

4 Child Poverty and Brain Development

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What's a nice cognitive neuroscientist doing in a field like child poverty? Why would anybody work with ill-defined concepts such as socioeconomic status (SES) when they could be manipulating binocular cues to depth perception with precision? Allow me to explain, by way of presenting my current work and how I came to it.

The first decades of my career were spent working to understand the neural bases of vision and visual cognition, using behavioral research methods with normal participants and brain-damaged patients, as well as by using event-related potentials (ERPs) and functional magnetic resonance imaging (fMRI) to study normal brain function more directly. The 1980s and 1990s were rewarding times to be involved in such research, as neuroscience first grappled with fundamental questions about how humans perceive the world.

Being a cognitive neuroscientist in the early days of the field satisfied many of my intellectual needs and wants. I could engage with age-old questions of mind and brain in a productive new way, asking how we come to know the visual world and how our brains implement or enable our minds. Of course, the deepest philosophical readings of these questions will never be settled with experiments, but to me it was nevertheless exciting to work on various empirical issues concerning mind–brain relations.

Perverse as it may sound, I also enjoyed the conceptual messiness of early cognitive neuroscience. In a mature science, there is a theoretical framework that guides us to the next questions to ask and provides models of the kinds of explanations that could count as answers. There is also a stable of methods recognized as able to deliver relevant evidence. With a framework, questions, and methods in place, the path of scientific progress is relatively clear. But, at the risk of sounding flippant, where's the fun in that? The cognitive neuroscience of the 1980s offered few of the conveniences of a mature science, but plenty of messy intellectual fun. Coming up with questions, deciding what kinds of data were relevant to answering them, parsing the array of possible answers, and examining the

theoretical assumptions that underlay these decisions – these were necessary everyday thought processes for us.

But, for all the intellectual joy of grappling with issues of mind and brain in a developing science, I did feel that one thing was missing from my work life: social relevance. So, as the field of cognitive neuroscience matured, I began to wonder whether it might now be applicable to real-world problems. I had a particular real-world problem in mind: childhood SES and its association with cognitive development and life chances.

Thus began my current research program. It was born shortly after my daughter, the connection between the two being my daughter's babysitters. The women I hired were of low SES status, having grown up on welfare. They were themselves receiving state assistance, and worked part-time as cleaners and babysitters to better support their own children. I got to know them and their families well, and became very fond of them.

I was also shocked by what I learned of their lives. One shock was the incredibly high level of stress and the frequent, uncontrollable misfortunes in their lives. I have middle-class friends who have suffered tragedies, and of course people from any social class can be their own worst enemies and create ongoing problems for themselves. But what I saw in my small foray into the world of low SES was different – there was an unrelenting cascade of problems, many related to living in bad neighborhoods, sending their children to bad schools, and having no financial cushion, often compounded by ignorance about health; fear or mistrust of doctors, teachers, and police; and a hopelessness (not hard to understand) that hampered their efforts to avoid future problems.

Another shock came from getting to know their children. Their daughters and sons and nieces and nephews began life with the same evident promise as my daughter and her friends. Yet, as the years went on, I saw their paths diverge. This led me to do some reading. From the psychology and epidemiology literatures I learned that childhood SES predicts many important life-long traits, including mental health, cognitive ability, and academic achievement. As these are all related to brain function, and I was a cognitive neuroscientist, it gave me a handle to try to understand the long-term effects of poverty on children: I could study how the brain develops in poverty.

In early work, with then graduate student Kim Noble and colleague Hallam Hurt, we investigated the neurocognitive profile of poverty in children. That is, we asked: Does poverty affect all cognitive systems of the brain evenly, across the board, or do certain systems bear the brunt of poverty while others are relatively spared? The answer to this question would helpfully constrain the kinds of causal, mechanistic explanations

Childhood
SES -->
cognitive
development,
life
chances???

Low SES =
high level of
stress,
frequent
uncontrollable
misfortunes --
living in bad
neighborhoods
, sending kids
to bad
schools,
having no
financial
cushion,
ignorance
about health,
fear/mistrust of
doctors,
teachers, &
police,
hopelessness
hampering
efforts to avoid
future
problems

Childhood
SES (= high
stress level) --
> life-long
traits: mental
health,
cognitive
ability,
academic
achievement --
all related to
BRAIN
FUNCTION

we might look for, as well as the kinds of **targeted interventions** we might consider. The answer we found, across several studies of children from kindergarten age to adolescence, was that **poverty has distinctly uneven effects on neurocognitive function**. The **executive functions of prefrontal cortex, language functions of left perisylvian cortex, and declarative memory processes of the medial temporal lobe** all tend to show pronounced disparities between poor children and middle-SES children, controlling for potential confounders such as race, ethnicity, and birth weight.

