Book Review


This is the “Decade of the Brain,” which only makes official a scientific phenomenon that began long before the nineties: biological psychiatry is in the ascendant. Our contemporary era of biological and genetic hegemony has given rise to a revisionist tradition that attacks the biological school’s theoretical underpinnings and empirical results on a combination of philosophical, ideological, historical, political, and scientific grounds. The revisionists have produced some classics, most notably Thomas Szasz “The Myth of Mental Illness” [1961], Lewontin, Rose, and Kamin’s “Not in Our Genes” [1984], and Stephen Jay Gould’s “The Mismeasure of Man” [1981], but like most out-gunned guerilla movements, the revisionists (who, in different contexts, are called anti-biologists, anti-hereditarians, or anti-psychiatrists) mix an admirably quixotic intellectual passion with the guerilla’s tendency for oversimplification, fanaticism, and even cruelty.

“Pseudoscience in Biological Psychiatry,” by Colin A. Ross and Alvin Pam, a contribution to this anti-biological tradition, displays most of the weaknesses of its forebears and few of their strengths: the blunt lack of subtlety typical of Szasz’ argumentation, without the bracing vigor of his rhetoric; Kamin’s propensity for ad hominem argument without his dogged attention to detail; and Gould’s preference for the historical over the contemporary, without the elegance of his style or the breadth of his knowledge of biology and evolution. This book adopts a peculiar format, halfway between an edited volume and a co-authored monograph. The first two chapters, which form the heart of the text, are by Pam and Ross, respectively; the third (an idiosyncratically annotated bibliography of papers from The American Journal of Psychiatry) and concluding chapters are also contributed by Ross. In between are four chapters by other authors that range from hackneyed (Ellen M. Borges’ “A Social Critique of Biological Psychiatry,” to cheerfully meshuggeneh (Harry Wiener’s “The Genetics of Preposterous Conditions,” which the editors introduce with this ringing endorsement: “The reader is invited to see what flaws, if any, can be found in Wiener’s exposition”).

That said, it may come as a surprise that the fundamental reason I was disappointed by the mish-mash of arguments presented by Ross, Pam, and colleagues is that in large part I agree with their conclusion: biological psychiatry, and the biogenetic, neuroscientific mode of explanation of which it is a part, have been oversold, to the serious detriment of the scientific study of behavior. Ross and Pam, like their predecessors, are fighting a good fight for the wrong reasons and guaranteeing their own defeat in the process.

The case for biological psychiatry rests on two pillars. The first is empirical: an ever-growing body of evidence purports to show that variation in behavior is associated with variation in the genotype; that drugs can effectively ameliorate the behaviors typical of serious psychopathology; and that neurological phenomena are reliably associated with manifestations of mental illness. The second pillar is theoretical: it is claimed on the basis of the empirical results that psychopathology is properly considered a biological phenomenon rather than a psychological one. The opponent of biological supremacy must choose which of these “pussums” to chase, and Ross and Pam, like earlier anti-biologists, take off after the wrong one and attempt to build a case against the empirical basis of the biological school.

Ross and Pam never question the biological psychiatrist’s fundamental assumption that it is reasonable to divide psychological phenomena between the psychological and the biological. Although they are skeptical of E. Fuller Torrey’s assertion that schizophrenia is a “brain disorder,” Ross and Pam themselves produce statements like, “Biological factors enter into all behavior, including symptoms, but only rarely are the cause and significance of a patient’s behavior primarily biologically driven.” What is a “biological factor,” and how can it “enter into” behavior? What is the difference between the “cause” and “significance” of behavior, and what does it mean for behavior to be “biologically driven?” Very difficult questions, and the answers are not to be found in this book. Ross and Pam tacitly accept the biological psychiatrists’ assertion that behavior is either biological or psychological, and furthermore that the contest between the two depends on the outcome of the empirical studies outlined above. Is a behavior heritable? Can it be altered by drugs? Is it associated with neurological findings? Then it must be biological. Ross and Pam stake their ground on the argument that the psychobiologists’ empirical findings are illusory, based on transparently flawed “pseudo-scientific” methodology.

The difficulties of making this improbable case leave Ross and Pam in a position roughly equivalent to that of tobacco companies as regards the link between smoking and lung cancer. Yes, it’s true that we can’t randomly assign humans to smoking and non-smoking conditions, and in the absence of strict experimental control it is always possible to poke little holes in causal inferences based on correlational associations between smoking and disease. Ross and Pam trot out all the usual suspects: special MZ twin environments, correlated environments in presumably separated twins, methodological and diagnostic inconsistencies in older studies, and difficulties in defining spectrum concepts of mental illness. But, as is always the case when these
arguments are presented (and they have been presented many times before, and much more compellingly) the "methodologically correct" study, the one that would presumably demonstrate vanishingly small heritabilities, is never cited because it does not exist. Quasi-experimental (as opposed to pseudo-) science proceeds by studying a phenomenon from every angle, each with different methodological distortions, and observing whether consistent results emerge across methodologies: heritability and psychobiological association, for whatever they are worth, have passed this test beyond plausible refutation.

