and function. On the one hand, enhanced neuronal structure and brain function may occur as a result of additional environmental stimulation and play a protective function against neuronal degradation (Fillit et al., 2002). Alternatively, enhanced neuronal networks fostered through cognitive experiences may delay cognitive decline even in the face of morphological and functional deterioration in the aging brain; the later hypothesis seems congruent with Stern's (2007) concept of cognitive reserve.

A major question focuses on the directionality of the relation between cognitively stimulating activities and brain reserve: to what degree do education or other forms of cognitive reserve represent initial brain capacity versus to what degree does brain capacity reflect the effects of cognitive stimulation? Experimental studies with animals in which cognitive stimulation is manipulated provide one design for examining the directionality of relations between neural plasticity and cognitive stimulation (Kemperman, Kuhn, & Gage, 1997; Kramer et al., 2004). In animal research, neurons and synapses and also glial cells that permit and enhance neuronal function have been shown to be altered though cognitive stimulation, with greater effects in young animals but also occurring albeit more slowly in mature animals. Neurogenesis associated with cognitive stimulation has been found in the hippocampus of animals and humans both in young and in old age (Eriksson et al., 1998). Some support for neurogenesis in other cerebral cortical has been reported but is debatable.

There are a number of important questions regarding the relation between neural and cognitive plasticity that have not been fully answered (Kramer et al., 2004). Does cognitive experience at earlier ages affect the potential for neurogenesis at later ages that might provide reserve capacity? How long do newly generated hippocampal neurons in adults survive? Is their longevity dependent on continued environmental stimulation? Are there enough newly generated neurons at a given time to contribute to improved behavioral performance? How are these new neurons integrated morphologically and functionally with the existing neural networks?

The concepts of availability, reconfiguration, and customization are complemented by the cortical "disconnection" hypothesis (Hayes & Cabeza, 2008; O'Sullivan et al., 2001). Cortical disconnection is hypothesized to lead to a loss of functional integration of neurocognitive networks. A plausible anatomical

substrate for functional disconnection is disruption of the white matter tracts that link the components of distributed cognitive networks. The capacity of a brain region such as the prefrontal cortex to become activated may not be attenuated by age; rather, the coordination of the whole neural response is impaired, implying a loss of functional connectivity. However, functional imaging techniques provide only indirect evidence of cerebral disconnection and depend on the assumption of a linear relation between neural activity and hemodynamic response that may not hold in elderly subjects. In relation to plasticity, reorganization of large, distributed cognitive networks is likely to depend on integrity of white matter tracts.

Just as there are multiple types of plasticity at the neural level, there are also multiple types of cognitive abilities/processes at the behavioral level. The interplay between different neural levels of plasticity and various cognitive abilities is an important area of inquiry. For example, there may be differential effects with regard to declarative (conscious, explicit memory) versus nondeclarative memory (not dependent on conscious processes, such as skill learning). A challenge is presented by the fact that different types of memory may involve very different underlying neural mechanisms. While declarative explicit memory relies on the medial temporal lobe (hippocampus), nondeclarative memory is not dependent on the medial temporal lobe and may be associated with different neural substrates depending on the nature of the task. For example, individuals learning a finger-tapping sequence consciously engage very different sets of brain regions than individuals learning the same sequence implicitly (nondeclarative memory), even though the behavioral demands of the task are identical (Grafton, Hazeltine, & Ivry, 1995; Poldrack, 2000).

Based on the what we have stated so far with respect to neural plasticity, we take the position throughout the rest of this chapter that it is important to go beyond a single-indicator approach to plasticity. That is, any theory about plasticity needs to explain the limiting conditions of (a) existing structures (e.g., the brain), (b) specialized substructures (e.g., particular circuitry used in the brain), (c) potential adaptation in the recruitment of substructures to manage higher-order tasks (e.g., flexibility in recruiting different networks to achieve the same result), and (d) potential change in structures and recruitment due to environmental stimulation.

### Behavior Plasticity

Cognitive plasticity has been examined at multiple behavioral levels, just as neural plasticity involves multiple levels. Cognitive training research has focused on cognitive processes (e.g., processing speed and inhibition; Ball et al., 2002; Jones et al., 2006), primary mental abilities (e.g., inductive reasoning, spatial orientation, and episodic memory; Schaie & Willis, 1986), higher-order cognitive constructs (e.g., fluid intelligence and executive functioning; Jaeggi et al., 2008), and global cognition involving multiple cognitive domains (Fried et al., 2004). In addition, the impact of noncognitive interventions (e.g., exercise and nutrition; Colcombe & Kramer, 2003) on cognition has been examined.

Most behavioral cognitive interventions have focused on processes and abilities previously shown in longitudinal research to exhibit relatively early age-

related decline. These interventions have focused on memory, executive functions, and other cognitive abilities. The most targeted regions are those that show the greatest atrophy and functional decline. The effectiveness of these interventions is still under investigation.

### Indices

Earlier in this chapter, we have discussed how individual differences in cognitive abilities and skills can be measured. This section discusses the number and types of cognitive skills that are used in the behavioral indices of cognitive decline (asymptotic performance).

