

## Executive Function

One overarching question this week is: What are the origins of individual differences in executive function?

The behavioral data can take us pretty far on this issue. Firstly, a wide range of behavioral data has shown that there is developmental continuity in EF skill level: Individual differences in EF tasks and related abilities in infancy and early childhood predict EF abilities later in childhood as well as in adulthood (e.g. Friedman et al). These individual differences themselves are largely genetic (e.g. Friedman et al., 2008; Rothbard & Rueda, 2005), and the longitudinal associations between infant and adult EF performance appears to be in large part explained by genetic factors -- as shown by the MZ vs DZ twin data (and rather opaque structural equation modeling) in Friedman et al. However, experience and training also plays an important role in EF, especially when strong interventions are provided: Children's EF abilities improve with training (particularly those who start out with low EF performance), such as Adele Diamond's *Tools of the Mind* program or similar activities requiring EF engagement in Montessori schools. Thus, EF abilities are not fixed at birth or even in infancy.

Overall, behavioral data can provide the "bones" of an answer: In populations with typical Western experiences, much of the variance in individual performance is attributable to genetics, and some is due to (shared or not-shared) experiences. When individuals' experiences include consciously-designed, 'high-dosage' intervention/training programs, this experience can result in substantial increases in EF skill.

Genetics can begin to put some meat on these explanatory bones, by identifying genes that differ between individuals with low and high EF, and exploring their effects on the molecular level. The work we read cited a few particular genes that are associated with EF abilities-- for instance, polymorphisms related to the dopamine system (DAT1, COMT val/ met, mentioned by Friedman et al); and SNPs (single nucleotide polymorphisms) related to glucocorticoid and catecholamine activity in the prefrontal cortex (mentioned by Blair). Even more tantalizing are recent findings from epigenetics -- a field that has shown us that DNA expression is itself affected by early experience. Our experiences turn genes on and off, breaking the traditional model in that experience appears to affect our (expressed) genotype, not just our phenotype. In the case of EF, the effects of early stress on subsequent EF abilities are produced through epigenetic mechanisms-- changing activity levels of gene expression that result in changes in glucocorticoid receptor density in the hippocampus (see below).

This epigenetic mechanism brings us to the crucial role of neuroscience data in providing a satisfying explanation for EF individual differences. Ultimately, the effects of genetics and experience on behavior are mediated through the brain: Our genes plus our experience generate our individual neural structures, the activities of which generate our behavior. Thus, while behavioral data provides the 'bones' of a satisfying explanation for individual differences -- by specifying the relative importance of genetics and what experience can change -- neuroscience is needed, in addition to genetic and behavioral data, to flesh out the answer into a satisfying explanation of the phenomenon.

In the case of EF, one particular explanation in our reading bridged all of these levels of analysis (in the Blair paper), and thus seemed particularly satisfying to me. Although I'm not sure I can do the neural explanation justice here in one paragraph, here's the basic story.

The behavioral phenomenon is that individuals raised in stressful environments in early life typically have lower EF abilities later in life. The neural data give one major part of a

satisfying explanation for this phenomenon: In early life, high stress results in high levels of cortisol (in humans) or corticosteroids (in rodents). This influences the development of specific brain structures important for EF, by programming the stress response systems and the PFC to engage in reactive/reflexive processes rather than reflective self-regulation. We even can explain how this works: At very high levels of stress hormones, a specific type of receptor primarily located in the PFC becomes saturated, and another type of receptor, which is mostly present in sub-cortical and posterior regions, becomes active, promoting a reflexive instead of reflective response. Genetics and epigenetics adds an additional piece to this explanation, by showing that experience not only affects hormonal/neural systems directly, but also affects these systems by changing gene expression: typical development of the neural stress-response system is mediated through epigenetic effects on genes coding for glucocorticoid receptor density.

Thus, adding the neural level of analysis allows us to flesh out our answer regarding the origins of individual differences in EF, and provide a much more satisfying explanation for how genetic predispositions and environmental effects interact to produce different levels of EF ability across individuals.

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