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The Development of Behaviors: Constancy and Plasticity

I. Neural Plasticity

Experience can control the development of the brain

Until the 1960s, it had generally been assumed that once the brain had formed, new neurons were not generated and new synapses did not form. After childhood, the brain had stopped developing (see Pascual-Leone et al. 2005). However, with the discovery of adult neural stem cells and the discoveries that new synapses can be induced by experiences such as learning, this idea of neural stasis has been replaced by the hypothesis of neural plasticity*—that learning, memory, and even healing can occur through the continued development of the brain.

**Sometimes called neuronal plasticity or just neuroplasticity.*

In the Thirteenth Edition of *Developmental Biology*, there are numerous examples demonstrating neural plasticity:

- Neural stem cells form new neurons in adult vertebrates, and these make synapses with the neurons already in place.
- The human brain continues to grow at the fetal rate for around two years after birth; and even in adolescence, remodeling is obvious.
- Experiencing maternal care can influence brain function through the glucocorticoid and estrogen receptor pathways.
- Sexual experience can regulate neuronal growth in rats.
- Learning causes changes in the synapses of mammalian brains.
- The mammalian visual system is regulated by light-induced experiences.

Just as the mammalian visual system is regulated by light-induced experiences, so the auditory system is conditioned by hearing. Congenital hearing impairment affects auditory nervous system development, and the implantation of sensory prostheses (such as cochlear implants) that activate the auditory system have prevented these hearing deficits and allow the normal maturation of the auditory system. There is a developmental window for this plasticity, such that intervention needs to occur within the first 2–4 years of life (Kral and O'Donoghue 2010; Kral and Sharma 2012).

The book did not discuss some of the medical aspects of neural plasticity that may enable the nervous system to heal itself. Indeed, there is a range of new therapies that attempt to direct the plastic nervous system to form certain neuronal pathways that may have been damaged by stroke (Nojima et al. 2012).

Thus, the genes we receive at fertilization are not telling us "who" we are. Rather, we are a complex intermixing of inheritance and experience. The plasticity of the nervous system, wrote Barton Childs (2003) enables us to be individuals and "allows us to escape the tyranny of our genes."

Maladaptive aspects of neural plasticity

However, new evidence shows that developmental plasticity may also help confine us.

Phantom limb syndrome. Individuals who have a limb amputated often feel pain in the *absent* appendage. This "phantom limb" phenomenon occurs in a majority of amputees, and it appears to be caused by a reorganization of the human cerebral cortex following the amputation. The cortical connections of the neurons from the removed limbs are thought to have become re-connected to neurons in the area around them. This causes the neural activity within the surrounding area of the cortex to be misinterpreted as coming from the amputated limb. Experiments on individuals with phantom limbs have shown that the body image we have is generated by the brain (Mosely and Brugger 2009).

Chronic pain syndromes. Related to phantom limb syndrome is chronic pain at sites which had been injured but which are now healthy. During the period of tissue damage, painful stimuli and inflammation can cause an elevation of input from the peripheral neurons to the central nervous system. Prolonged nociception (the input of signals indicating an above-threshold experience that becomes interpreted as pain) from the periphery will then induce neuroplastic responses at the cortical level (in the brain). This alters the organization of the cortical neurons associated with the painful site. Individuals experiencing complex regional pain sydrome, carpal tunnel syndrome, and chronic low back pain often have an altered "map" of the connections between the periphery and brain, as well as a global reduction in the grey matter of the prefrontal cortex and right thalamus (Maihöfer et al. 2003; Apkarian et al. 2004; Seifert and Maihöfner 2011).

Maladaptive predictive adaptive responses. The textbook mentions "predictive adaptive responses" as a type of developmental plasticity wherein environmental conditions channel the organism into a particular phenotype. Mammalian fetuses experiencing protein depravation in utero, for instance, are seen to establish a metabolism that expects a nutient-deficient environment. If the environment is stocked with nutrients, however, this prediction is wrong, and the mismatch causes the adult to make more fat cells than otherwise expected. The mechanism of this "prediction" appears to be the methylation of particular genes such that the maternal intake of food regulates the expression of certain genes encoding those liver enzymes crucial to how food will be used. These patterns may last the rest of the organism's life.

It is possible that the brain may make similar predictive adaptive responses early in its development. It is also possible that stress (which is something *perceived* by the brain and which then influences hormone and cytokine production) induces DNA methylation differences and that these DNA alterations may be responsible for the phenotypes of these people later in life, even though they now live in relatively unstressful situations. Michael Kobor's laboratory, for instance, is trying to explain the observation that the age of onset of certain illnesses (including cardiovascular disease and susceptibility to infections) generally correlates negatively with socioeconomic class during the first years of life, even if the person has a high socioeconomic status as an adult (Miller et al. 2009.) In other words, low socioeconomic status during the first years of life gives one a statistically higher chance of having certain illnesses relatively early in life. They find that early-life low socioeconomic status leaves a "biological residue" of decreased glucorticoid signaling and increased proinflammatory signaling even when the person's perceived levels of stress as an adult are low and one's socioeconomic status is high. (Interestingly, maternal "warmth" during childhood may isolate one against these negative effects [Chen et al. 2011].)

Also, adolescents with high stresses in childhood showed slightly different DNA methylation patterns than their peers without such stresses (Essex et al. 2013). "Stress" is a difficult thing to measure, as it is something "perceived" and interpreted. But this research points to the efficacy of those programs which attempt to help the health of adults by interventions during infancy.

II. Constancy

We know very little about the constancy of behaviors. Even *C. elegans*, with its simple nervous system (302 neurons) and relatively simple environment (an agar plate full of *E. coli*), has experience-mediated changes during neural development (Gilbert and Jorgensen 1998; Bozorgmehr et al. 2013). As seen above, the environment can play a large role in human behavior, making correlations between specific genes and specific behaviors weak. It is also problematic to identify the genes that make behaviors solely by mutations. It may take a thousand steps to make something, but only one bad step is needed to break it. Specific behaviors may be the output of entire networks, and the genes of these networks produce proteins that interact in certain ways to make certain behaviors. In mammals, probably the best examples of genetic influences on behaviors are found in dog breeds where specific behaviors have been selected by breeders (Spady and Ostrander 2008; Hall and Wynne 2012). These behaviors may be the outcome of several genes being selected at the same time.

Behavior is the "last phenotype," the messiest and most environmentally influenced. Embryologist Charles Otis Whitman, the director of the Wood Hole Marine Biology Laboratories, was one of the founders of ethology, and initiated attempts to look at the developmental neurobiology of behavior. Today, this is one of the greatest frontiers of developmental biology.

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