The lacunae in Ross' and Pam's presentations of psychiatric genetics provide clues to the underlying conceptual problem in their broader case against biological psychiatry. Ross, astoundingly, appears completely unaware of the existence of genetic epidemiology. He claims that psychiatric genetics is based on the mistaken notion that, "If it runs in families, it must be genetic," and provides ridiculously obvious examples to invalidate his straw man. Pam's discussion is a little more sophisticated. He understands, at least, that psychiatric genetics is not based on familial associations. He does, however, appear to believe that modern psychiatric genetics is still based on Mendelian principles, with satisfaction that the risks for schizophrenia in relatives of affected probands are not in accordance with the predictions of "Mendelian theory" (p. 18). Of course they are not, but then again psychiatric genetics has not been based on Mendelian theory for at least 30 years. The predominant statistical paradigm in modern psychiatric genetics is the multifactorial threshold (MFT) model, according to which genetic transmission is the result of the additive combination of independently assorting loci. Pam finally gets around to the MFT model, but dismissively, at the end of his chapter. He calls it "a statistical or conceptual artifact," used by biological determinists to explain away anomalous findings.

This is the crux of the matter. Throughout the book, Ross, Pam, and especially Wiener ridicule recent behavior genetic reports of heritabilities of "preposterous" characteristics like political attitudes [Eaves et al., 1989] and television watching in children [Plomin, Corley et al., 1990]. What, exactly, is preposterous about the notion that there is a genetic component to individual differences in television watching? The implausibility resides in the possibility that there would be a major gene for television watching, for the obvious genetic reason that television did not exist during our evolution, and also because the idea violates our introspective intuition that political attitudes and television habits are the results of complex, multi-threaded interactions among our physical bodies, our consciousness and our environment. But neither of these objections presents a problem for a MFT model of inheritance. There is no gene for "I Dream of Jeannie." Rather, there is a multitude of genes, many with pleiotropic and epistatic effects, all of them expressed in exquisite interaction with the environment, which, when their effects are grossly summed together, add up to a risk for tuning in Nickelodeon at 8:00 Monday to Friday. And this process, in its nearly infinite complexity, is what we introspectively perceive as the psychological determination of our behavior. There is nothing preposterous about it.

Polygenic transmission is neither a statistical nor a conceptual artifact: it is a real hypothesis about the genetic etiology of human behavior. And here is where Ross, Pam, and the whole anti-psychiatric tradition has missed the boat, because the great irony of the success of the MFT model in identifying heritable components in the transmission of everything from schizophrenia to marital status [McGue and Lykken, 1992] is that the etiological consequences of the model provide no basis for undertaking a biological research program to explain the etiology of the phenomena. In fact, to the extent that MFT models are tested against competing genetic models that include a big gene (and when they are compared in the realm of human behavior, the MFT model is almost invariably superior), the MFT model decisively refutes the possibility that a reductionist research paradigm will bear fruit. What are the causes of television watching in children? Other than repeating the banality that they include "genetic influence," the behavior geneticist armed only with the MFT model is unable to say.

The anti-reductionist etiological consequences of the MFT model are a half-acknowledged secret in the domain of neuropsychiatric genetics. Faraone and Tsuang [1985], before embarking on a review decisively favoring multifactorial threshold models, put it this way:

The mode of inheritance has substantial implications for etiological research and clinical practice. A conclusive demonstration that a single major locus is involved in schizophrenia would hold the promise that a relatively direct biochemical pathway accounts for the psychophysiology of the disorder. If a multifactorial polygenic model describes the mode of transmission, the search for a simple biochemical pathway is likely to be less fruitful.

(p. 44)

Faraone and Tsuang then cite Kidd [1981], who put it as follows: "Moreover, the [MFT] model can provide little insight into the genetic system. The very nature of the model relegates genetic factors to an amorphous pool of small indistinguishable components" (p. 374).

Despite these reservations, genetic epidemiology has provided the springboard for biomedical investigations of major psychopathology and, more recently, molecular genetic investigations of human intelligence. Are we now ready to embark on the neurobiology of television watching, the molecular genetics of divorce? If not, it must be explained how such complex human behaviors (which are, remember, demonstrably heritable) differ from Huntington's disease and (so most of us would like to think) schizophrenia.

The problem with biological psychiatry is not its assertion that MZ twins are more similar politically than their DZ counterparts, or that drugs are often an effective treatment of psychiatric symptoms, or that the brains of very anxious people are different than the
brains of normal people. All of these assertions are true, not preposterous but obvious and even trivial, and the opponent of biological psychiatry who sets out to refute them is barking up the wrong tree. What’s worse, biological psychiatry’s opponents are so busy barking that they have failed to notice that the real possum is sitting comfortably out on a limb, just a couple of trees down. No amount of barking will bring him to ground, but a few simple questions will shake the tree hard, from its roots. Try this the next time someone explains to you that the heritability of something is greater than zero, or that differences in behavior are associated with differences in brains: Ask, “So what?” and, “What next?”

REFERENCES