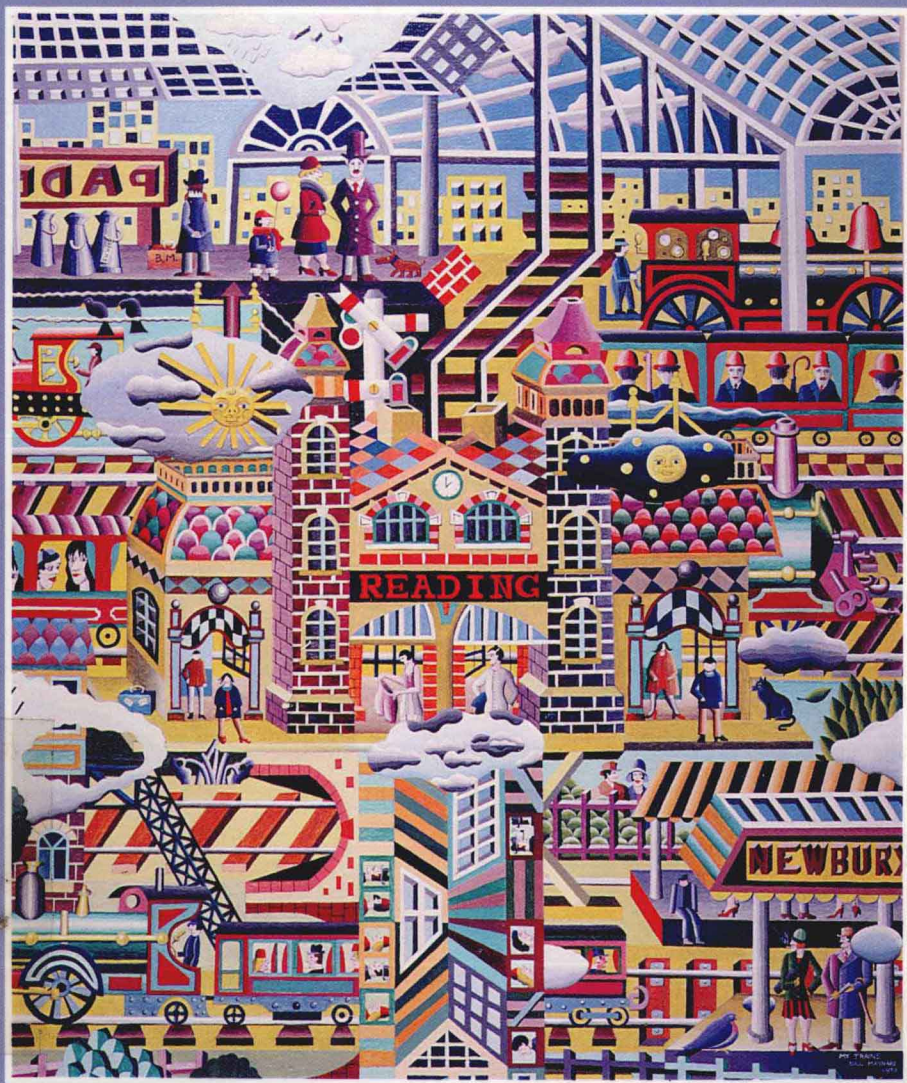


AUTISM

an introduction to psychological theory



Francesca Happé

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Preface

This book is intended as an introduction to current thinking about autism. There are many excellent practical guides to autism for parents and teachers (Wing 1971, Howlin & Rutter 1987, Aarons & Gittens 1991, Baron-Cohen & Bolton 1993). Wonderful and evocative books have been written by the parents of children with autism, which give insight into the everyday life and personal development of the individual (e.g. Park 1987, Hart 1989, McDonnell 1993). Increasingly, able individuals with autism are telling their own stories, a testament to their courage and talents (e.g. Grandin 1984, Grandin & Scariano 1986, Miedzianik 1986, Williams 1992). For those with an interest in theoretical and research issues, there are books which put forward a single author's theory of the condition (e.g. Frith 1989a, Hobson 1993a). There are also weighty collections of chapters by experts, each writing in detail about a particular facet of the disorder (Schopler & Mesibov 1983, 1985, 1987, Cohen et al. 1987, Dawson 1989, Baron-Cohen et al. 1993b).

This book aims to serve a function not intended by any of these books: to give a concise and readable introduction to current research and theory in the field of autism. As far as possible I have tried to give a balanced overview of the field. However, I have also attempted to synthesize and critically assess work in the area – which necessarily introduces my own perspective. I hope that this will encourage readers to think critically and formulate their own research questions and hypotheses.

Although this book is not a practical guide to the care and education of people with autism, I hope that it may be of interest to parents and teachers, who are in many senses the true experts. The primary intended audience, however, is undergraduate and postgraduate students of psychology or related subjects, who – like me – find themselves captivated and mesmerized by the enigma of autism.

