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Mind, Brain, and Education in Socioeconomic Context

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Mind, Brain, and Education in Socioeconomic Context

Martha J. Farah

Introduction

Ten years ago, when I was just becoming interested in the relation between child development and socioeconomic status, I attended a small workshop sponsored by the McDonnell Foundation to discuss new directions in developmental cognitive neuroscience. At the time I knew virtually nothing about development *or* SES but, since the meeting was so small and informal, I decided to present some ideas on the topic of “cognitive developmental neuro-sociology” for the sake of getting feedback from the experts present. Although everyone gave me a good-natured hearing, one person took me aside afterward and offered a wealth of information, advice, and encouragement. He continued to educate me through subsequent correspondence and a visit to his lab in Toronto. That person was Robbie Case. By guiding me to relevant literatures on socioeconomic disparities and childhood development, of which I had been embarrassingly ignorant, and by encouraging me to try working in this area for which I was little prepared, he was instrumental in helping turn the vague musings of that small meeting into the program of empirical research described here.

What would a field with the inauspicious name “cognitive developmental neuro-sociology” be about? To me, it represented a new approach to the age-old problems of social stratification and the persistence of poverty. Why, in advanced societies that seem to offer opportunity for all, do some people remain poor? Why do many families remain poor across generations? These questions have occupied sociologists for as long as their field has existed, and have been answered in many ways.

Marxist approaches to the persistence of poverty emphasized purely economic factors that create and maintain social stratification (Marx, 1867). Functionalist accounts highlight the many ways in which society as a whole is served by the enduring presence of a lower class (e.g., Weber, 1923). The concept of a Culture of Poverty emphasizes causes within individuals and their subculture, rather than

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46 external societal forces, in perpetuating poverty across generations (Lewis, 1965).
47 Each account undoubtedly captures some truth about the complex and multifactorial
48 processes that confine children born of poor parents to lifelong poverty.

49 Cognitive neuroscience may offer yet another perspective on the problem by illu-
50 minating the ways in which the experience of growing up poor reduces people's
51 ability to escape poverty. Neuroscience research on the effects of early experi-
52 ence on animal brain development suggests how childhood poverty might constrain
53 human brain development. Specifically, the reduced opportunities for stimulating
54 experience and increased stress of poverty would be expected to exert a negative
55 influence on neurocognitive development. Without good neurocognitive develop-
56 ment, intellectual and educational attainments are limited, which in turn limits
57 upward socioeconomic mobility.

61 **Education, Socioeconomic Status, and Child Development**

62
63 In principle, education is an equalizer that provides all individuals in our society
64 with the opportunity to fulfill their intellectual potential and prepare for worth-
65 while employment. In practice, these benefits of education are often less available
66 to individuals of low socioeconomic status for a variety of reasons (see Arnold &
67 Doctoroff, 2003, for a review). Schools attended by low-SES students are gener-
68 ally less well funded than other schools. This results in lower quality education
69 and worse educational outcomes for students at such schools (Phillips, Voran,
70 Kisker, Howes, & Whitebook, 1994; Pianta, La Paro, Payne, Cox, & Bradley, 2002).
71 Attitudes of teachers and parents also play a role, with lower and more negative
72 expectations of lower SES students (Alexander, Entwistle, & Thompson, 1987;
73 Battin-Pearson et al., 2000; McLoyd, 1990). Finally, even before they enter school,
74 low-SES children lag behind their middle-class counterparts by most measures of
75 cognitive development (e.g., Bayley Infant Behavior Scales and IQ scores) and
76 school readiness (e.g., preliteracy skills such as letter recognition) (Brooks-Gunn
77 & Duncan, 1997). They enter the school system in need of an enriched educa-
78 tional experience, but often their lack of preparation is simply compounded by an
79 inadequate school system (Arnold & Doctoroff, 2003).

