The SAGE Encyclopedia of Lifespan Human Development

Developmental Plasticity

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Developmental plasticity is the ability of an organism to tailor its phenotype (i.e., its observable traits) to environmental conditions and to its somatic condition. This ability is widespread in nature: It exists in all life forms, including mammals, birds, fish, reptiles, amphibians, invertebrates, and plants. This entry discusses developmental plasticity, its evolutionary origins and function, the physiological mechanisms that mediate it, individual differences, and sensitive periods.

Origins

Humans exhibit developmental plasticity in morphological, psychological, and behavioral traits, and these often change in concert. For example, given equal access to energy, females who grow up in harsh environments (e.g., violent neighborhoods) tend to mature faster, experience menarche earlier, develop an interest in having children at a younger age, engage in sex earlier, and have their first child at a younger age. This suite of correlated responses might be adaptive: It shifts an individual to earlier reproduction in conditions where life expectancy is low. Humans share this *life history* response with other mammals—including certain species of monkeys and rodents—suggesting ancient evolutionary roots. However, more recently evolved traits, such as human language, also exhibit developmental plasticity. Thus, the origins of developmental plasticity might be different for different traits. Despite variation in origins, in each case, similar selection pressures might have favored developmental plasticity.

Function

Developmental plasticity entails long-term effects of earlier experiences. The adaptive value of such lasting effects depends, among other things, on the stability of environmental conditions. If the environment is stable generation after generation, so that all individuals experience the same conditions, there is no value to plasticity: Natural selection might favor fixed phenotypes that are specialized for the prevailing conditions (e.g., all polar bears are exposed to cold climates, therefore all polar bears develop fur coats). In contrast, if conditions are variable between individuals but are stable within a single lifetime (e.g., some people are consistently exposed to safe conditions, others to dangerous conditions), natural selection might favor developmental plasticity. Early experiences predict later experiences; hence, individuals can adjust their development based on them. If, however, an environment is variable even within lifetimes (e.g., the same person experiences safe and dangerous conditions over time), natural selection can favor lifelong flexibility, as opposed to lasting effects of earlier experiences. In humans, some traits are fixed (e.g., number of eyes), others are developmentally plastic (e.g., life histories), and still others exhibit lifelong flexibility (e.g., the ability to learn new skills).

The adaptive value of developmental plasticity depends also on the stability of somatic conditions. For example, if early-life stressors (e.g., famine, toxins) hinder the construction and maintenance of a healthy body (e.g., cell repair, immune functioning), individuals are likely to face a faster somatic decline and may tailor their development based on their poor prospects (e.g., by accelerating their life histories). This response does not depend on autocorrelation in the external environment (e.g., if there is drought today, there will be drought tomorrow) but on autocorrelation of an organism's internal condition (e.g., if my body is in a poor state today, it will also be tomorrow, irrespective of the external environment). The external and internal accounts of developmental plasticity are mutually compatible and their

predictions often converge, but not always. For example, the internal account predicts that within a given environment, individuals who have incurred lasting somatic damage (e.g., due to parasite exposures) will develop faster life histories than individuals who have not incurred such damage. The external account would not predict this, as all individuals in this environment will be equally likely to suffer in the future from death and disability caused by external conditions.

Physiological Mechanisms

The physiological pathways through which developmental plasticity affects an individual's phenotype operate at different levels and through diverse mechanisms. Early in the development, stem cells can be affected by environmental conditions, in turn determining the growth and differentiation of organs and tissues. Subsequently, there may be changes in the speed of maturation of organs and in the functioning of physiological control systems as a result of interactions with the environment. The relative growth of organs will be the result of factors such as tissue metabolic demands, growth hormone levels, and blood supply. Control systems that will be affected by the prenatal and early postnatal environments are hypothalamic function, stress systems—the hypothalamic–pituitary–adrenal axis and the sympathetic nervous system—and appetite. Parallel to effects on the individual's development are possible effects on commensal species, such as the intestinal microbiota, in turn having further reciprocal consequences for the developing individual.

Development in early life is especially susceptible to environmental variations in nutrients, stress, and toxins. The physiological mediators of these environmental factors are placental function, the stress system, hormones, and immunity. The phenotype of many bodily structures can be affected: from cellular organelles (e.g., mitochondria), through cells (e.g. neurons; pancreatic, cardiac, and adipose cells), to organs (e.g., liver, lungs, gut, gonads). In turn, phenotypic function is affected, again in a large range of outcomes, including behavior, learning, stress responses, immune responses, metabolism, reproduction, and aging. These physiological pathways, leading from environmental challenge to phenotypic function, are complex, diverse, and a basis of human developmental plasticity.

On a cellular level, the physiological mechanisms underlying developmental plasticity are often the result of epigenetic processes. Epigenetic modifications are changes in gene expression that do not alter the underlying gene sequence. Hence, phenotypic variations in humans are often caused by differences in long-term *programming* of gene function rather than the genetic sequence *per se*. In contrast to the gene sequence, epigenetic mechanisms are dynamic and potentially reversible, making them an interesting target for therapeutic interventions. Indeed, clinical trial research on drugs aimed at the epigenetic machinery is on the rise for both physical and mental diseases. Epigenetic mechanisms include DNA methylation, histone modifications, and noncoding RNAs. Epigenetic modifications induced during development are both highly cell- and gene-specific.

