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Introduction

Book section

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Introduction

What causes conditions such as autism and schizophrenia? We have long known that they run in families and therefore must have a genetic dimension, but until now no one has ever been able to specify exactly what it is or discover the critical genes. The same is true of the known environmental, non-genetic factors: it has not been possible to bring them together under one theory, and certainly not to relate them to any genetic mechanism. Indeed, where autism is concerned, leading authorities have recently gone on record to declare that the time has come to give up the search for a single explanation.¹

This book sets out a new approach to mental development and disorder based on recently discovered genetic effects involving not just the inheritance of genes, but their expression. The imprinted brain theory is unique in relating these novel aspects of gene expression to brain development, symptoms, behaviour, and physical side-effects in a way that reveals a new view of the mind, both normal and disturbed. The genetic mechanism in question (so-called imprinting) links together previously unconnected syndromes and suggests that many—and perhaps most—of the symptoms of mental illness can be understood in terms of it. An added bonus is that the theory may be able to explain not only the genetics of mental illness but also some environmental causes of disorders like autism and schizophrenia, such as poisons, poverty, or pathogens. Furthermore, the fact that diet is now known to

affect gene expression means that the theory might even ultimately be able to cast light on the controversial dietary dimension to autism.²

The fundamental genetic insight from which the theory derives is the recent discovery that genes from the mother and from the father are in conflict over the size of their child. Those from the mother favour restraint in growth because she has to gestate and give birth to the baby. But the father's genes demand more of her resources, which are from his point of view the biological equivalent of a free lunch for his offspring. The result has been graphically portrayed as a tug-of-war, and the new theory suggests that a win for the father can sometimes produce children with autistic tendencies, whereas a win for the mother can occasionally result in children being born with an enhanced vulnerability to psychotic disorders in later life. A balanced, no-win situation results in normal development, and is by far the most likely outcome. However, early environmental insults, such as starvation of the mother during pregnancy, can produce essentially the same result because they can both affect gene expression and mimic these genetic effects. Starvation during pregnancy has been shown to alter the expression of key growth-determining genes and could also be seen as mimicking the resource-reducing effect of maternal genes, perhaps explaining why it significantly increases the likelihood of psychotic illness in the children born in such circumstances. Increased birth-weight, on the other hand, might also explain part of the apparent epidemic of milder forms of autism in modern societies, where standards of living have reached unprecedented levels.

However, these novel genetic effects and the environmental factors that sometimes work with them are only part of the story. Where understanding the mind and mental development is concerned, recent research into autism has been revolutionary, and forms the second leg on which the new theory stands. The reason that autism has begun to reveal so much about the mind and mental development is that autistics have been shown to lack—or at least often to be significantly deficient in—certain key cognitive skills which make up normal mental functioning. Whenever such deficits are discovered in science, dramatic advances often result because the deficit-condition indicates the critical

factors for normal development. Visual agnosia and optic ataxia provide telling examples.

Visual agnosia describes a bizarre affliction in which, following brain damage, a person can competently grasp and manipulate objects, but cannot name or recognize them until they can feel them by hand. So, if confronted with a yellow fruit, a person with visual agnosia may remark that the object is definitely yellow, but be unable to say if it is a lemon or a banana until they pick it up and feel its shape. However, optic ataxia describes a parallel disorder in which a person can correctly recognize and name seen objects, but cannot grasp or manipulate them competently, despite having nothing whatsoever wrong with their hands or motor co-ordination independent of sight. These syndromes, along with a great deal of other evidence, reveal that human beings have two distinct but complementary visual systems, each with its own pathway in the brain. Normally we are never aware of them because they work together seamlessly, but in these two rare disorders the failure of one reveals the existence of the other in a way that would otherwise be difficult to discern.³

In a similar kind of way, autistic deficits in key cognitive skills have revealed the basis of normal development and given us a unique and unexpected insight into the mind. Specific cognitive skills often missing or deficient in autism reveal how the normal mind works and explain both the social and cognitive difficulties of autistics. Thanks to these insights, the new theory proposes that the spectrum of autistic disorders is mirrored by a psychotic one, and that where autistics show deficits in mental development, psychotics show pathological overgrowth: cancers of the mind, so to speak. And just like visual agnosia and optic ataxia, the contrasting deficits of autism and psychosis reveal two parallel cognitive systems that normally blend more or less perfectly, but expose their fundamental differences when one or the other disorder supervenes.