80 The research summarized in this chapter is aimed at understanding the ways in
81 which childhood poverty, including experiences prior to school entry, affect cog-
82 nitive development. The correlations between SES and performance on standard-
83 ized tests such as IQ tell us that SES must be related to brain development, as cognitive
84 ability is a function of the brain. Yet little is currently known about the relation-
85 ship between SES and brain development. Open questions include the specific
86 neurocognitive systems that correlate with SES, the impact of these neurocogni-
87 tive disparities on school readiness and school achievement, and the mechanisms
88 by which these disparities emerge. The research summarized here includes work by
89 me, my colleagues, and others, aimed at answering these open questions.

The Neurocognitive Profile of Childhood Poverty

For a cognitive neuroscience approach to be helpful in understanding cognitive development in poverty, the relations between socioeconomic status and the brain must be relatively straightforward and generalizable. The first question to be addressed is therefore: Can we generalize about the neurocognitive correlates of socioeconomic status, that is, the specific neurocognitive systems that are, and are not, correlated with SES?

Although most research on SES and child development has involved relatively broad-spectrum measures of cognition such as IQ or school achievement, there is evidence that points more specifically to associations with language development and executive function. The literature on language development is the most extensive in this regard, documenting robust SES disparities in vocabulary and phonological awareness among other linguistic abilities (see Whitehurst, 1997, for a review). SES disparities in executive functions associated with prefrontal cortex have also been noted. In the one such study, Mezzacappa (2004) tested a large group of urban 6-year-olds of varying SES on a computerized task that allows different components of attention to be assessed (the Attention Network Task, Rueda et al., 2004). He found the strongest relation with SES in what he termed “executive attentional” processes. Lipina, Martelli, Vuelta, and Colombo (2005) studied the development of working memory and inhibitory control in infancy by administering Diamond’s (1990) “A-not-B” protocol to healthy infants from poor and nonpoor families. They found a significant disparity between the two groups.

These studies tell us that language and executive function, two types of ability that reflect the operation of specific neural systems, develop differently in children depending on SES. However, these studies do not tell us whether the SES disparities in cognition are limited to these neurocognitive systems, whether other specific systems are also affected, or whether the SES disparity in neurocognitive development is global, affecting all systems. To answer this question, it is necessary to assess the development of a set of different neurocognitive systems together in the same children. This is what we have done in a series of three studies.

In an initial study, we compared the neurocognitive performance of 30 low- and 30 middle-SES African-American Philadelphia public school kindergarteners (Noble, Norman, & Farah, 2005). The children were tested on a battery of tasks adapted from the cognitive neuroscience literature, designed to assess the functioning of five key neurocognitive systems. These systems are described briefly here:

- The *Prefrontal/Executive* system enables flexible responding in situations where the appropriate response may not be the most routine or attractive one, or where it requires maintenance or updating of information concerning recent events. It is dependent on prefrontal cortex, a late-maturing brain region that is disproportionately developed in humans.

- 136 • The *Left perisylvian/Language* system is a complex, distributed system encom-
137 passing semantic, syntactic, and phonological aspects of language and dependent
138 predominantly on the temporal and frontal areas of the left hemisphere that
139 surround the Sylvian fissure.
- 140 • The *Medial temporal/Memory* system is responsible for one-trial learning, the
141 ability to retain a representation of a stimulus after a single exposure to it
142 (which contrasts with the ability to gradually strengthen a representation through
143 conditioning-like mechanisms), and is dependent on the hippocampus and related
144 structures of the medial temporal lobe.
- 145 • The *Parietal/Spatial cognition* system underlies our ability to mentally represent
146 and manipulate the spatial relations among objects and is primarily dependent
147 upon posterior parietal cortex.
- 148 • The *Occipitotemporal/Visual cognition* system is responsible for pattern recog-
149 nition and visual mental imagery, translating image format visual representations
150 into more abstract representations of object shape and identity, and reciprocally
151 translating visual memory knowledge into image format representations (mental
152 images).

