Medical Comorbidity and Schizophrenia: Editors’ Introduction

by Richard C. Josiassen and Barbara Schindler

Abstract

Schizophrenia is a heavily biologically determined psychiatric disorder with unequivocal evidence for a major genetic component in its transmission. In addition to its genetic determination, schizophrenia is also associated with increased morbidity and mortality with illness and suffering and with death rates that are higher than would be expected. One of the intriguing possibilities is that there may be some biological link between genetic predisposition to schizophrenia and to other medical diseases. This research on medical comorbidity, originally presented at the Tenth Annual Pennsylvania Conference on Schizophrenia at Norristown State Hospital, requires further research and understanding.


Schizophrenia is a psychiatric disorder, and psychiatric disorders are medical disorders. However, the most obvious or direct manifestations of schizophrenia—the signs and symptoms by which we diagnose the disorder, trace its course, and familiarly recognize it—are for the most part not what we usually think of as medical, but rather behavioral, psychological, and social. Therefore, this edition of the Schizophrenia Bulletin on medical comorbidity is, in a way, meant to remind us about a fact that we easily forget.

Although schizophrenia is a psychiatric disorder, we know that it is a largely biologically determined psychiatric disorder with unequivocal evidence for a major genetic component in its transmission. We also know that in addition to its largely genetic determination, it is also associated with increased morbidity and mortality, with illness and suffering, and with death rates that are higher than would be expected. Some of this increased morbidity and mortality can be explained by things we understand about the disease. However, our understanding is far from complete, and further research is required.

One of the intriguing possibilities that has not yet been explored is that there may be some biological link between the genetic predisposition to schizophrenia and the predisposition to other medical diseases. We know, of course, that any genetically determined disorder has a genetic presence in every cell—not just in the brain, not just in the central nervous system, but in every cell of the body. So there is the potential for gene expression in any tissue. It is easy to develop conceptual schemes for why there might be physical disorders linked to schizophrenia. Is polydipsia or atypical response to medication or the unusually high rate of tobacco consumption a manifestation of schizophrenia-linked genes expressed in widespread organ tissue? At this point, these are ideas rather than facts. But they are exciting ideas that may lead to a better understanding of this disorder.

Can the study of comorbidity help elucidate the nature of schizophrenia? Some people think you can only understand schizophrenia when you take it apart at its molecular level. Direct clinical observation has little to offer in terms of adding new molecular knowledge. But the whole is always greater than, or at least differ-

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ent from, the sum of the parts. Direct and thoughtful analytical observation is still a useful tool of psychiatric research. Piaget's observations on cognitive development did not depend on his knowledge of cerebral or synaptic function and remain independently valid. Kraepelin correctly identified manic-depressive psychosis long before anything specific was known about its genetic and metabolic base. All of Darwin's great work depended on direct observation, as did most of Pavlov's discoveries and those of many other illustrious scientists.

In one of his provocative editorials, Joseph Wortis, the founding editor of the journal of Biological Psychiatry, wrote that "scientists with good training in the basic disciplines can enrich the clinical field by bringing their sound habits, skepticism, and caution into the clinic, where great opportunities will be found" (Wortis 1979, p. 719). He went on to note that Julius Wagner von Jauregg, the psychiatrist who won the Nobel Prize in 1927 in medicine and physiology for his discovery of the fever therapy of paresis, told his students in Vienna that at the Steinhof Asylum where he worked as a pathologist, it was common knowledge among the veteran nurses that a bout of fever could induce remission in the back-ward patients. "It was a pot of gold sitting in the middle of the road," he said, "and nobody would pick it up" (Wortis 1979, p. 719). There are plenty of gold nuggets still around, awaiting discovery.

Reference

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