Another physiological mechanism at the cellular level relevant to life-span developmental plasticity processes is that of telomeres. The telomere is a protein–DNA complex that protects chromosome ends. It is a highly regulated and dynamic complex that shortens over the course of cell divisions, leading to attrition of chromosome ends. The enzyme telomerase can compensate for this attrition by adding telomeric sequences to the end of chromosomes, hence elongating them again. However, in many human cell types, the levels of telomerase are limiting, and telomeres become shorter throughout the life span. Telomere length is therefore a marker for risks of common, often comorbid, diseases of aging as well as for

mortality risk. In other words, as humans grow older, the average telomere length declines, and mortality increases. Telomere length is partly heritable and partly determined by nongenetic influences such as early environmental stress. In developmental studies, shorter telomere length is used as a biological marker of accelerated aging, or a faster life history strategy, as stress early in life is associated with shorter telomeres.

Individual Differences

Although all people exhibit developmental plasticity, some people are more affected than others by the same experiences (e.g., the death of a parent). Understanding these individual differences can illuminate why individuals benefit or suffer differentially from such experiences as nurturance or abuse and why clinical interventions are more or less effective for different individuals at different times. One line of research investigates genotype-by-environment interactions. This work examines how genotypic factors shape individuals' levels of responsiveness to specific environmental influences. For example, maltreatment in childhood predicts antisocial behavior in some adolescent males and not others depending on their genotype. A second line of work examines how earlier experiences shape later levels of plasticity. For example, people who grow up under extreme conditions—either very stressful or very supportive—may develop heightened reactivity in neurobiological stress systems, which might enhance their developmental plasticity. An open question is whether individual differences in developmental plasticity are biologically adaptive or a product of nonadaptive processes, such as genetic or developmental noise.

Sensitive Periods

As developmental plasticity concerns lasting effects of experience on ontogeny, it implies sensitive periods, in which experience shapes phenotypic development to a larger extent than in other periods. Sensitive periods are important for scientific as well as applied reasons, such as the timing of interventions. In humans, there are sensitive periods in a wide variety of traits, including aspects of metabolic physiology and skeletal morphology, visual and auditory perception, food and mate preferences, attachment styles, stress responses, and reproductive strategies. In nonhuman animals, such as rodents and birds, researchers do not only document sensitive periods but are also able to modify aspects of them (such as their onset and offset) through environmental or pharmacological manipulation. This work holds great promise for future interventions in humans.

See alsoAdaptation; Adaptive Functioning; Development; Developmental Origins of Health and Disease; Developmental Theory; Developmental Trajectories; Ecology; Evolution; Evolutionary Theory; Goodness of Fit; Life History Theory; Nature–Nurture; Plasticity; Sensitive Period

- plasticity
- sensitive period
- life histories
- phenotype
- polar bears
- autocorrelation
- genes

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Further Readings

Beijers, R., Buitelaar, J. K., & de Weerth, C. (2014). Mechanisms underlying the effects of prenatal psychosocial stress on child outcomes: Beyond the HPA axis. European Child & Adolescent Psychiatry, 23, 943–956.

Belsky, J., Steinberg, L., & Draper, P. (1991). Childhood experience, interpersonal development, and reproductive strategy: An evolutionary theory of socialization. Child Development, 62, 647–670.

Blackburn, E. H., Epel, E. S., & Lin, J. (2015). Human telomere biology: A contributory and interactive factor in aging, disease risks, and protection. Science, 350(6265), 1193–1198.

Ellis, B. J., Boyce, W. T., Belsky, J., Bakermans-Kranenburg, M. J., & van IJzendoorn, M. H. (2011). Differential susceptibility to the environment: An evolutionary neuro-developmental theory. Development and Psychopathology, 23, 7–28.

Ellis, B. J., & Del Giudice, M. (2014). Beyond allostatic load: Rethinking the role of stress in regulating human development. Development and Psychopathology, 26, 1–20.

Frankenhuis, W. E., & Fraley, R. C. (in press). What do evolutionary models teach us about sensitive periods in psychological development? European Psychologist. doi:10.1027/1016-9040/a000265

Groothuis, T. G., & Taborsky, B. (2015). Introducing biological realism into the study of developmental plasticity in behaviour. Frontiers in Zoology, 12(S1), 1–14.

Hanson, M. A., & Gluckman, P. D. (2014). Early developmental conditioning of later health and disease: Physiology or pathophysiology? Physiological Reviews, 94(4), 1027–1076.

Rickard, I. J., Frankenhuis, W. E., & Nettle, D. (2014). Why are childhood family factors associated with timing of maturation? A role for internal state. Perspectives on Psychological Science, 9, 3–15.

Takesian, A. E., & Hensch, T. K. (2013). Balancing plasticity/stability across brain development. Progress in Brain Research, 207, 3–34.