153
154 Not surprisingly, in view of the literature on SES and standardized cognitive
155 tests, the middle-SES children performed better than the low-SES children on the
156 battery of tasks as a whole. Also consistent with the literature just reviewed, the
157 Left perisylvian/Language system and the Prefrontal/Executive system showed sub-
158 stantial disparities between the low- and middle-SES kindergarteners. Indeed, the
159 groups differed by over a standard deviation in their performance composite on
160 language tests, and by over two thirds of a standard deviation in the executive func-
161 tion composite. The other neurocognitive systems tested did not differ significantly
162 between low- and middle-SES children, and in fact differed significantly less than
163 the first two.

164 In a subsequent study we attempted to replicate and extend these findings in an
165 older group of children with a different set of tasks. We tested 60 middle-school
166 students, half of low and half of middle SES, matched for age, gender, and ethnic-
167 ity (Farah et al., 2006). These children completed a new set of tests designed to
168 tap the same neurocognitive systems as the previous study. In addition, instead of
169 considering “prefrontal/executive” to be a single system, we subdivided it into three
170 subsystems each with its own tests:

- 171
172 • The *Lateral prefrontal/Working memory* system enables us to hold information
173 “on line” to maintain it over an interval and manipulate it, and is primarily depen-
174 dent on the lateral surface of the prefrontal lobes. (Note that this is distinct from
175 the ability to commit information to long-term memory, which is dependent on
176 the medial temporal cortex.)
- 177 • The *Anterior cingulate/Cognitive control* system is required when we must resist
178 the most routine or easily available response in favor of a more task-appropriate
179 response and is dependent on a network of regions within prefrontal cortex
180 including the anterior cingulate gyrus.

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- 181 • The *Ventromedial prefrontal/Reward processing* system is responsible for regu-
182 lating our responses in the face of rewarding stimuli, allowing us to resist the
183 immediate pull of a attractive stimulus in order to maximize more long-term
184 gains.
185

186 A second important difference between this and the previous study concerned
187 the tests of the Medial temporal/Memory system. In both of the tasks used to assess
188 memory in the previous study, the test phase followed immediately after the initial
189 exposure to the stimuli and memory per se may not have been the limiting factor
190 in performance. The tasks that we used in the second study included a longer delay
191 between initial exposure to the stimuli to be remembered and later test.

AQ2 192 As with the younger children, sizeable and significant SES disparities were
193 observed for language and executive function. In addition, it was possible to
194 discern which aspects of executive function were most sensitive to SES. The
195 Lateral prefrontal/Working memory and Anterior cingulate/Cognitive control sub-
196 systems showed SES disparities. Finally, with a longer delay between exposure
197 and test in the memory tasks, we also found a difference in the Medial tempo-
198 ral/Memory system. SES was not associated with significant differences in the
199 Parietal/Spatial cognition system, the Occipitotemporal/Visual cognition system, or
200 the Ventromedial prefrontal/Reward processing system.

201 Finally, we assessed neurocognitive profile in 150 first graders of varying ethnic-
202 ities whose SES spanned a range from low through middle (Noble, McCandliss, &
203 Farah, 2007). As before, we used a battery of age-appropriate tasks designed to tap
204 the different neurocognitive systems. Also as before, the Left perisylvian/Language
205 system showed a highly significant relationship to SES, as did the Medial tempo-
206 ral/Memory system and the executive functions Lateral prefrontal/Working memory
207 and Anterior cingulate/Cognitive control. In addition, there was an SES gradient in
208 Parietal/Spatial cognition.