FRANCESCA HAPPÉ
MRC Cognitive Development Unit

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Neil O'Connor and Beate Hermelin gave me my first opportunity to meet people with autism, when I was still an undergraduate. Other colleagues should also be thanked, for their intellectual generosity and practical advice; Simon Baron-Cohen, Dermot Bowler, Chris Frith, Peter Hobson, Jim Russell and Marian Sigman. Friends have also helped me to write this book, by discussing ideas, and putting up with my sometimes autistically-narrow interest in this area; Daniel, Liz, Fran, James and Caroline.

Some of the material in this book first appeared in the course handbook for the University of Birmingham's distance learning course on autism. I am grateful to Tina Tilstone and the members of the course's steering committee for their help and advice.

Finally, I would like to thank my family for never failing to give support, enthusiasm and encouragement. A. M. D. G.

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Chapter 1

Introduction

The aim of this book is to acquaint you with current research and thinking about autism, in a concise and comprehensive way. Clearly it cannot be exhaustive in this respect – or it would become like so many “hand-books” which are so large they need two hands to lift! Further reading is suggested in two ways – references in the text will allow you to find out more about specific issues raised, while suggested reading (usually in the form of books or review articles) appears at the end of each chapter, allowing you to deepen your knowledge of those aspects of autism which particularly interest you. Throughout the book the discussion of points has been kept as brief as possible, in the hope that the book will provide a manageable overview of autism, tying together a number of quite different areas. It should whet your appetite for the more detailed consideration of aspects of autism, provided by the suggested readings.

Explaining autism: levels of explanation

If a Martian asked you what an apple is, you might reply that it is a fruit, or that it is something you eat, you might describe it as roundish and red, or you might try to give its composition in terms of vitamins, water, sugars, and so on. The way you answer the question will probably depend on why you think the Martian wants to know – is he hungry, does he want to be able to recognize an apple, or is he simply curious?

Similarly, different types of answer can be given to the question “What is autism?” None of these answers is *the* answer, since each answer is appropriate for a different sense of the question. In order to find the right answer for the question in any one context, we need to think about our reasons for

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asking. One can think about this distinction between the different senses of a question in terms of different *levels of explanation*.

In the study of autism, three levels in particular are useful; the biological, the cognitive, and the behavioural. It is important to keep these levels distinct, because each of the three levels does a different job in our understanding of autism. So, for example, to inform the search for a cure for a disorder, it may be appropriate to look at the biological nature of the problem, while to inform management it may be more important to consider the behavioural description of the problem.

Morton & Frith (1994) have introduced a specific diagrammatic tool for thinking about levels of explanation in developmental disorders such as autism. Figure 1.1, taken from Morton & Frith (1994), shows their causal models of the three levels and the possible relations between these levels, in different types of disorder. Pattern (a) is the case of a disorder defined by its unitary biological origin (O), which may have diverse effects at the cognitive and behavioural levels. An example of this type of disorder might be fragile X syndrome, as currently conceptualized; individuals are said to have fragile X syndrome on the basis of chromosomal analysis of their genetic material. However, not all individuals so defined have the same cognitive or behavioural features: while many will have severe learning difficulties (mental handicap) and show gaze avoidance, others may have normal intelligence and appear socially well adjusted. Pattern (b) shows a disorder with multiple biological causes, and several different behavioural manifestations, but a single defining cognitive deficit (\emptyset). Autism may be one such disorder (see Ch. 5). Dyslexia, according to some cognitive theories (also modelled by Morton & Frith 1994, e.g. Snowling 1987), may be another example; a number of biological causes may converge in causing a cognitive deficit in the phonological system, leading in turn to multiple behavioural manifestations (e.g. slow reading, poor spelling, poor auditory

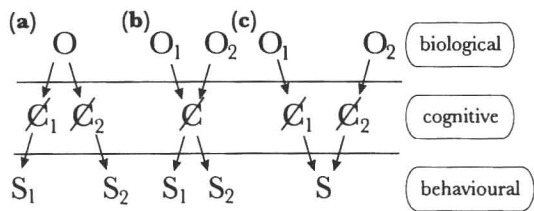


Figure 1.1 Morton & Frith's (1994) causal models of three types of disorder (by kind permission of the authors).

memory, poor rhyme and sound-segmentation skills). Pattern (c) is the case of a disorder defined by its behavioural features (symptoms, S) alone, with multiple biological causes and cognitive natures. Attention deficit disorder, as currently diagnosed, may be such a disorder; children who show extreme distractibility, for whatever reason, may be grouped together under this label for the purposes of treatment and management.

