BOOK REVIEW: *Twin Research*

Matt Ridley. 2003. *Nature via Nurture: Genes, Experience, and What Makes Us Human*. New York: HarperCollins.

Reviewed by

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Bench scientists tend to scowl at the science—public opinion interface as a sleaze zone of exaggeration, sensation-seeking, misreporting, and in the worst case, "junk science." The scowl is usually accompanied by a personal decision not to get involved. That assessment is alas often on target. But it doesn't address the fact that interface there must be. The quality of the interface is important, beginning with the elementary point that science depends for its funding on tax dollars and voluntary contributions. Disengagement from public opinion isn't an option.

Matt Ridley is among the handful of authors who have harvested deserved acclaim for rendering the complexities of genetics into everyday idiom. This is his fourth and as I believe, his best effort. He commences his journey with an example of the messy science-public interface—the story about Craig Venter's headline-grabbing stunt concerning the announcement of the Human Genome Project result. Venter's Celera Genomics was party to agreement with the International Genome Project and the Sanger Institute for a joint

announcement of the result, but a week prior to the agreed release date Venter went public with the news that the human genome had far fewer genes (30,000) than anticipated. To this he added the spin that the result "refutes" the idea of biological determinism. "The wonderful diversity of the human species," he proclaimed, "is not hard-wired in our genetic code. Our environments are critical." Thus a leading genome scientist casually abandoned a solemn agreement in order to capture the limelight of the highest profile science story of the decade. It was a disconcerting example of opportunism trumping basic professional ethics.

Ridley easily disposes of Venter's spin and adroitly converts it to a launch pad for his study. The real issue isn't the nature vs nurture, but how nature and nurture interplay to produce the diversities and uniformities of human nature. His alternative formulation, nature via nurture (or experience), asserts that "genes are designed to take their cues from nurture." "My argument in a nutshell is this: the more we lift the lid on the genome, the more vulnerable to experience genes appear to be." (4) Otherwise stated, we've come a long way from the Beadle-Tatum dictum 'one gene, one enzyme, one protein', which expressed the core of unidirectional genic thinking that in its day was good enough for the Nobel Prize.

Ridley devises an ingenious fantasy to organize his complex material. He invites the reader to imagine twelve men assembled at the fashionable Biarritz spa in 1903. They are Francis Galton, William James, Hugo De Vries, Ivan Pavlov, John B. Watson, Ivan Pavlov, Emil Kraepelin, Sigmund Freud, Emile

Durkheim, Franz Boas, Konrad Lorenz, and Jean Piaget. Each grasped something important about human nature although each also committed errors, especially errors exaggerating the scope of their insights. Ridley suggests that if this company really did assemble, they would have entangled in argument rather than pooled their genius into a higher synthesis. However, their conflicts are serviceable to Ridley as a guide to the century-long entanglement of the nature-nurture controversy. His twelve apostles provide a thread of biographical anecdotes which he uses to exemplify the interaction of doing science with personal strengths and failings and with social context. This stream of the book adapts it to a general audience, but it also enables him to discuss the controversies about human nature that so often swirled around the great men. The controversies aren't merely human interest diversion; they are themselves evidence relevant for interpreting human nature.

Readers will be gratified that the author devotes considerable space to behaviour genetics and even more gratified that he clarifies numerous aspects of the field, such as the meaning of heritability, that are stumbling blocks for the non-specialists. Twin studies in particular are discussed in detail; Ridley leaves no doubt that they provide strong evidence for trait heritability, including one of the five personality factors (neuroticism) and intelligence. That brain size correlates with intelligence has long been suspected, based on comparative evidence of encephalization in mammals, primates in particular. But can it be proved? The discovery of a gene, or genes, for *g* would settle the question. The investigation of the PLP and

ASPM genes as candidates for the *g* gene is reviewed and declared inconclusive, but the finding of a 95 percent correlation of gray matter in MZ twins is recognized to be a significant confirmation that gray matter volume is "due completely [sic] to genetic factors." Ridley leaves it with the statement that "some of the genes of *g* will soon be found." The role of environment in determining *g* seems to be mainly of a background character. He sites Eric Turkheimer's study of 350 twin pairs, many of whom were drawn from impoverished families, as evidence that nutritional and sanitation components of nurturing impede brain development. While elucidating exactly what deficits impede development is an important research program, the global conclusion, *that* failure to thrive imposes life-long losses, seems obvious and remedial, at least in principle. On the other side of the coin is the rise of average IQ scores at the rate of five points per decade (the Flynn Effect). This is probably due to improvements in nutrition, sanitation, and health care, plus increased stimulus to realize native ability. These conditions have produced steady increases in a range of skills, such as athletic performance, as well as height and strength.