209 In sum, although the outcome of each study was different, there were also com-
210 monalities among them despite different tasks, different children, and different ages
211 of testing. The most robust neurocognitive correlates of SES appear to involve the
212 Left perisylvian/Language system, the Medial temporal/Memory system (insofar as
213 SES effects were found in both studies that tested memory with an adequate delay)
214 and the Prefrontal/Executive system, in particular its Lateral prefrontal/Working
215 memory and Anterior cingulate/Cognitive control components. Children growing
216 up in low-SES environments perform less well on tests that tax the functioning of
217 these specific systems.
218

219 Neurocognitive Development and Academic Achievement

220
221
222
223 SES disparities in executive function, memory, and language would be expected
224 to impact school success in a variety of ways, compounding the challenges faced
225 by low-SES students in school. Abundant research has documented the importance

of executive function for self-regulation and the importance of self-regulation, in turn, for school readiness and academic achievement more generally (e.g., Blair & Razza, 2007; Case, 1992; McClelland et al., 2007; Mischel, Shoda, & Rodriguez, 1989; Posner & Rothbart, 2005). The importance of memory ability for learning is obvious. Even when conceptual rather than rote learning is the goal, the ability to retain the particulars of facts or illustrations supports students' more abstract understanding. Finally, language is not only a subject of study in school but the medium through which most knowledge and skills are taught.

One pathway through which language ability affects school success is through its influence on reading ability. Kim Noble addressed the roles of language ability and SES on schoolchildren's reading ability in her dissertation research. She pointed out that, of the many aspects of language predictive of early reading, the most powerful predictor is "phonological awareness" (Bradley & Bryant, 1983; Wagner & Torgesen, 1987). This refers to our ability to attend to the sound structure of the language, as when we judge whether or not two words rhyme. Given earlier findings that phonological awareness is correlated with SES (Noble et al., 2005; Noble et al., 2007; Wallach, Wallach, Dozier, & Kaplan, 1977), we were led to ask: Does the SES gradient in phonological awareness account for the SES gradient in reading ability? By assessing SES, phonological awareness, and reading ability in the sample of first graders from our earlier study, we found that SES was correlated with reading ability above and beyond its correlation with phonological awareness.

Furthermore, SES and phonological awareness were not independent in their influences on early reading ability. At lower levels of SES, reading ability was well predicted by phonological awareness, whereas the relationship was weaker at higher levels of SES. Put another way, at higher levels of phonological awareness, all children mastered reading, whereas children with lower levels of phonological awareness were better readers if they came from higher levels of SES. The benefits of a higher SES background appear to buffer children against the effects of low phonological awareness (Noble, Farah, & McCandliss, 2006). A subsequent imaging study clarified the nature of this buffering effect. It might have reflected better functioning of the visual word decoding regions of the brain or other compensatory strategies used with a given level of visual word decoding. Our fMRI evidence showed that the visual word decoding area itself (in the left fusiform gyrus) was more active for higher SES children at a given level of phonological awareness, suggesting that the enriched literacy environment of higher SES homes affects the neural bases of visual word decoding per se (Noble et al., 2006).

Mechanism: Disentangling Causes and Effects

Why do different aspects of brain function come to be associated with SES? Do the associations discussed so far reflect the effects of SES on brain development, or the opposite direction of causality? Perhaps families with higher innate language,

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271 executive, and memory abilities tend to acquire and maintain a higher SES. Given
272 that the direction of causality is an empirical issue, what data bear on the issue?

273 The methods of behavioral genetics research can, in principle, tell us about the
274 direction of causality in the association between SES and the development of specific
275 neurocognitive functions. However, these methods have yet to be applied to that
276 question. They have been applied to a related question, namely the heritability of IQ
277 and SES. Cross-fostering studies of within- and between-SES adoption suggest that
278 roughly half the IQ disparity in children is experiential (Capron & Duyme, 1989;
279 Schiff & Lewontin, 1986). If anything, these studies are likely to err in the direction
280 of underestimating the influence of environment because the effects of prenatal and
281 early postnatal environment are included in the estimates of genetic influences in
282 adoption studies. Additional evidence comes from studies of when, in a child's life,
283 poverty was experienced. Within a given family that experiences a period of poverty,
284 the effects are greater on siblings who were young during that period (Duncan,
285 Brooks-Gunn, & Klebanov, 1994), an effect that cannot be explained by genetics.
286 In sum, multiple sources of evidence indicate that SES does indeed have an effect
287 on cognitive development, although its role in the specific types of neurocognitive
288 system development investigated here is not yet known.