Life expectancy is a measure of the performance of the cognitive system. It indicates the number of tasks that are completed over the intended lifespan. The behavioral indices of cognitive decline are examined using a method that is similar to the method used for the present study. The further the opportunity for cognitive decline is delayed (e.g., the later the onset of cognitive decline), the more improved the conditions. The behavioral indices serve to provide a measure of cognitive decline that is more than the method used in the present study (e.g., Ball et al., 2002; Jones et al., 2006) in the development of the behavioral indices of cognitive decline (e.g., Jones et al., 2006) in the development of the behavioral indices of cognitive decline. The behavioral indices of cognitive decline are found in the behavioral indices of cognitive decline. The behavioral indices of cognitive decline are found in the behavioral indices of cognitive decline.

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related decline or to be associated with cognitive impairment. Thus, interventions have focused on fluid and process-based abilities (reasoning, speed, working memory, and executive functioning). There has been a parallel between the abilities targeted for training and the brain areas and structures of interest. The greatest atrophy in cortical volume has generally been reported for the prefrontal regions and somewhat smaller atrophy for the temporal and parietal areas. Executive control, fluid abilities, and some memory processes that have been the target of intervention are supported by prefrontal and temporal regions of the brain.

### Indices of Plasticity at the Behavioral Level

Earlier in this chapter, we noted that cognitive plasticity has most commonly been conceptualized as an individual's latent cognitive potential or the individual's cognitive capacity under certain specified conditions. Hence, observable indicators or behavioral indices of cognitive plasticity are needed. For a number of cognitive researchers (Harlow, 1959; Thorndike, 1911), key behavioral indicators of intellectual capacity include the capacity to learn a cognitive skill, the rate at which the skill is learned, and the highest performance (asymptote) reached (Zimprich, Rast, & Martin, 2008).

Life span theorists (Baltes, 1987) have identified three levels of performance that provide a profile of an individual's plasticity. *Baseline performance* indicates the individual's initial status (level) of performance on cognitive tasks without intervention or support. *Baseline plasticity* refers to the extended range of possible performance (performance improvement) when additional resources are provided. For example, this form of plasticity has been examined shortly after participants are taught memory strategies, such as the method of loci. This level of plasticity involves brief instruction in a strategy or the provision of a cognitive resource but little or no practice in use of the strategy. *Developmental reserve capacity* or *developmental plasticity* refers to the further range of performance improvement that occurs as a result of the opportunity or context within which cognitive resources can be fully activated (e.g., through extensive practice that optimizes strategy or cue utilization). Recent training research has compared the range of performance improvement under baseline plasticity versus developmental plasticity conditions. A greater range of plasticity was found under the developmental reserve plasticity condition (extended practice with the strategy or mnemonic) than the baseline plasticity condition in memory training research using the method of loci (Brehen, Li, Muller, von Oertzen, & Lindenberger, 2007; Jones et al., 2006). It was suggested that the greater range of plasticity shown in the developmental plasticity condition is due to information binding (Craik, 2006) involved in use of the method of loci strategy, that is, the formation of associations between novel words to be remembered and different loci in a certain sequence. Functional imaging during the various phases of the training study indicated that greater activation in the medial temporal lobe was found for participants who exhibited greater plasticity during the developmental reserve capacity phase; activation of the medial temporal lobe was hypothesized to be related to formation of associations and with information binding.

## Cognitive Training and Behavioral Plasticity

The contexts in which behavioral cognitive plasticity have been most commonly studied has been the behavioral training studies that target those fluid- and process-based abilities that exhibit relatively early age-related decline (Kramer & Willis, 2003). Several criteria have been involved in evaluating the effectiveness of cognitive training, and these criteria are of interest in the study of cognitive plasticity (Ball et al., 2002).

*Magnitude of performance improvement* on the target ability of training for the intervention group in comparison to control groups has been the primary outcome of interest and thus an indicator of the range of cognitive plasticity that could be evoked under the intervention condition. A testing-the-limits condition has been used in some training studies to examine the asymptotic level of training improvement under increasingly demanding conditions. For example, in a series of memory training studies using the method of loci, asymptotic level was assessed by increasing the speed of word recall (Kliegl, Smith, & Baltes, 1989) or by engaging in dual tasks of walking and memory recall (Li, Lindenberger, Freund, & Baltes, 2001).

Although cognitive plasticity is concerned with change at the intraindividual level, most training studies have reported performance improvement or plasticity at the level of the group mean; hence, information on the proportion of individuals exhibiting reliable intraindividual change is obscured, as is the absolute range of plasticity and asymptotic levels attained. Given that plasticity is an intraindividual concept, comparison of the individual's performance after training with performance at earlier developmental periods prior to training would be optimal. However, longitudinal data on intraindividual change are rarely available. Comparison of asymptotic levels across age-groups is problematic given that the groups may differ on factors other than age that affect plasticity.

A second criterion in training studies has focused on the maintenance of training effects. It is argued that temporal durability of effects is required in order for training outcomes to be meaningful and of lasting benefit. Although the number of training studies examining long-term maintenance has been limited (Jones et al., 2006), temporal durability of training effects on primary mental abilities have been reported for 5 years within a clinical trial (Willis et al., 2006) and up to 14 years after training in smaller studies (Boron, Willis, & Schaie, 2007; Willis & Nesselroade, 1990).

