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The Imprinted Brain

HOW GENES SET THE BALANCE BETWEEN AUTISM AND PSYCHOSIS

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Jessica Kingsley Publishers
London and Philadelphia

First published in 2009
by Jessica Kingsley Publishers
116 Pentonville Road
London N1 9JB, UK
and
400 Market Street, Suite 400
Philadelphia, PA 19106, USA

www.jkp.com

Library of Congress Cataloging in Publication Data
A CIP catalog record for this book is available from the Library of Congress

British Library Cataloguing in Publication Data
A CIP catalogue record for this book is available from the British Library

ISBN 978 1 84905 023 4

ISBN pdf ebook 978 184642 950 7

Printed and bound in Great Britain by
Athenaeum Press, Gateshead, Tyne and Wear

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Preface

The history of science is not always what it ought to be. By rights, Galileo should have had Foucault's pendulum to prove that the Earth turns, and Darwin—not Mendel—should have discovered genetics. Instead of introducing an arbitrary term to avoid it, Einstein should have boldly predicted the expanding universe, and so on. No one knows what the future will say of our times, but if history were the way it should be rather than the way it is, some may look back and think that the late Bill Hamilton—had he lived—should have been the author of this book. As I explain in the main text, Hamilton was the originator of the so-called *selfish gene* view of modern Darwinism which Richard Dawkins famously popularized in the book of that title. The critical findings described here only emerged after Hamilton's untimely death in 2000, and no one knows how differently things would have worked out had he lived. At the very least, Hamilton's own acknowledged autistic tendencies (see pp. 34–35) and his ground-breaking insights into genetic and mental conflict would surely have made the theory outlined here of enormous interest to him, and—who knows?—he might even have been the first to formulate it, so naturally does it follow from the lead he gave. But history, alas, is not always as it should be, and men of genius such as Hamilton do not always live to reap the harvest that should have been theirs. Nevertheless, he was the one who laid the foundations on which his heirs have built and remains the person to whom I owe a fundamental debt of gratitude in writing this book.

However, the greatest debt of gratitude I owe is to my colleague and

co-author, Bernard Crespi, Killam Research Fellow in the Department of Biosciences at Simon Fraser University in Vancouver. At a time when I was temporarily incapacitated, he took up the idea that imprinting might underlie autism and paranoia and both generalized it to include psychosis in general and did much to secure its factual foundations. Without his help, I would have proceeded much more slowly and uncertainly, and probably missed many important insights altogether—particularly where genetics and brain science are concerned. I am particularly indebted to him for parts of Chapters 5 and 6, and for the originals on which the diagrams in Chapters 6 and 7 are based. Where original research has been done by Professor Crespi, I cite his or our joint publications which provide the detailed references; where I cite the original sources myself, the work was done by me independently or in parallel with him.

Additionally I must thank Ahmad Abu-Akel, Ken Aitken, Chris Ashwin, Abdallah Badahdah, Simon Baron-Cohen, Kingsley Browne, Benedict Carey, Martin Conway, Charles Crawford, Diana Fleischman, Temple Grandin, Francesca Happé, Ayla and Nick Humphrey, Satoshi Kanazawa, Ben Kaplan, Nicola Knight, Patrick McNamara, Alex and Marian Monto, Charlotte Moore, Randolph Nesse, Daniel Povinelli, Bill Shropshire, John Skoyles, Sarita Soni, Thomas Suddendorf, Teresa Tavassoli, Paul and Gilles Tréhin, Alfonso Troisi, Gus Uht, Tony Vladusich, and Andy Wells. I must also thank Lily Morgan of Jessica Kingsley Publishers and an anonymous reviewer for their kind assistance and sound advice on the manuscript.

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.