289 Many different aspects of childhood SES could affect neurocognitive develop-
290 ment. Some do so by their direct effects on the body and some by less direct
291 psychological mechanisms. Three somatic factors have been identified as significant
292 risk factors for low cognitive achievement by the Center for Children and Poverty
293 (1997): inadequate nutrition, lead exposure, and substance abuse (particularly
294 prenatal exposure).

295 The role of nutrition in SES disparities in brain development has been diffi-
296 cult to resolve because nutritional status is so strongly correlated with a host of
297 other family and environmental variables likely to impact neurocognitive develop-
298 ment, including all of the potential mechanisms of causation to be reviewed here.
299 Although nutritional supplementation programs could in principle be used as an
300 "experimental manipulation" of nutritional status alone, in practice these programs
301 are often coupled with other, non-nutritional forms of enrichment or affect children's
302 lives in non-nutritional ways which perpetuate the confound (e.g., children given
303 school breakfast are less often late or absent). In addition, poor nutrition may syn-
304 ergize with other forms of childhood deprivation in impairing brain development.
305 Iron-deficiency anemia is known to afflict about one quarter of low-income children
306 in the United States (CHPNP 1998) and is known to impair brain development when
307 severe.

308 Lead is a neurotoxin to which children of lower SES are more likely exposed.
309 Even at relatively low levels of lead in the blood, under $10 \mu\text{g/dL}$, there is a sys-
310 tematic relationship between lead level and IQ (Surkan et al., 2007). As with
311 nutrition, the effect of lead synergizes with other environmental factors and is more
312 pronounced in low-SES children (Bellinger, Leviton, Wateraux, Needleman, &
313 Rabinowitz, 1987).

314 Prenatal substance exposure is a third factor that affects children of all SES levels
315 but is disproportionately experienced by the poor. Maternal use of alcohol, tobacco,

316 marijuana, and other drugs of abuse have been associated with adverse cognitive
317 outcomes in children (Chasnoff et al., 1998). Although the highly publicized phe-
318 nomenon of “crack babies” might lead one to view prenatal cocaine exposure as a
319 major contributor to the SES disparities noted here, there is little evidence that it
320 plays a role. In her 2001 review of the literature on this topic, Frank offered the
321 following tentative conclusion, pending new evidence: “there is no convincing evi-
322 dence that prenatal cocaine exposure is associated with developmental toxic effects
323 that are different in severity, scope, or kind from the sequelae of multiple other
324 risk factors. Many findings once thought to be specific effects of in utero cocaine
325 exposure are correlated with other factors, including prenatal exposure to tobacco,
326 marijuana, or alcohol and the quality of the child’s environment” (p. 1613). Indeed,
327 we recently compared the performance of cocaine exposed and nonexposed children
328 on the task battery used by Farah et al. (2006) and found no differences (Hurt et al.,
329 submitted).

330 The set of potentially causative somatic factors just reviewed is far from com-
331 plete. There are SES gradients in a wide variety of physical health measures, many
332 of which could affect children’s neurocognitive development through a variety of
AQ3 333 different mechanisms (Adler et al., 1994). In addition, the typical psychological
334 experiences of childhood differ sharply between poor and nonpoor families, and
335 these differences also contribute to the differing neurocognitive outcomes for the
336 children of these families.