A third criterion and one that is currently of particular interest focuses on training transfer. Until recently, most training studies have focused on a single cognitive process (verbal memory, inhibition) or primary ability (reasoning), and the original interest was in the magnitude of training effects and the asymptote achieved. However, there has been increasing interest in the breadth of transfer achieved. Near transfer has focused on demonstration of training effects to one or more indicators of the ability trained; there is consensus that training does result in near transfer. However, there is much less consensus or empirical evidence for far transfer, that is, demonstration of training effects on cognitive processes or abilities that are conceptually and empirically distinct from the cognitive target of training.

A major limitation to the study of transfer has been lack of consensus on how to define or assess varying levels of transfer. Definitions of transfer have

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varied from similarity of the stimuli used in assessment materials to similarity in strategies used in various cognitive measures to the requirement for assessment of transfer at the latent construct level, demonstrating common shared variance across various indicators. We discuss this further in the section on methods to examine plasticity.

The second (durability) and third (transfer) criteria discussed with regard to training studies are of interest with regard to the concept of cognitive plasticity. Although cognitive plasticity has been most commonly studied in the context of training studies, definitions of cognitive plasticity have not focused on issues of durability or transfer. Indeed, in studies of neural plasticity, increased activation is usually assessed for very limited time periods. Kramer et al. (2004) have alluded to issues of durability and transfer in the numeration of questions still to be addressed: how long do newly generated neurons survive; do new neurons remain functionally specialized, or do they become functionally generalized; and how are new neurons are integrated with existing neural networks?

*Dose-response* relations are a fourth issue in training research related to cognitive plasticity. The question is whether the range of plasticity varies with the length or intensity of the treatment. Most training studies have been relatively brief, and thus the dose-response issue has received limited attention. Several studies have shown an increased magnitude of effect (range of plasticity) with booster sessions that supplemented the initial intervention (Ball et al., 2002; Jaeggi et al., 2008; Willis et al., 2006). Some recent studies also suggest that there may be age differences in dose-response relations (Brehmer, Li, Müller, Oertzen, & Lindenberger, 2007; Jones et al., 2006).

### Cognitive Plasticity in Dyads

Definitions of cognitive plasticity have typically focused on intraindividual change. However, recent research on collaborative cognition has examined the range of enhancement of cognitive performance-associated dyadic cognitive activity or problem solving. It is possible that collaborative cognition could be considered a form of baseline plasticity within Baltes's levels-of-plasticity approach. Collaboration with others provides resources for enhancing one's range of performance in a manner similar to use of cognitive strategies. It appears that the effects of cognitive collaboration may be positive and facilitative of elders' everyday cognitive performance on a variety of tasks (i.e., prose recall, wisdom-related advice giving, comprehension of printed materials, and route planning/everyday life management; Strough & Margrett, 2002). At the same time, positive effects appear to be facilitated by having familiar social partners (Gould, Kurzman, & Dixon, 1994), explicit collaborative instructions, and tasks that do not rely on immediate memory recall. In addition, further research conceptualizing individual plasticity as embedded within the spousal or intergenerational dyads may be helping to explain individual differences in plasticity (Martin & Wright, 2008).

### Sociocultural Plasticity

Much of the conceptualization and empirical work on cognitive plasticity has focused on neural plasticity examined over very brief intervals or on performance enhancement in behavioral interventions that span days, weeks, or

at most, months. However, cognitive plasticity can also be considered at the phylogenetic or sociocultural level as included in the biocultural constructivist approach discussed previously (Li, 2003).

Cohort-sequential longitudinal studies comparing the level of cognitive functioning of different birth cohorts at the same chronological age have suggested a range of cognitive plasticity that equals or exceeds that found across the ontogeny of a single individual, on average (Schaie, 2005). For example, there has been an average increase in reasoning ability performance on the order of over 1.5 standard deviations when comparing the performance of the 1896 birth cohort to the 1973 cohort. While inductive reasoning exhibits the largest positive cohort trend, positive cohort trends have also been shown for some spatial orientation and perceptual speed tasks. In contrast, number ability has exhibited curvilinear cohort trends. Significant or "massive IQ gains" on the order of 5 to 25 points within a single generation have been reported by Flynn and known as the "Flynn effect" (Dickens & Flynn, 2001; Flynn, 1984, 2007). Flynn and colleagues have reported that the largest cohort differences in intellectual functioning have been found for what are commonly known as fluid abilities. Less or no cohort gains have been found for acculturated skills acquired through schooling and commonly known as crystallized intelligence.

From a life span perspective, the question arises whether the findings of massive IQ gains (Flynn effect) represent a phenomenon unique to a specific historical period and to the post-World War II cohort or whether they are indicative of a long-term societal or evolutionary change. When cohort differences are examined over a broader historical period and a wider range of cohorts, the phenomenon of cohort gain in intellectual performance becomes more complex than described by the Flynn effect (Schaie, 2005; Schaie, Willis, & Pennak, 2005). Both fluid and crystallized abilities have exhibited significant positive gains, particularly in the early 1900 birth cohorts. The magnitude of cohort gain appears to have been greater in the early 1900s than that cited for the post-World War II cohorts. In addition, positive, negative, and curvilinear cohort trends have been observed. Moreover, cohort trends vary for different abilities within the same historical period.

Those studying cognition from a broad coevolutionary perspective propose that advances in cognition as would be represented in cohort and generational effects are due primarily to an accumulation of cultural resources and knowledge across time. This perspective has been largely nondevelopmental. It is concerned primarily with secular trends in level of cognition but with little consideration of how culture impacts developmental change.