Chapter 1

Autism and Its Compensations

Once, autism was thought to be a condition with few if any positive aspects, but today a more balanced view has emerged. In this chapter I aim to show that autism is not simply a combination of deficits as was once supposed, but is associated with some remarkable sensory and cognitive compensations. As we shall see, research into autism suggests that human beings have evolved two parallel ways of thinking. One, which you might call *people-thinking, mentalistic cognition*—or more simply *mentalism*—is wholly concerned with understanding human beings, their minds, motives, and emotions; the other, which by contrast you could call *things-thinking* or *mechanistic cognition* is concerned with understanding and interacting with the physical, non-human universe of inert objects. It is to the latter that we owe our technological, scientific, and material mastery of the world, and we shall see that although autistics symptomatically have deficits in mentalistic people-thinking, they are often superior where basic sensory sensitivity is concerned, and can sometimes show extraordinary abilities in mechanistic things-thinking. Furthermore, this way of looking at autism suggests that the exact opposite cognitive configuration—superior mentalistic skills with deficits in basic senses and in mechanistic cognition—could also exist. Later I shall argue that it indeed does in psychotic disorders such as schizophrenia and that what passes for normality is nothing but a more or less stable balance of both tendencies. The fundamental insight is that autism is part of a much bigger picture which includes

both psychosis and sanity, and lies at the heart of human genius, as I shall argue in my conclusion.

But this is to anticipate. First, let me set the scene with a brief historical summary of what we have come to know about autism.

Autism, schizophrenia, and Asperger's syndrome

The word *autism* was originally coined by one of the founding fathers of modern psychiatry, Eugen Bleuler (1857–1939), to describe a style of thinking found in schizophrenia (itself another Bleuler coinage).[1] He derived it from the Greek αυτοσ (autos) meaning "self," and defined it as "detachment from reality, together with the relative and absolute predominance of the inner life."[2] In 1936 the paediatrician, Hans Asperger (1906–1980), gave a lecture at the Vienna University Hospital in which he described the characteristics of "autistic psychopaths," and in 1938 and 1944 published details of more such cases.[3,4] Meanwhile, in 1943 the psychiatrist, Leo Kanner (1896–1981), also published an account of 11 children at Johns Hopkins University Hospital in Baltimore who were suffering from what he called "early infantile autism." He concluded that "Profound aloneness dominates all behaviour" in the autistic child, adding that "We must, then, assume that these children have come into the world with innate inability to form the usual, biologically provided affective contact with people, just as other children come into the world with innate physical or intellectual handicaps."[5a]

It is not known whether Asperger's discovery influenced Kanner or even if he knew of its existence. In any event, Asperger remained largely unknown to the English-speaking world until the 1980s[6] and his name was not so much as mentioned in one of the most well-informed and wide-ranging reviews of the autism literature published in the 1960s.[7] Recently an account of six cases diagnosed in 1926 as various types of "schizoid personality disorder" has been translated from the original German. Some of these subjects were also described as "autistic" by its author and bear striking similarities to the type of high-functioning

autism now associated with Asperger.[8,9] However, even earlier accounts certainly exist.[7a,10] Indeed, there is evidence of a five-year-old autistic boy having been admitted to Bedlam in 1799. The case was described in a textbook of 1809 by John Haslam, the Apothecary of Bethlehem Hospital, and this may well be the earliest description of autism in the psychiatric literature.[11] Nevertheless, authors such as Jane Austen (1775–1817) and Sir Arthur Conan Doyle (1859–1930) appear to have had an implicit awareness of features of autism and to have been able to portray autistic characters in their fictional works quite independently of science and psychiatry[12] (for more on Conan Doyle, see pp. 196–198).

Today autism and schizophrenia are thought to be separate disorders, and Bleuler himself later substituted *dereistic* for *autistic* as a description of schizophrenic thought that was less likely to be misunderstood.[13] However, a major theme of this book will be to argue that there is indeed an important link between them. Later I shall suggest that autism and psychoses such as schizophrenia could be seen as extremes on a single line of development—with normality balanced precariously in between. Just as both over-sensitivity and under-sensitivity to light or sound will cause visual or hearing short-comings, so we shall see that both over- and under-sensitivity to your own and other people's minds can cause the contrasting mental deficits seen in autism and psychosis. If this is so, you might wonder to what extent the early association of the two disorders in psychiatry was an anticipation of what is now beginning to look like a profound connection ultimately explicable in terms of genetic findings that are only now coming to light.

