

Exercise 3: Developmental Origins of Adult Disease: Evolution, Environment, Phenogenetics, and Disease

After completing this exercise, you will be able to: (1) describe a framework for thinking about health and development across the life span; (2) explain what is meant by a life course approach to the study of adult chronic disease; (3) discuss the work of DJP Barker and related research on "fetal origins of adult disease"; (4) describe the mechanisms responsible for associations between fetal/early life factors and adult chronic disease; (5) describe some challenges of researching fetal origins of adult disease; (6) discuss some of the implications of this work for maternal and child health policies and programs around the world.

This exercise is based on Figures 2 and 3 in Gluckman, PD and Hanson, MA. (2004). Living with the past: evolution, development and patterns of disease. Science. 305:1733-1736.

Figure 2 depicts "A general model of how intergenerational, genetic and environmental, and prenatal and postnatal factors interact to create a pathway of altered disease risk in adulthood."

Figure 3 is a depiction of the Predictive Adaptive Response Model (PAR). Also shown on the page with Figure 3 is an alternative depiction of the PAR model developed by Gluckman and his colleagues in a later publication (Gluckman, et al., 2005).

Note that a number of technical concepts and terms related to molecular genetics are introduced in this paper. Please review the Appendix for a general explanation of some basic concepts.

1. What kinds of time periods are involved for the "past history of population" to affect the genotype?
2. What is meant by "epigenetic change," and how do "intergenerational environmental effects" have an influence?
3. Does the link between "prenatal environment" and "epigenetic change" relate to the Barker hypothesis?
4. What is the significance of a "match" between the prenatal and postnatal environment?
5. What is the meaning/significance of the "predictive adaptive response" or "developmental plasticity," and how does this relate to the "birth phenotype"?
6. Why does the model show that the "postnatal environment" has strong a direct effect on the "adult phenotype"?
7. Are the links between "fetal phenotype," the "adult phenotype," and "disease risk" indicative of a life course model of disease?
8. What is the point the authors are trying to make in the last figure? How does this relate to the prospects for an emerging epidemic of chronic disease in less-developed countries as they improve their economic conditions?