339 **Psychological Influences on Neurocognitive Development** 340 **in Poverty** 341

342
343 As with potential physical causes, the set of potential psychological causes for
344 the SES gap in cognitive achievement is large, and the causes are likely to
345 exert their effects synergistically. One difference between low- and middle-SES
346 families that seems predictable, even in the absence of any other information, is that
347 low-SES children are likely to receive less cognitive stimulation than middle-SES
348 children. Their economic status alone predicts that they will have fewer toys and
349 books and less exposure to zoos, museums, and other cultural institutions because
350 of the expense of such items and activities. This is indeed the case (Bradley, Corwyn,
351 McAdoo, & Garcia Coll, 2001) and has been identified as a mediator between SES
352 and measures of cognitive achievement (Bradley & Corwyn, 1999; Brooks-Gunn &
353 Duncan, 1997; McLoyd, 1998).

354 Such a mediating role is consistent with the results of neuroscience research
355 with animals. Starting many decades ago, researchers began to observe the pow-
356 erful effects of environmental stimulation on brain development. Animals reared in
357 barren laboratory cages showed less well-developed brains by a number of different
358 anatomical and physiological measures, compared with those reared in more com-
359 plex environments with opportunities to climb, burrow, and socialize (van Praag,
360 Kempermann, & Gage, 2000; Rosenzweig, 2003).

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361 Other types of cognitive stimulation are also less common in low-SES homes, for
362 example parental speech designed to engage the child in conversation (Hoff, 2003).
363 The average number of hours of one-on-one picture book reading experienced by
364 children prior to kindergarten entry has been estimated at 25 for low-SES children
365 and between 1000 and 1700 for middle-SES children (Adams, 1990). In addition to
366 material limitations, differing parental expectations and concerns also contribute to
367 differences in the amount of cognitive stimulation experienced by low- and middle-
368 SES children (Lareau, 2003).

369 Another major difference in the lives of low- and middle-SES individuals con-
370 cerns levels of stress, and this has been related to differences in child development
371 (Evans & English, 2002). The lives of low-SES individuals tend to be more stress-
372 ful for a variety of reasons, some of which are obvious: concern about providing
373 for basic family needs, dangerous neighborhoods, and little control over one's work
374 life. Again, research bears out this intuition: Turner and Avison (2003) confirmed
375 that lower SES is associated with more stressful life events by a number of different
376 measures. The same appears to be true for children as well as adults, and is apparent
377 in salivary levels of the stress hormone cortisol (Lupien et al., 2001).

378 Why is stress an important consideration for neurocognitive development?
379 Psychological stress causes the secretion of stress hormones, which affect the brain
380 in numerous ways (Gunnar & Quevedo, 2007; McEwen, 2000). The immature brain
381 is particularly sensitive to these effects. In basic research studies of rat brain devel-
382 opment, rat pups are subjected to the severe stress of prolonged separation from
383 the mother and stress hormone levels predictably climb. However, the effect of a
384 brief handling (minutes per day), which also separates the animal from its mother,
385 appears beneficial. Both prolonged maternal separation and brief handling affect
386 later-life stress regulation ability and memory ability as a result of their impact
387 on hippocampal development. The salutary effect of brief separations appears to
388 result from the intensified nurturing behavior that follows the separation. The more a
389 mother rat licks her pup following a brief stressor, the better regulated the pup's later
390 response to stressors and the better its learning ability (Liu, Diorio, Day, Francis, &
391 Meaney, 2000). This suggests that the high stress of poverty will take a toll on
392 children's brain development, especially the development of the Medial tempo-
393 ral/Memory system, but that differences in parenting may strongly modulate those
394 effects.

395 Our current research is attempting to make use of the description of the SES
396 disparities in specific neurocognitive systems to test hypotheses about causal
397 pathways. Drawing on the earlier findings indicating robust SES differences in
398 Perisylvian/Language and Medial temporal/Memory systems, we are now testing
399 hypotheses concerning the determinants of individual differences in the develop-
400 ment of these systems in children of low SES (Farah et al., in press).

401 The participants in this research were 110 low-SES middle-school students from
402 a cohort of children enrolled at birth in a study of the effects of prenatal cocaine
403 exposure (see Hurt et al., 1995). Approximately half of the children have been
404 exposed to cocaine prenatally and half have not. Maternal use of cocaine as well
405 as amphetamines, opiates, barbiturates, benzodiazepines, marijuana, alcohol, and

406 tobacco are ascertained by interview and medical record review at time of birth and,
407 for all but the last three, maternal and infant urine specimens.