Dickens and Flynn (2001) and Flynn (2007) have proposed that individuals' environment is largely matched to their IQ level. Through a *multiplier effect*, an individual with a higher IQ either seeks or is selected for a more stimulating environment, leading to further increases in IQ. The impact of small environmental changes could result in significant IQ gain due to the multiplier effect. By a similar process, a social multiplier effect can occur if intellect increases by a small amount for many persons in a society and leads across time to further reciprocal interactions between ability and environment. Increase in a person's IQ is thus influenced not only by his or her environment but also by the social multiplier effects occurring for others with whom the person has contact. The

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question remains of what determines the domain of development or cognition that is impacted by culture and environment.

Drawing on Darwin's work, Flynn suggests that an *X factor* may determine those aspects of cognitive development that are impacted by the environment (Dickens & Flynn, 2001). The X factor need not be inherently related to the developmental domain impacted. For example, introduction of technology (e.g., use of menus or linear thinking) might increase public attention and use of computer-based or electronic devices, leading to increased inductive reasoning. In the Schaie (2005) model, the X factor is represented as a period effect.

In a related coevolutionary approach, Tomasello and others (Tomasello, 1999) have proposed mechanisms for social transmission of cultural knowledge. Humans have evolved forms of social cognition unique to humans that have enabled them not only to create new knowledge and skills but also, more important, to preserve and socially transmit these cultural resources to the next cohort/generation. Cultural learning thus involves both social transmission of cultural knowledge and resources developed by one person and also sociogenesis or collaborative learning and knowledge creation.

### Time Frame: Short-Term and Long-Term Plasticity

The sampling of time will influence analysis and interpretation of intraindividual plasticity and variability (Martin & Hofer, 2004). Indeed, we might expect that different intervals will yield different patterns of plasticity and result from different influences on the individual. For example, we might consider sampling moment to moment (attentional lapses), within test (fatigue and practice), within session (fatigue, order effects, and motivation), within day (time-of-day effects), across days or weeks (environmental perturbations, physical health, and practice), or months or years (characteristic change trajectory). In many cases, short-term plasticity or change contains meaningful information about psychological processes of interest. In fact, there may be many different manifestations of short-term plasticity and several possible theoretical interpretations. For example, intraindividual short-term variability in functioning may be an early indicator of deficits (Schaie, 2000) or later-onset declines.

With respect to the differentiation–dedifferentiation (Li, 2002) or the “common cause” hypothesis, intraindividual short-term variability might be indicative of the structure of aging-related changes. Intraindividual short-term plasticity could also indicate adaptive behavior to cope with a stressful event or the limitations that go along with physical ailments. As an example, an increase in short-term intraindividual variability in extreme old age is not necessarily maladaptive. Maintaining low levels of variability in developmental outcomes such as well-being or health requires a high level of regulatory effort. If, however, individual energy levels are low (e.g., Fairclough, 2001), it might be maladaptive to invest energy in the stability of a particular outcome variable. Given limited resources, such efforts might affect functioning in other life domains, such as social activities. Instead, setting wider margins between a maximum and a minimum value for the variability of a particular outcome variable may reduce the effort required to balance functioning in both domains. As a

consequence, within- and across-domain short-term intraindividual variability might increase, and this range of plasticity indicates a successful adaptation with respect to the outcome of well-being.

Theories of development that attempt to explain age-related changes within individuals need to consider specifications of which short- and long-term intraindividual change relations are theory relevant and to which time scales predictions apply (Schaie, 1989a, 1989b). In addition to specifying that there should be age-related changes in both explanatory concepts (e.g., in speed) and aging-related outcomes (i.e., fluid intelligence), it is possible to specify what longitudinal short- and long-term relations are to be expected or if the theory even applies to particular time frames of change. To illustrate this point, one may consider the fact that to solve a new cognitive task, it is most adaptive to quickly acquire or learn the required skills. Thus, high levels of plasticity are expected in individuals coping best with the environment. However, several studies have reported that larger variability in performance in the long term may be a predictor of individuals at risk for strong cognitive declines and even dementia. Thus, any theory of plasticity needs to specify the relevant time frames because the explanations and predictions for short-term plasticity may not be the same as those for long-term plasticity. In this context, hypotheses about within-person changes need to be formulated and tested separately from hypotheses about population mean changes (e.g., Zimprich & Martin, 2002).

So far, there is little research on systematic intraindividual aging-related short-term plasticity, that is, on the interaction between short-term intraindividual plasticity of explanatory parameters and intraindividual changes in other short-term developmental outcome parameters (e.g., Li, Aggen, Nesselroade, & Baltes, 2001; Nesselroade, 2001; Strauss, MacDonald, Hunter, Moll, & Hultsch, 2002). There is also very little research on the predictive value of short-term intraindividual plasticity on later short-term variability, long-term changes, or long-term outcomes. With adequate research designs, the examination of short-term intraindividual plasticity offers new possibilities for developmental research in many domains of psychological functioning. Results from these study designs may help answer questions about the generality versus specificity of plasticity and about age-related differences in the range of observable intraindividual short-term plasticity. In the long run, these designs might add to the specificity of theories and predictions and support the development of tools allowing the early detection of problematic development.