Autism is a disorder that usually first becomes apparent in childhood, mainly as a result of failure to develop normally. A consensus panel of the American Academy of Neurology recommends that a child with any of the following symptoms should be evaluated for possible autism: no babbling, gesturing, pointing, or waving good-bye by 12 months; no single words by 16 months; no two words spoken together spontaneously by 24 months; and any loss of language or social skills at any time.[14] As the last item suggests, children can develop normally

Typical symptoms and signs of autism

- deficits in non-verbal communication such as eye-contact, facial expression, and body language
- self-absorption, egocentricity, and lack of awareness of and insensitivity to others, with difficulty in establishing relationships, friendships, or peer-relations
- delay, or total lack of language competence, with communication deficits or peculiarities in speech, gesture, and conversation
- repetitive and/or stereotyped movements (such as hand-flapping), with distress over change and insistence on routine, or a compulsion to carry out rituals
- fragmented sensory perception with inability to generalize, and pre-occupation with parts rather than wholes
- abnormal pre-occupation with or intensity of interest in one subject or activity, perhaps with isolated areas of expertise and/or exceptional rote memory alongside more general cognitive impairment

Additional features often found associated with autism

- mental retardation (found in about 75% of cases)
- unusual beauty, often looking younger than they are, with a characteristic "autistic look" described by Kanner as "beatific serenity"
- odd or unusual gait
- difficulties with hand-writing
- insensitivity to pain, often combined with indifference to cold (and sometimes lack of fear of heights and an amazing ability to survive falls)

- synaesthesia (mixing of perceptual categories) with confusion between different senses
- problems with depth-perception, "white-out" effects and other visual deficits, particularly in relation to moving objects, strange places, or novel situations
- unusual sensitivity to smell, sound, or other sensory perceptions, sometimes with sensations of "sensory overload"
- allergic or phobic reactions to specific foods, smells, or sensory perceptions, with resulting fastidious food preferences and avoidances
- bowel disorders
- sleep disorders
- epilepsy (25–35% of cases)
- intolerance of itchy and/or tight clothing
- chronic anxiety, often with excessive startle and fear reactions
- fear of crowds and strangers, and dislike of socializing
- panic reactions at being touched or hugged by people
- a liking for being wedged in small, enclosing spaces, or tightly squeezed into corners
- a fascination for spinning objects
- enthralment with machines, mechanisms, and gadgets of all kinds

up to a certain point, and then regress; while others can appear to have early delays in these respects that are later fully compensated and leave no lasting deficits.[15] Typical symptoms and signs of autism are set out in the box above along with a number of other features often mentioned in connection with autism although not found in all cases (for a more exhaustive discussion see [16].)

An important aspect of diagnosis in autism is the extreme variability of the symptoms. Autistic children within the same family can have strikingly different autistic traits,[17] and authorities point out that "None of the criteria exactly describes every individual with autism. Autism presents in a myriad of ways; every individual with autism is different and unique, and has features that would lead a person superficially examining them to say that this person can't have autism."[18a]

Another factor that has bedevilled diagnosis is confusion of autism with schizophrenia and other mental disorders, despite the fact that almost from the beginning some writers could see that they were antithetically different in many respects.[7] What we would now call autism was often given labels such as "schizoid personality disorder" or even "childhood schizophrenia" in the past, and more recently there was also a tendency to diagnose children as autistic but to substitute "schizophrenic," "psychotic," "borderline," or some similar diagnosis for the same symptoms when they grew up. Indeed, I shall argue in a later chapter that even today it is easy to confuse autism with psychotic disorders, and that although the symptoms might seem very similar, closer examination shows them to have completely different causes. Nevertheless, mis-diagnosis—principally of autistics as suffering from various forms of psychotic illness but also psychotics mis-diagnosed as autistics—has been a major factor in confusing autism with psychosis. This has certainly been true in forensic psychiatry where, despite the high numbers of autistics in secure prison hospitals,[18b] autism has tended to be regarded as a much less convincing plea of mitigation in the courts than has schizophrenia. A case in point is that of Theodore J. Kaczynski, otherwise known as the Unabomber, who has recently been diagnosed as a high-functioning autistic, but who was prevailed upon to plead insane on account of schizophrenia at his trial by his lawyers. Kaczynski's autism and associated social isolation resulted in a highly atypical career of lone terrorism which occasioned the longest and most expensive manhunt in the twentieth century, and was only brought to an end when he attempted to communicate his ideas by publishing *The Unabomber Manifesto.*[19] The result is that a classic case of autism has gone down in legal history as one of