If the ontogeny of intelligence is relatively stable across a wide variety of nurturance, schizophrenia exemplifies an extreme of multifactoral causality. The prevalence of schizophrenia is about 1.3 percent of adult populations, slightly less than bipolar disorder. The condition was first recognized by Emil Kraepelin; Eugen Bleuler improved the diagnostic criteria and conferred the name, schizophrenia. The extraordinary mingling of thought disorder (memory, attention, problem solving) and emotional disorder (ranging from

catatonic flattening of effect to traumatic paranoid delusion) has meant that the development of reliable diagnostic criteria, etiology, and treatment regimes has been slow and error-burdened. Psychoanalysts, Ridley tells us, were the first to propose that the catatonic symptoms of schizophrenia were the result of harsh and unsympathetic mothering (the 'refrigerator mother'). This diagnosis was extended to autism. The diagnosis had rebound effect on parents, who, already stressed by the autistic child's behaviour, were stressed even more by the thought that their parenting induced the disorder. But even as the nurturing etiology achieved dominance, twin studies by Aaron Rosenoff and Franz Kallmann between the wars were revealing high concordance (68 percent and 86 percent, respectively) of schizophrenia in MZ twins (as opposed to 15 percent concordance in DZ twins). (104) Despite intense hostility from psychiatrists, post-war studies of the heritability of schizophrenia resulted, in 1988, in the discovery of the "schizophrenia gene," so identified by a marker on chromosome 5. However, this finding proved to be one of many faulty identifications in the initial boom years of molecular genetics: it turned out that all but six chromosomes had schizophrenia markers. (107) The error was driven by extending the single gene etiology of Huntington's chorea and cystic fibrosis (confirming the Beadle-Tatum dictum) to all neurogenic illness. Neurotransmitters were another line of attack. Initially dopamine was singled out, but there proved to be five kinds of dopamine on different chromosomes; the attempt to identify a deficiency specific to schizophrenia fizzled. The next neurotransmitter investigated was serotonin. It too failed, but the data derived from the then new microarray techniques for measuring gene expression supported the concept that schizophrenia involves some dysfunction in

prefrontal cortex synapses. Ridley next follows this extraordinary story down the paths of influenza viral involvement, developmental dysfunction, and deficiency of cellular release of the fatty acids. In each case he diligently specifies the evidence that stimulated a research path and the outcomes that denied the research a univocal conclusion. After this careful preparation, Ridley suggests that schizophrenia may not simplify to univocal causality but may require circular causality, where A and B are mutually cause and effect. If so, the situation is “a perfect illustration that nature and nurture both matter.” (124)

The most overt confrontation between nature and nurture is the blank slate agenda of conditioning theory (Pavlov, Watson, Skinner) and nativism in the form of imprinting, promoted by Lorenz, Tinbergen, Piaget, Harlow, and cognitive psychology. Conditioning theory had historical roots back to John Locke. In the eighteenth and nineteenth centuries, it was associated with meliorist agendas such as utilitarian liberalism and socialism. Meliorism was thrust into high focus by the rise of social welfare professions. Pavlov’s experiments, if not his theory, assumed that the psychological and neurological space between sensory excitation and the motor response could be black boxed without loss of descriptive accuracy. Watson and Skinner, by contrast, declared triumphantly that there was nothing in the black box, hence there was no limit to meliorist social control. (Their initiative was paralleled in anthropology by Franz Boas, who attacked the concept of an evolutionary development for the human species in a graded gene-culture co-evolutionary hierarchy). Ridley’s treatment of this confrontation was for me the most

illuminating and instructive section of the book. He organizes his material to show how Lorenz' key insight into the existence of critical developmental times (an idea inaccessible to conditioning theory) escaped the limitations of Lorenz's own partiality for "instincts" to become the detailed and complex description and explanation of the developmental biology of many species. A proper summary of this material is not possible in this limited space. Suffice to say that the nature via nurture theme finds its most persuasive and instructive elucidation in this portion of the book (125-176). I read it three times, and continued to learn on each run.

Having praised the book's merits, what might be said in criticism? I feel that his enthusiasm leads Ridley to underestimates the extent of the attachment to the nurture-only point of view. The American Anthropological Association, for example, has adopted a statement on race which declares, directly contrary to Ridley's view, that "human cultural behaviour is learned, conditioned into infants beginning at birth, and always subject to modification … Our temperaments, dispositions, and personalities, regardless of genetic propensities, are developed within sets of meanings and values that we call 'culture'. Studies of infant and early childhood learning and behavior attest to the reality of our cultures in forming who we are … it is a basic tenet of anthropological knowledge that all normal human beings have the capacity to learn any cultural behavior" (www.aaanet.org/stmts/racepp.htm). This is exactly where Franz Boas positioned anthropology eighty years ago. It is a perspective that optimizes the meliorist vision. But what if all normal persons

cannot learn algebra or excel in sport? What if there are indeed heritable race differences in behaviour? The struggle isn't over yet.