## Processes and Mechanisms Associated With Plasticity

Traditionally, in the study of both brain and behavior, the focus has been primarily on distinct, relative narrow domains, such as regions of interest in the brain, localized areas of activation, and specific primary mental abilities. However, there is growing awareness that cognitive functioning either in brain

or in behavior needs to be understood in terms of networks or maps rather than in terms of cognitive abilities or structures. The concept of connectivity maps or maps of connectivity must be seen as underrecruitment of resources, as seen as determined by the organization of cortical maps. Research has shown that different roles at different levels of any particular system depend on task difficulty. For example, similar tasks being performed by different parts of the brain may be related to different cognitive strategies (e.g., Craik, 2006) of intelligence. The concept of plasticity shown in the brain has been attributed to individual cognitive strategies or to the strengthening of connections and the organization of connectivity maps.

Cognitive and neural plasticity and customization of cognitive abilities (representing customization to overcome a greater focus on flexible cognitive approaches to the study of concepts of competence) may use alternative cognitive strategies or functional deficits to customize one's cognitive abilities.

Availability of neural plasticity (works) appears to be related to the noted by Mercado (2002) that more synapses in the brain provide the factors provide the leading to greater efficiency or speed of plasticity. The creation of stronger and more efficient for flexibility to recruit different abilities required by experience.

It is becoming clear that unidirectional relationships between brain do not always exist. Experience and behavior influence neuronal structure and stimulation. An important this reciprocity between



or in behavior needs to be conceptualized as involving connectivity and networks or maps rather than isolated abilities and brain regions. Relating specific cognitive abilities to specific brain regions is overly simplistic. Likewise, the role of connectivity must be given greater attention in the study of activation and underrecruitment in functional imaging; activation in any region must be seen as determined partially by the capacity for connectivity across broad cortical maps. Research findings suggest that different brain regions serve different roles at different stages of cognitive skill learning. The involvement of any particular subset of brain regions in cognitive skill acquisition depends on task difficulty, an individual's level of expertise, and the particular tasks being performed. The specific set of cortical regions that become activated may depend on the specific strategies used. Finally, connectivity may be related to the concept of information binding (Brehen et al., 2007; Craik, 2006) of interest in cognitive training research. The increased range of plasticity shown in Baltes's developmental cognitive reserve condition has been attributed to information binding (i.e., extensive practice in use of cognitive strategies or cognitive resources); this extensive practice may involve strengthening of certain connections or perhaps reconfiguration or customization of connections.

Cognitive and neural connectivity also underlies the capacity for reconfiguration and customization. A broad network or map of cortical modules or cognitive abilities (representing connectivity) must exist for reconfiguration and customization to occur. Reconfiguration and customization are instances of a greater focus on flexibility and adaptability that characterize more recent approaches to the study of development and plasticity. A central feature of the concepts of compensation and active reserve is the individual's flexibility to use alternative cognitive processes or neural resources to adapt to pathology or functional deficits. Compensation represents the ability to reconfigure and customize one's cognitive resources in the face of new challenges.

Availability of neural structures (e.g., neurons, synapses, and cortical networks) appears to be a necessary but not sufficient condition for plasticity. As noted by Mercado (2008), larger brain regions, more neurons (neurogenesis), or more synapses in themselves may not result in greater plasticity. Rather, these factors provide the limiting conditions for increased larger cortical networks leading to greater functional capacity and resolving power. Likewise, neural efficiency or speed of processing per se may not be the most critical feature of plasticity. The critical feature in connectivity is not solely the presence of stronger and more efficient connections but also the specific capacity required for flexibility to reconfigure and customize connections and cortical maps. Different abilities require different connections across circuits; neural plasticity guided by experience establishes the patterns of connection.

It is becoming increasingly evident that there is a reciprocal rather than unidirectional relation between brain, experience, and behavior. Changes in the brain do not always lead in a unidirectional manner to changes in behavior. Experience and behavior also significantly impact changes in the brain. Enhanced neuronal structure and brain function may occur as a result of environmental stimulation. An important task for future research is further explication of how this reciprocity between brain, experience, and behavior functions.

## Developmental Issues and Plasticity

### Sensitive Periods in Development

"Critical" or sensitive periods have been of interest in both neural and behavioral development and have been closely associated with learning and cognition. Traditionally, the concept of "critical" or sensitive periods included the conditions that learning or plasticity occur over a short, sharply defined period in the life cycle and that this learning was subsequently irreversible (Thomas & Johnson, 2008). However, more recent findings suggest that critical periods may not necessarily be sharply timed or irreversible; thus, "sensitive periods" rather than "critical periods" is the more commonly used terminology. Moreover, there appear to be multiple sensitive periods within a specific domain. For example, within the auditory domain, there are different sensitivity periods for different facets of speech processing and for music perception.

Sensitivity or plasticity is reduced in lower-level systems before it is reduced in high-level cognitive systems (Huttenlocher, 2002; Thomas & Johnson, 2008). Synapses, the structures through which neurons communicate, are initially overproduced in the brain, and the environment selects which ones are retained to support function. The density of synapses has been considered to be one indicator of plasticity since experience can alter connective strength of synapses. Synaptic density function peaks at different times in different regions of the brain, with the prefrontal cortex (middle frontal gyrus) showing the latest peak in synaptic density; this region is associated with higher-level cognition.