In connection with the subject of **confounding variables, or “confounders,”** let me mention that this new research direction certainly put me back in the middle of **conceptual messiness**. The word “confounder” seems to label a clear category, distinct from the variable of interest. But when the variable of interest is **SES**, which we know is associated with all kinds of differences, from nutrition to child-rearing practices, how do we decide which of these associated factors is a confounding variable (so we should try to measure effects of SES above and beyond effects of nutrition and child-rearing practices, for example) and which is part and parcel of SES, as an influence of child development? My belief is that there are no blanket right answers to this question, and that any of a number of reasonable decisions will be defensible, so long as one is clear about the decision in the reporting of the results. By and large, we have considered race and ethnicity to be confounders, but have viewed nutrition and child-rearing practices as part and parcel of SES for both practical (difficult to measure) and theoretical (these are more consistently associated with SES than are race and ethnicity) reasons. But the effects of these factors on development, in the context of SES and more generally, are of great interest and importance.

Having established at least a preliminary determination of the neurocognitive profile of poverty, we could begin to inquire into its antecedent causes and its structural brain correlates. To illustrate this process I will recount one strand of research emanating from the early finding that most surprised us: **the relation of learning and memory to SES**. Initially, I expected **declarative learning** (learning *that* something happened, as opposed to the kind of how-to learning that underlies skill formation, for example) to be unaffected by SES. I had no rationale for this expectation except that, as a cognitive ability, acquiring new declarative memories seemed too “nuts and bolts” to be subject to the complex influences of SES. But we soon learned that this was not true, and that memory testing and imaging of **the medial temporal structures required for declarative memory** showed **SES gradients, with better performance and larger regional brain volumes in higher SES children**. Why would this be?

Low-SES Children --> poorer executive functions of the prefrontal cortex (PFC), poorer language functions of the left perisylvian cortex, poorer declarative memory processes of the medial temporal lobe

SES =
Nutrition,
Child-Rearing
Practices

To study:
CAUSAL
FACTORS
(SES?) -->
neuro-
cognitive
profile,
structural
brain
correlates

SES -->?
Learning &
Memory
(Declarative
Learning)

High SES Children --> better performance in declarative learning memory tests & larger regional brain volumes in the medial temporal structures (associated with declarative learning)

Research with animals provided a promising explanation, which then found converging support from research with human children. Starting with pioneering research in rodents, it has been shown that early life stress adversely affects the development of the hippocampus, a key structure for learning and memory, located within the medial temporal lobe. It has also been found that certain kinds of early maternal nurturance can buffer the rat pups' hippocampi from these effects. Stress, as noted at the outset of this essay, is much more common in low-SES families. And maternal availability and related behaviors are themselves affected by stress and by other features of SES. In sum, we had a promising hypothesis based on experimental animal research that, on the face of things, seemed extendable to human childhood and SES.

Our own early work then confirmed that, in human children, early life parental nurturance predicted declarative learning ability in later childhood, and also predicted differences in hippocampal volume. Several excellent studies by other research groups, with larger samples of children, showed SES effects on hippocampal volume. One study even tied things up with a beautiful bow, showing that SES predicts hippocampal volume and that this effect is entirely accounted for by differences in stress levels and maternal behavior, just as the original rat studies would have predicted!

This emerging area of research remains a kind of “basic science” – that is, science aimed at understanding what, how, and why things are the way they are. It would be premature to dictate child policy solely on the basis of a dozen good studies of SES and medial temporal memory processes. But already it buttresses existing arguments about the importance of reducing the stress experienced by young children and parents of low SES. Considering that the earliest studies on the subject were published just ten years ago, and most of the research has been published in the last five years, I think we can look forward to substantive help from cognitive neuroscience in improving the life chances of poor children.

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In rodents:
Early Life
Stress + Early
Maternal
Nurturance
(Maternal
Availability &
Related
Behavior) -->
no
compromise in
hippocampus
development

In rodents:
Early Life
Stress (e.g.,
shock,
punisher,
unpleasant
stimulus) -->
under-
developed
hippocampus
(key structure
for learning &
memory)

In human
children:
Early Life
Paternal
Nurturance -->
Declarative
Learning
Ability in Later
Childhood &
increase in
hippocampal
volume