408 As part of the ongoing study of these children, a research assistant visited the
409 home of each child at ages 4 and 8 and administered the HOME (Home Observation
410 and Measurement of Environment, Caldwell & Bradley, 1984). The HOME includes
411 an interview with the mother about family life and observations of the interactions
412 between mother and child. The HOME has a number of different subscales relevant
413 to different aspects of the child's experience. We combined a number of different
414 subscales indicative of the amount of cognitive stimulation provided to the child to
415 make a composite measure of Environmental Stimulation, and a number of different
416 subscales indicative of the amount of social/emotional nurturance provided to the
417 child to make a composite measure of Parental Nurturance. The subscales used for
418 each composite, along with representative items, were as follows:

- 419
- 420 ● The *Environmental Stimulation composite* for 4-year-olds was composed of
421 *Learning stimulation* ("child has toys which teach color," "at least 10 books are
422 visible in the apartment"), *language stimulation* ("child has toys that help teach
423 the names of animals," "mother uses correct grammar and pronunciation"), *aca-*
424 *ademic stimulation* ("child is encouraged to learn colors," "child is encouraged
425 to learn to read a few words"), *modeling* ("some delay of food gratification is
426 expected," "parent introduces visitor to child"), and *variety of experience* ("child
427 has real or toy musical instrument," "child's art work is displayed some place in
428 house"). For 8-year-olds, the subscales used for the cognitive stimulation com-
429 posite were: *Growth fostering materials and experiences* ("child has free access
430 to at least ten appropriate books," "house has at least two pictures of other type of
431 art work on the walls"), *provision for active stimulation* ("family has a television,
432 and it is used judiciously, not left on continuously," "family member has taken
433 child, or arranged for child to go to a scientific, historical, or art museum within
434 the past year"), *family participation in developmentally stimulating experiences*
435 ("Family visits or receives visits from relatives or friends at least once every other
436 week," "family member has taken child, or arranged for child to go, on a trip of
437 more than 50 miles from his home").
 - 438 ● The *Parental Nurturance composite* for 4-year-olds: was composed of: *Warmth*
439 *and affection* ("parent holds child close 10–15 minutes per day," "parent con-
440 verses with child at least twice during visit") and *acceptance* ("parent does not
441 scold or derogate child more than once," "parent neither slaps nor spansks child
442 during visit"). For 8-year-olds, the subscales used were *Emotional and verbal*
443 *responsivity* ("Child has been praised at least twice during past week for doing
444 something," "parent responds to child's questions during interview"), *encourage-*
445 *ment of maturity* ("family requires child to carry out certain self-care routines,"
446 "parents set limits for child and generally enforce them"), *emotional climate*
447 ("parent has not lost temper with child more than once during previous week,"
448 "parent uses some term of endearment or some diminutive for child's name when
449 talking about child at least twice during visit") and *paternal involvement* ("Father
450 [or father substitute] regularly engages in outdoor recreation with child," "Child

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451 eats at least one meal per day, on most days, with mother and father [or mother
452 and father figure]”).

453
454 Two other variables with the potential to account for differences in neurocogni-
455 tive development included in our analyses were maternal intelligence and prenatal
456 substance exposure. The former was measured by the Weschler Adult Intelligence
457 Scale–Revised (WAIS–R). Maternal IQ could influence child neurocognitive out-
458 come by genetic mechanisms or by its effect on the environment and experiences
459 provided by the mother for the child. Prenatal substance exposure was coded for
460 analysis on an integer scale of 0–4, with one point for each of the following sub-
461 stances: tobacco, alcohol, marijuana, and cocaine. Use of other substances was an
462 exclusionary criterion.