### Developmental Differences in Plasticity

A major question in current research on sensitive periods focuses on the explanatory mechanisms that may underlie reduction in plasticity across development (Thomas & Johnson, 2008). Three alternative explanations are (a) termination of plasticity due to maturation, (b) self-termination of learning, and (c) stabilization of constraints on plasticity (without a reduction in the underlying level of plasticity). The first alternative suggests that reduction in plasticity is due to changes in neurochemistry of a particular brain region, resulting in an increased rate of pruning of synapses and thus the "fossilization" of existing patterns of functional connectivity. The second and third explanations, in particular, may be of most interest with respect to cognitive plasticity in the later part of the life span. The second explanation argues that the process of learning itself can produce changes that may reduce plasticity in a system (Thomas & Johnson, 2006). Learning could reduce plasticity in several ways. First, learning could lead to neurobiological changes that reduce plasticity. Alternatively, prior learning may place a heavy demand on the neural system's computational resources such that new learning would have to compete for these resources, thus limiting the potential for new learning. A third option is that prior experience or learning puts the neural system in a nonoptimal state for new learning—that is, additional time would be required to reconfigure the system, and thus new learning would take longer than if the prior learning or experience had not occurred. The third explanation suggests that there may not be an end to plasticity per se but that the

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potential for plasticity may be “hidden” because of certain constraining factors; these constraining factors become increasingly stable with development. This third explanation may be useful in understanding evidence for certain types of plasticity observed in adulthood. For example, connectivity between various sensory systems (tactile and visual) appears to dissipate significantly with age; however, when sighted persons are deprived of vision, there is a sudden and sharp increase in activation of the visual cortex during tactile perception. This activation suggests that some level of connectivity (plasticity) remains but is not activated until visual input is constrained.

Recent findings in research on second-language acquisition provides insight into several issues regarding developmental differences and similarities in cognitive plasticity across the life span (Birdsong, 2006). First, adults and young children appear to differ in how they acquire a second language. Young children generally acquire a second language through large amounts of exposure to the new language. In contrast, second-language learning in adults appears to be most efficient when they adopt explicit strategies for learning the language and when they are more responsive to feedback than children. As a result, adults often learn a second language at a faster pace than young children. Indeed, there appears to be little evidence for total loss of plasticity to acquire a second language in adulthood. However, the final level attained is generally lower the older the age of acquisition. Second-language acquisition also provides insight into the association of prior learning (first-language acquisition) on future plasticity. While early research suggested that different areas of the cortex were activated for a second language, recent research findings indicate that the higher the level of competence in the second language, the more likely that the same cortex areas will be activated as for one’s first language. This suggests that certain brain areas have become optimized for processing language (perhaps during acquisition of the first language) and that increased proficiency in the second language tends to activate the same brain areas. Thus, subsequent plasticity may be tempered by the processing structures created by earlier learning. Earlier in this chapter, we also discussed plasticity in adulthood as involving reconfiguration and customization of neural networks (Mercado, 2008).

Plasticity in adulthood may also vary across the multiple subskills involved in a complex task, such as second-language acquisition, as a result of sensitivity or plasticity being reduced in lower-level systems before it is reduced in high-level cognitive systems (Huttenlocher, 2002). For example, there may be greater or earlier loss of plasticity in phonology than in lexical semantics. Thus, the older second-language learner may find acquisition of new vocabulary easier than acquiring new sounds or new grammar.

In summary, developmental differences in cognitive plasticity may not be solely or even primarily associated with maturational changes, such as loss or deterioration in basic neural structures or functions. Rather, prior learning and experience may play a critical role in determining both the range of cognitive plasticity later in the life span as well as the mechanisms involved. Prior learning may reduce the availability of neural computational resources or may reduce the flexibility of cortical networks required for new learning. In earlier sections of this chapter, the important role of reconfiguration and customization of cortical modules and networks for continued cognitive plasticity

in adulthood was discussed (Mercado, 2008). An important area of future research would be to examine in greater detail, at both the neural and the behavioral level, the role and impact of prior learning and experience on subsequent cognitive plasticity.

### Methodological Issues in the Study of Plasticity

To examine individual-level plasticity, research designs must be sensitive to all types of plasticity in order to identify patterns and magnitudes of plasticity within individuals. Given both systematic and stochastic sources of fluctuation in individual characteristics over short periods of time, such designs must also be sensitive to intraindividual variation (Nesselroade, 1991). Temporal sampling designs can take many forms. One such design, the measurement burst design (Nesselroade, 1991; Nesselroade & Schmidt-McCollam, 2000), utilizes intensive measurements over a short period in time and follow-up with further measurement burst sessions after longer intervals and permits a window on individual-level characteristics as well as long-term change. Obtaining multiple, closely spaced assessments at each wave allows local temporal smoothing of data for each individual by averaging across multiple assessments. The addition of multiple indicators at each assessment permits further improvements in the modeling of random error components in models that incorporate true change. By improving measurement precision through multiple assessments or multiple indicators, such designs will increase statistical power to detect cognitive change at both the individual and the aggregate sample level and permit greater understanding of intraindividual processes within and across different time intervals. Indeed, measurement burst designs incorporating multiple indicators would permit an optimal opportunity for evaluating systematic short-term fluctuation and change. A further consideration is the context in which measurements are obtained. Most temporal sampling designs are performed under random contextual backgrounds, but individual variation in performance can be evaluated against defined contexts as well—more akin to experimental paradigms (e.g., levels of stress and dual-processing tasks).