schizophrenia, and there is no way of telling how many other similar cases there may be.

Despite the fact that Kanner specifically remarked in his original report that "Even though most of these children were at one time or another looked upon as feebleminded, they are all unquestionably endowed with good *cognitive potentialities*,"[5b] Kanner's name has become associated with a more severe degree of disability. One reason may be that 8 out of 23 cases of autism reported by him in 1946 featured *mutism*: in other words, serious language deficits amounting to an inability to speak.[20] By way of contrast, Asperger's original cases were described as having well-developed speech and even "talking like grown-ups" in early childhood. What is now known as *Asperger's syndrome* shares many of the same central deficits and the restricted, repetitive patterns of behaviour, interest, and activity seen in classical autism, but is distinguished by the absence of delays or deficits in language and of obvious signs of cognitive impairment in childhood. Today about half of all children diagnosed with Asperger's syndrome have relatively advanced verbal skills and are sometimes described as *verbalizers*.[21a] Indeed, a leading clinician comments that from his experience he considers that children and adults with Asperger's syndrome just have a different, and not necessarily defective, way of thinking.[22] Alternatively, Asperger's syndrome is sometimes described as *high-functioning autism*[23]—and this was the actual autistic diagnosis of the Unabomber, mentioned just now.

Estimates of the relative proportion of high- to low-functioning autism vary, but a recent study in the UK concluded that 55 per cent of people with an autistic disorder are low-functioning, and 45 per cent are high-functioning.[24] However, in early childhood the distinction is not always clear-cut. According to the same clinician I have just cited:

> At one point in a child's early development, autism is the correct diagnosis, but a distinct subgroup of children with autism can show a remarkable improvement in language, play and motivation to socialize with their peers between the ages of four and six years. The developmental trajectory of such children

> has changed and their profile of abilities in the primary or elementary school years is consistent with the characteristics of Asperger's syndrome.[21b]

For reasons such as this, there is now a tendency to think of classical, Kanner autism and Asperger's syndrome as the principal examples of *autism spectrum disorder*, or ASD for short (others include Rett syndrome, disintegrative disorder, and pervasive developmental disorder not otherwise specified).*

In the very first case of autism he described, Kanner recounts how the child in question was asked to subtract 4 from 10, and replied: "I'll draw a hexagon!"[5c] Of course, this is the wrong answer, but a hexagon is a six-sided figure, and even being able to make this connection so quickly and spontaneously hints at an unconventional geometric rather than arithmetic way of thinking that might be seen in some ways as more intelligent—and certainly more creative in certain respects—than the "correct" answer. Nor was this an isolated or untypical finding. As early as 1960, American writers on autism were pointing out that the autistic child is "not mentally retarded in the ordinary sense of the word, but rather is a child with an inadequate form of mentation which manifests itself in the inability to handle symbolic forms and assume an abstract attitude."[25] Today some leading authorities are arguing that the perceived association between autism and mental retardation is not based on the fact that they usually have common causes but is more likely to be because the presence of both greatly increases the probability of a clinical diagnosis.[26] Indeed, according to the latest research, intelligence in autistics has generally been under-estimated, and they are not as impaired in fluid intelligence as many theories predict. On the contrary, autistic intelligence is revealed by the most complex single test of general intelligence in the literature: Raven's

---

* For a fictitious, but remarkable insight into the world of a child with Asperger's syndrome see Mark Haddon's novel, *The Curious Incident of the Dog in the Night-time*. However, perhaps the most striking of all depictions of high-functioning autism in literature is found in Albert Camus' classic novel, *L' Étranger*, published in 1942.

