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A post-genomic view of behavioral development and adaptation to the environment



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ABSTRACT

Recent advances in molecular genetics and epigenetics are reviewed that have major implications for the bio-behavioral sciences and for understanding how organisms adapt to their environments at both phylogenetic and ontogenic levels. From a post-genomics perspective, the environment is as crucial as the DNA sequence for constructing the phenotype, and as a source of information in trying to predict phenotypes. The review is organized with respect to four basic processes by which phenotypes adapt to environmental challenges, with an emphasis on the data for humans: (1) developmental plasticity, (2) epigenetic mechanisms, (3) genotype-environment correlations, and (4) gene × environment interactions.

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Introduction

Two landmark events in the history of genetics that occurred during the lifetime of many working scientists have each heralded paradigm shifts with broad implications for the bio-behavioral sciences. The discovery of the molecular structure of DNA in 1953 by Watson and Crick ushered in a fertile period of research generated by the successful integration of molecular genetics within the paradigm of the Modern Synthesis. This perspective viewed natural selection as the key mechanism for the evolution of new life forms from within-species variation generated principally from random mutations of structural DNA, the sole biological agent involved in heritability. Fifty years later momentum would build for a new paradigm that would call into question and eventually overturn this dominant paradigm. Completed in 2003, the Human Genome Project (HGP) was a principal catalyst in this

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genetics revolution. This 13-year, \$3 billion project, coordinated by the US Department of Energy and the National Institutes of Health, with additional contributions coming from the UK, Japan, France, Germany, China, remains one of the largest single investigative projects in modern science. Once the principal goal of sequencing the three billion chemical units in the human genome was accomplished, the next step was to identify the genetic variants that increase the risk for common diseases.

In announcing on June 26, 2000, that the first draft of the human genome project had been achieved, then US President Clinton said it would “revolutionize the diagnosis, prevention and treatment of most, if not all, human diseases.” This statement accurately reflected the optimistic consensus among scientists at that time. Instead, after another decade of research the results of the HGP have yielded very little gain in medical practice, as the common disease variants investigated have turned out to explain just a tiny fraction of the genetic risk (Goldstein, 2009). However, if the project did not revolutionize medicine, it did revolutionize science. The HGP, and the research directions it stimulated in molecular genetics, epigenetics, and genomics, have led to a radically new understanding of the fundamental relationship between genotype and phenotype that was largely unanticipated by most scientists at the inception of the HGP and still poorly understood today.

For example, one of the primary goals of the Human Genome Project was to identify all the genes in human DNA. Before the HGP, some scientists had estimated that the known three billion or so DNA letters necessitated a hundred thousand or more genes, to match the one million or so proteins in the human organism (Bernot, 2004). Some scientific estimates of the number of genes in the human genome at the start of the project were as high as 200,000. In 2004, researchers from the International Human Genome Sequencing Consortium (IHGSC) of the HGP shocked the scientific community with a new estimate of just 20,000–25,000 genes in the human genome. This is the same range as in mice and roundworms, and considerably less than the 32,000 genes found in an ear of corn (Schnable et al., 2009). Such a wildly miscalculated prediction necessitated a rethinking of the basic assumption that each protein was produced by a specific gene, with each gene containing the instructions for making just one protein. These assumptions created the expectation of a near perfect correlation between level of anatomical complexity in a species and the degree of complexity in their DNA. However, we now know that there is no such correlation. The genetic complexity of many simpler organisms like algae, mosses and salamanders exceeds that of many complex species of birds and mammals, including humans, a situation known as the C-value paradox. This would prove to be just one of many assumptions of the previous paradigm that would fall in the face of strong counter evidence.

The goal of this paper is to review more recent advances in molecular genetics that have major implications for the bio-behavioral sciences informed by genetics. In particular, we consider how to accommodate this body of research into a general framework for understanding how organisms mesh with environments. From a post-genomics perspective, the environment is as crucial as the DNA sequence for constructing the phenotype, and as a source of information in trying to predict phenotypes. Matching phenotypes with their environments is the critical adaptive problem, at both phylogenetic and ontogenic levels. After a brief summary of the assumptions of evolutionary models of development, we organize our discussion with respect to the basic processes by which phenotypes become adapted to their environments, with an emphasis on the data for humans.

Basic concepts: The perspective of evolutionary psychology

The basic evolutionary model of development emphasizes the smooth, reliable development of adaptations—mechanisms designed to solve problems that were recurrent over evolutionary time. The current view of evolutionary psychology is that these problems were solved by evolving a set of psychological mechanisms designed to deal with these specific recurrent problems. A key point for development is reliability across the range of environments that constitutes the evolutionarily expected range of environmental variation. That is, no matter how complex the transactions between genes and environments, ultimately adaptations must be reliably evolving across the range of evolutionarily expected environments.

An important general issue therefore is whether the environment being matched is an environment that is part of the evolutionary history of the organism—implying the concept of an evolutionarily

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