I hope that this book will appeal to a wide readership, though there is one type of reader in particular to whom I know this book will appeal, and that is to all those with an interest in or concern with autism. My story begins with the discovery and naming of autism, and

insights from research into autism spectrum disorders provided the key ideas which inspire the theory I shall be setting out. As readers will then I hope see for themselves, autism is the key which unlocks the secrets of the mind!

New theories usually need new terminology, and so in the first two chapters I introduce the twin basic concepts I use in relation to the mind and cognition, and explain why they are preferable to alternatives. The next two chapters are devoted to psychosis, and primarily to the extraordinary Schreber case: the most discussed in the entire psychiatric literature. Why yet another discussion? First, because Schreber's own account illustrates so many important features of paranoid schizophrenia at first hand, and without any intervening interpretation. But more importantly as far as this book is concerned, his symptoms stand in striking contrast to those of autism, and take on a new meaning once they are seen in this context. No one has ever looked at Schreber—or at any other psychotic—in exactly this way before. And although, as I point out, there have been a few who have groped towards the solution I offer here, no one has ever before propounded the simple insight that psychoses such as this are the mirror-image of autism. However, some images in mirrors have to be seen to be believed—particularly where they reverse mentality rather than left and right—and this is why I devote two entire chapters to portraying this one. Without illustration, the basic idea might seem far-fetched, but begins to look very different if examined in the light of what psychotics such as Schreber actually report.

Nevertheless, the striking antithetical symmetry of autism and psychosis would remain just an appearance unless some plausible mechanism could be found to explain it. Chapters 5 and 6 are devoted to revealing what this mechanism is and to showing how imprinted genes routinely produce syndromes with such reversed symptoms. Chapter 5 concentrates on imprinting as the first genetic factor explaining mental disorders such as autism and psychosis, and Chapter 6 adds sex as a second. Readers will find that Schreber makes yet another appearance here, and that the new theory casts an intriguing new light not only on paranoia, but on homosexuality as well.

In my concluding chapter I discuss the implications of the new theory for helping us to understand genius and the madness that is often associated with it. I also consider some literary and cultural insights to which the new ideas give rise, and raise a previously unsuspected possibility thrown up by the view of mental development proposed here. This is that there could be so-called savantism on the psychotic side of the spectrum just as there is on the autistic one, and conclude by considering psychoanalysis as a telling case in point. As I suggest in my final remarks, the imprinted brain theory could offer a new conceptual foundation for modern psychiatry and psychotherapy comparable to that which psychoanalysis promised but failed to deliver.

At the time of writing, the genes implicated in autism and psychosis remained largely unknown. However, the theory set out here makes clear predictions about what kind of genes they should be, how those genes should be expressed, and what kind of effects they should be found to have on development, the brain, and behaviour. Scientific theories ought to be testable, and a precise prediction which is later confirmed is always more persuasive than a retrospective re-interpretation, so this in itself is a good reason for writing this book. As readers will see, it clearly sets out what future researchers should find and as such provides a map to a new conceptual continent, with empirical landmarks and factual features clearly indicated. Nevertheless, and as I shall point out later, there is already much tantalizing evidence confirming the theory, some of it from physical side-effects of the critical genes. Indeed, the strongest single link between genetics and a mental disorder known at the time of writing seems made to order for the imprinted brain theory (that between duplication of maternal chromosome 15 and psychosis explained on pp. 164–165). And as I hope to show, the new way of looking at things makes striking new sense of a lot of what is currently known about the mind and mental illness.

New theories, however well founded on fact or otherwise, inevitably reflect on existing knowledge and ideas, and, even if flawed in themselves, can sometimes cast important new light on longer established ones. Here again the imprinted brain theory has much to offer,

and certainly reflects on some well-worn controversies. An example might be nature/nurture, where it suggests that nurture is only an effective factor in psychiatric illnesses such as schizophrenia and autism where it mimics natural, genetic effects: nurture via nature, you might say, by contrast to the conventional wisdom of nature via nurture.

But when all is said and done, and whatever the final outcome, one thing is clear. Nature, not conventional wisdom, will have the last word! Given the rapid progress now being made in neuroscience and genetics, we should not have to wait long for her verdict.