Progressive Matrices.[27] Such findings have been interpreted to suggest that Asperger's syndrome in fact involves superior abstract reasoning ability or higher general fluid intelligence.[28]

For reasons which will be discussed in the next chapter, autistic people tend to perform poorly on subtests of intelligence that demand a high degree of communicative competence and/or social intelligence. An example would be comprehension tests, which require an ability to interpret the often implicit meanings, intentions, and understandings conveyed in a passage of writing. However, even where comprehension is concerned, there is a notable exception. A recent study compared autistics with normal subjects on tests of comprehension that involved sentences which demanded both verbal and visualization skills such as *The number eight when rotated 90 degrees looks like a pair of eye-glasses.* As the researchers point out, in sentences such as this the linguistic content must be processed to determine what is to be imagined, and then the mental image must be evaluated and related to the verbal meaning. Normal subjects only used mental visualization when necessary, but autistics were found to use it even when it was not, and the researchers comment that they were probably "thinking in pictures" much of the time. Indeed, the study suggested that as a result autistics might be better at visualizing linguistic information than normal people are.[29]

Autistics are also superior to normal when it comes to copying impossible figures,[30] and the same is true of more straightforward visualizing ability. High-functioning autistics often have a remarkable eye for detail, and notice things that might escape the attention of others. That such impressions are not without an objective basis was recently demonstrated when the vision of a group of people with ASD was compared with that of non-autistic controls. Astonishingly, all 15 of the ASD subjects tested had superior eyesight, which was 2.79 times better than average (giving a score of 20:7, meaning that they could see details from 20 feet that an average person could only see at 7 feet). As the researchers remark, this approximately two-to-three-fold superiority in vision is comparable to that of birds of prey, and their results suggest that increased visual acuity applies to individuals across

the autistic spectrum, making this yet another respect in which autistics outperform the normal population.[31]

Indeed, there is now evidence that autistics may have heightened sensitivities in most senses. For example, despite sometimes giving the impression of being deaf, people with ASD often have superior hearing, as a number of studies have confirmed where discrimination of pitch is concerned.[32,33] A study which explored sensitivity to touch found that people with ASD had a lower threshold for tactile stimulations than normal controls.[34] Another which investigated both touch and hearing in 20 adults with and 20 without ASD matched for sex, age and IQ found that the autistic subjects were hyper-sensitive in both hearing and touch. The two sensitivities appeared to be correlated, suggesting a shared underlying factor (and perhaps explaining autistic symptoms such as intolerance for loud noises and itchy clothing, dislike of being touched, or difficulties with distinguishing individual voices in noisy environments).[35] Another experiment compared 17 ASD subjects with 17 normal controls in a standard test of sensitivity to smell. The autistic group proved able to detect the test odour at a mean distance of 24.1 cm compared to 14.4 cm in the case of the controls. The study also found a quantitative relationship between level of enhanced sensory processing and the number of autistic traits, with greater severity of autistic behaviour related to higher sensory perception. However, there was no correlation between sensory thresholds and age or level of cognitive functioning, suggesting that hyper-sensitivity to smell might be a core feature in ASD.[36] Some parents of autistic children credit them with extra-sensory perception, and these findings suggest that there may indeed be a major core of truth in the claim—at least in so far as those children's sensory sensitivities do in fact go beyond the normal range.[37]

As long ago as the mid-1960s, Rimland remarked that "Judging from his excellent ability to reproduce nursery rhymes and melodies, his memory for spatial relations and his motor performance and finger dexterity, the child with infantile autism has a clear and precise focus on the physical, if not the psychological, aspects of reality."[7b] Summarizing a wide range of studies, a recent review concludes that "The level of