Analytically, different types of plasticity can be distinguished. Each type is related to different theoretical assumptions and explanations. Structural plasticity can be found on the level of changes in covariation patterns among variables across time. That is, the covariation patterns of different intellectual abilities within persons may change over time, suggesting that more or fewer factors are sufficient to explain observed behaviors. Empirically, Schaie, Maitland, Willis, and Intrieri (1998) investigated longitudinal measurement invariance of six primary mental abilities (inductive reasoning, spatial orientation, perceptual speed, numeric facility, verbal ability, and verbal recall) across a 7-year period in a sample of 984 individuals, disaggregated into six age-groups (32, 46, 53, 60, 67, and 76 years at first testing). They found some structural plasticity in the older age-groups suggesting that in fact the covariation patterns of abilities change in old age. Theoretically, it remains an open question as to how this finding can be explained. If fewer factors suffice to explain the structural relations in old age as the dedifferentiation hypothesis would suggest (e.g., Anstey, Hofer, & Luszcz, 2003; Ghisletta & Lindenberger, 2003;

Zelinsky & Lewis, 2003), this might be caused by an underlying third variable such as neurophysiological degradation, physical changes due to closeness to death, restricted environmental challenges stimulating the use of fewer abilities, or restriction in activities.

Mean level plasticity refers to the change in the quantity or amount of a cognitive ability over time. Although one might be interested in cognitive plasticity in individual persons, mean plasticity is usually examined using average (i.e., sample values of groups of) persons. This provides an estimate of the range of plasticity within normal development by comparing average with better than average performance. In fact, from empirical findings, accelerating decline at the transition from middle to late adulthood seems to be evident for some but not all cognitive abilities. The mean stability in middle age often reported might be still indicating plasticity, that is, to the degree the level of performance is maintained despite increases and changes in work and family demands (Martin & Mroczek, in press). That is, examining mean plasticity requires longitudinal data to determine the influence of environmental factors on cognitive development, the effects of chronic stressors on cognitive performance, the interaction between changes in different cognitive domains, and cohort differences between early and late middle age in the amount of cognitive performance change.

Differential plasticity refers to the potential changes in the consistency of individual differences in cognitive abilities across time. Conceptually, differential change implies that some individuals change to a larger (or smaller) amount than others, such that the rank order of individuals is different at different time points. Thus, differential plasticity indicates actual changes within individuals. As with the other types of plasticity, however, it should be noted that changes can be in the direction of increases, decreases, or stability. A strength of examining differential change is that relative change, that is, the degree to which individuals' cognitive performance changes in similar directions over time, is considered. However, from a low stability implying pronounced differential change, we do not know whether the sample of persons—let alone an individual person—increases or decreases in their cognitive ability level. Theoretically, differential plasticity would imply models assuming that different environmental or activity factors are actively shaping changes in cognitive performance; were these influences not differentially influencing intraindividual change, then only biologically or maturation-based changes that may be assumed to be more similar between persons could explain development.

Plasticity of variability refers to the fact that using correlations, we do not know whether variances change over time. That is, across time there might be perfect differential stability and no absolute change, but variances might increase or decrease (Preece, 1982). This would still be indicative of individual differences in plasticity, although both the mean level and the rank order of individuals might be perfectly preserved across time (cf. Hertzog & Dixon, 1996). Plasticity in variability would be needed to explain differential developments between an individual at risk for further and stronger declines and an individual repeatedly profiting from gains in performance because they are starting from high levels of performance. A better understanding of the processes leading to this dissociation of development could point to events or ages in the life course when persons respond particularly sensitive to even

small improvements or declines in performance, thus setting the stage for a developmental trajectory of lifelong gains or lifelong losses in performance.

Plasticity in dimensionality of change refers to the fact that if changes in cognitive abilities are highly related, this would suggest one factor or, at least, very few factors responsible for the individual plasticity observed; this factor would explain a similarly large degree of cognitive plasticity in most individuals. This could occur in stable environmental conditions leading to very similar cognitive activations or when similar physiological processes are strongly influencing performance despite environmental variations between persons and over time.

Plasticity of intraindividual variability refers to the fact that there is now sufficient empirical evidence to establish that intraindividual variability is a substantial source of systematic performance variability between people, especially in adults (Martin & Hofer, 2004; Nesselroade, 2001). In fact, recent studies suggest that intraindividual variability may predict later cognitive difficulties in older age (e.g., Rabbitt, Osman, Moore, & Stollery, 2001; Schaie, 2000). Even within the middle adult age range, there seem to be substantial interindividual differences in intraindividual change in particular cognitive functions (Ghisletta, Nesselroade, Featherman, & Rowe, 2002; Zimprich & Martin, 2002). Willis and Schaie (1999), in their review of longitudinal data on cognition, point out that there are significant individual differences above and beyond differences depending on design (i.e., cross-sectional versus longitudinal) and length of longitudinal interval. This renders it meaningful to examine individual differences in plasticity in cognitive functioning across the life span. Once sufficient data on life span cognition are available, it can be established to which degree individual differences in plasticity might be due to (a) individual differences in usage and training, (b) individual differences in the interaction between environmental demands and age-related levels of performance, or (c) individual differences in resilience and compensatory processes that individuals apply to maintain a high level of everyday functioning (e.g., Freund & Baltes, 2002).