463 We used statistical regression to examine the relations between the neurocogni-
464 tive outcome measures and the predictor variables Environmental Stimulation,
465 Parental Nurturance, maternal IQ, and polysubstance use, as well as the child’s
466 gender and age at the time of neurocognitive testing. Our results indicate that the
467 development of different neurocognitive systems is affected by different variables.

468 Children’s performance on the tests of Left perisylvian/Language was predicted
469 by average Environmental Stimulation. This was the sole factor identified as pre-
470 dicting language ability by forward stepwise regression, and one of two factors
471 identified by backward stepwise regression, along with the child’s age. In contrast,
472 performance on tests of Medial temporal/Memory ability was predicted by average
473 Parental Nurturance. This was the sole factor identified as predicting memory abil-
474 ity by forward stepwise regression and one of three factors identified by backward
475 stepwise regression, along with the child’s age and prenatal substance exposure.
476 The relation between memory and Parental Experience is consistent with the animal
477 research cited earlier (Liu et al., 2000).

478 Our analyses did not reveal any systematic relation of the predictor variables con-
479 sidered here to Lateral prefrontal/Working memory or Anterior cingulate/Cognitive
480 control function.

481 The relation between life experience and brain development for human beings is
482 undoubtedly more complex than for animals, but we can nevertheless be guided by
483 the animal research literature in formulating hypotheses to test. So far, the use of this
484 strategy has shown that different aspects of life experience, cognitive stimulation,
485 and parental buffering of stress act on brain development by different pathways and
486 affect the different neurocognitive systems to different degrees.

487 488 489 **Conclusions**

492 Educators are increasingly incorporating the ideas and findings of neuroscience into
493 their work, a trend that Robbie Case both foresaw and helped to bring about. Our
494 growing understanding of normal brain development and atypical brain develop-
495 ment is forming the basis for new and more effective educational practice. With

496 regard to normal brain development, cognitive neuroscientists have only recently
 497 shifted from the study of commonalities among brains to the study of individual
 498 differences in brain function. Educators, who must teach students of varying abil-
 499 ity, motivation, and cognitive style, will presumably not wait as long to apply the
 500 cognitive neuroscience of individual differences in their work.

501 The findings summarized in this chapter concern a major cause of individual
 502 differences in school readiness and academic performance, namely SES. The dif-
 503 ferent kinds of childhood experience that students of lower and higher SES bring
 504 into the classroom affects what they learn there. Reciprocally, the different kinds
 505 of schools attended by children of lower and higher SES also affect the potential
 506 for learning. The neural mechanisms involved in these processes are important sub-
 507 jects for future research in neuroscience and education. Of course, it does not take
 508 a proverbial rocket scientist or, for that matter, a neuroscientist to realize that chil-
 509 dren should have access to stimulating experiences, be protected from high levels
 510 of stress, and go to good schools. Nevertheless, a better understanding of the ways
 511 in which childhood experience and classroom instruction shape brain function will
 512 suggest new ways of preventing and remediating some of the disadvantages suffered
 513 by poor children.

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Chapter 11

Q. No.	Query
AQ1	Please check if edits to the sentence beginning “By guiding me ...” convey the intended meaning.
AQ2	Please check if ‘test’ should be changed to ‘tested’ in the sentence beginning “The tasks that ...” to convey the appropriate meaning.
AQ3	“Adler et al., 1997” has been changed to “Adler et al., 1994” as per the reference list. Please Check.
AQ4	“Hurt et al., 1995” is not given in the list. Please provide reference entry or delete citation.
AQ5	Please provide Volume ID for this reference.
AQ6	Please provide Volume ID, Pages for this reference.
AQ7	Please update.
AQ8	“Frank, Augustyn, Knight, Pell, Zuckerman, 2001” reference is not cited in the text. Please provide citation or delete from list.
AQ9	Please update.
AQ10	“Kass, 2003” reference is not cited in the text. Please provide citation or delete from list.
AQ11	Please provide remaining authors.