## Summary and Conclusion

In this chapter, we have conceptualized human development as representing the individual's lifelong capacity for adaptation. Development is thus assumed to be modifiable or plastic during all phases of the life span. Cognitive plasticity represents the individual's latent cognitive potential for adaptation and change. However, there are constraints and limits on cognitive plasticity, and these constraints and limits may vary by developmental period. A major concern in the study of cognitive plasticity, therefore, is to examine the range and limits of plasticity at various phases of the life span.

Cognitive plasticity occurs and can be studied at multiple levels—neural, behavioral, and sociocultural. Within each level, cognitive plasticity can also be studied on various time scales, ranging from momentary or short term to life span development (long term) and, finally, beyond the individual to plasticity on an evolutionary or phylogenetic time scale. In addition, at each level of cognitive plasticity, there are mechanisms and processes associated with the

nature of plasticity and with the range and limits of plasticity. Finally, there are developmental differences in plasticity (e.g., asymptote and mechanisms) across the life span. A comprehensive conceptualization or theory of cognitive plasticity would address each of these topics.

With regard to the levels at which cognitive plasticity can be studied, there is increasing evidence that the relation between levels is not solely unidirectional, from brain to behavior. Rather, there appear to be reciprocal relations between brain, experience, and behavior. Current neural structures (brain size, number of neurons, and synaptic density) do not totally determine cognitive behavior and potential. As Stern's concept of cognitive reserve suggests, cognitive potential can remain even when neural structure has been compromised. Neurogenesis can occur even in older humans and animals as a result of cognitively stimulating activities at the behavioral level. Enhanced neuronal structure may play a role in protecting against neuronal degradation. Likewise, experiences at the behavioral level shape the capacity of the brain both to reconfigure cortical modules and networks and to customize cortical modules as needed for various tasks. Efficient acquisition of new cognitive skills and behaviors in later life depends on reconfiguration and customization of cortical networks in the brain. Hence, reconfiguration and customization of cortical modules and networks represent critical mechanisms for brain plasticity.

The role of learning and experience in relation to cognitive plasticity is complex and can impact cognitive plasticity both positively and negatively. On the one hand, as discussed previously, cognitive stimulation at the behavioral level can lead to neurogenesis and desirable changes in the reconfiguration and customization of cortical modules and networks. These neural changes as a result of cognitive stimulation can occur in old age as well as earlier in the life span, although perhaps to a lesser degree in old age. On the other hand, prior learning and experience can place limits or constraints on future levels of plasticity. The role of prior learning experiences in limiting cognitive plasticity is particularly important in the later part of the life span. Research on sensitive periods suggests that prior learning and experiences may result in limitations on existing neural resources needed for the acquisition of new cognitive skills (i.e., plasticity). Additionally, prior learning and experience result in the establishment of cortical networks; these preexisting networks may make the reconfiguration and customization that is required for further plasticity more difficult and less efficient.

While there is increasing evidence that cognitive plasticity is possible during all developmental periods, constraints and limits on plasticity at each level become more evident with increasing age. At the neural level, there are constraints due to degradation of the neural structure (brain atrophy, number of neurons, and synaptic density). Likewise, there are constraints at the functional level in the brain. Flexibility in reconfiguration of cortical networks or in customization of cortical modules is reduced with advancing age. At the behavioral level, there appear to be constraints in terms of the asymptotic level of performance attainable with increasing age. Although cognitive interventions result in significant behavioral improvement, the highest level attained is lower for older adults compared to young adults. Likewise, efficiency of new skill acquisition appears to be compromised with age under conditions of "testing the limits." For example, increasing the speed at which older adults must perform or requiring

the engagement in dual tasks compromises the performance of older adults to a greater extent than those younger in the life span.

Finally with regard to plasticity at the sociocultural level, Flynn suggests that there are significant cohort differences in the range of plasticity during different historical periods. The Flynn effect suggests that the period after World War II may have been particularly supportive of plasticity with regard to fluid abilities. The cohort-sequential studies of Schaie suggest that different historical periods have been associated with significant cohort gains in crystallized versus fluid intelligence. The bioconstructionist perspective suggests that increasing cultural resources are required for cognitive plasticity with age but that utilization of these cultural resources becomes less efficient with age.

Future development of theories of plasticity will require further articulation of the dynamic interplay between different levels of cognitive plasticity (brain, behavior, and society/culture). There is increasing evidence that a reciprocal relation exists between plasticity in brain and behavior. The bioconstructionist perspective suggests further that there is also reciprocity between individual and cultural plasticity. Mechanisms and processes associated with cognitive plasticity at each level have been identified. Future theoretical development as well as empirical research is now needed to understand how these mechanisms change and adapt as a function of age and of prior learning and experience. Further consideration of the relation and interaction between short-term and long-term cognitive plasticity would facilitate an understanding of how development and adaptation occurs across the life span. Under what conditions is short- or long-term plasticity adaptive?

Finally, it is important to note that cognitive plasticity should serve to facilitate successful adaptation (i.e., development) across the life span. Plasticity, although often defined in terms asymptotic levels, faster and more efficient cortical networks or behaviors, or cognitive training effects, must ultimately involve a consideration of the individual's own developmental goals and life choices. Cognitive plasticity provides the individual with increased resources for better self-regulation in goal pursuits and in dealing with the challenges and adversities associated with development and aging. It is important that future theoretical development on cognitive plasticity include consideration of these higher-order strivings or needs of the aging individual.

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