Throughout this book, I will be using the notion of levels of explanation, to keep separate different issues and questions. In Chapter 3, the diagnosis of autism is discussed, and the focus is on the behavioural level – since autism is currently recognized on the basis of behavioural features rather than, for example, biological aetiology. In Chapter 4, the biological level is addressed, since evidence is now overwhelmingly in favour of a biological cause for autism. In Chapters 5 and 6 the remaining of the three levels is discussed – the cognitive level. Cognitive theories aim to span the gulf between biology and behaviour – between the brain and action – through hypotheses about the mind. This level – the level of cognition – is the primary focus for this book. The term “cognitive” as used here is not to be contrasted with affective. Rather, it is intended to cover all aspects of the working of the mind, including thoughts and feelings. This level of analysis might also be called the “psychological” level, except that psychology also includes the study of behaviour.

Keeping the three levels of explanation (biology, cognition, behaviour) distinct helps in thinking about a number of issues to do with autism. So, for example, people often ask whether autism is part of the normal continuum of social behaviour – are we all “a bit autistic”? The answer to this question is different at the different levels of explanation. At the behavioural level the answer may be “yes” – at least in some respects: the person with autism may *behave* much like the very shy normal person in some situations, and everyone shows some stereotypies (e.g. finger-tapping). However, at the biological level people with autism are almost certainly different from people who do not suffer from autism – something in the anatomy or neurophysiology of their brains is responsible for their handicap, and is not present in “normal” people. At the cognitive level too (according to the theory you hold), people with autism may be quite distinct, and not simply at one end of a normal continuum. So, for example, very different *reasons* may underlie apparently similar behaviour by the individual with autism and by the “normal” person – think of a person with autism and a “normal” rebellious teenager, both of whom may dress inappropriately for social situations. So, the autistic child’s social difficulties probably have a quite

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different cause (at the cognitive level) from the “normal” shy person’s – although the behaviours produced (avoiding large groups, social anxiety, inappropriate social behaviour such as odd eye contact) may be very similar.

Questions about the borderlands of autism are particularly relevant when one considers the most high-functioning people with autism. Chapter 7 discusses current research looking at this group, while Chapter 8 introduces the new and increasingly influential diagnosis of “Asperger’s syndrome”, which may be seen as a response to the recent focus on the more able end of the autistic continuum. Chapter 9 considers the borderlands of autism through a discussion of differential diagnosis, and the practical issue of assessing therapies and “cures”. Lastly, Chapter 10 re-examines the question, “Is autism part of the ‘normal’ continuum?”, and looks again at some of the special skills, as well as deficits, which people with autism exhibit.

Explaining autism: timescales of explanation

As well as trying to answer the question “What is autism?”, this book explores why or how autism occurs. In other words, it is concerned with *causal* theories of autism. In thinking about causal explanations it is useful to keep distinct not only three *levels* of description but also three *timescales*. Causes can be examined in terms of *evolutionary* time, taking as the unit for discussion the gene, and considering pressures acting in the process of natural selection. A second timescale of cause is *development*, where the individual (or the biological, behavioural or cognitive mechanism within the individual) is considered. Developmental time includes key features like the existence of critical periods in some systems, where a finite window of time exists for specific causes to have specific effects (e.g. imprinting in the chick) – the same causal agent acting on the organism after this time will not have the same consequences. Lastly, there is the time span of on-line mechanisms, the moment-to-moment or *processing* time.

In considering autism the latter two timescales are particularly important (see, for example, Ch. 6). Two examples may help to clarify the distinction, and to illustrate that the same deficit may have rather different effects in terms of disruptions to development and disruptions to processing.

Think of the effects of large quantities of alcohol acting as a cause on the three timescales. In evolutionary time, imagine that the existence of alcohol in foodstuffs leads to the selection of individuals with the ability to taste this substance and avoid consuming large quantities of foods contain-

ing alcohol – since being drunk does not increase reproductive success! In developmental time, alcohol has different effects – in large quantities it may hamper the physical and mental development of the fetus. Still in developmental terms, intake of large quantities of alcohol may have long-term effects on adults, for example cirrhosis of the liver. In terms of processing time, however, the effects of alcohol are usually pleasant – that’s why we drink it! In large amounts, however, it has effects on processing, for example causing slurring of speech and loss of balance. These are “on-line” effects in the sense that they persist only for so long as the maintaining cause is there – the high blood alcohol level. The developmental effects, however, may persist, even after the individual has sobered up.

Another illustration of the three levels might be the effect of lack of calcium on bone formation. It is currently believed that the level of calcium intake (amongst other factors) affects the strength of bones. However, this statement is true only at the developmental level. Women who drink a lot of milk in their twenties may be less likely to develop brittle bones in their sixties and seventies. However, drinking a glass of milk today will not stop you breaking your leg tomorrow! Calcium does not work on-line to strengthen bones, and there is no instant effect. Similarly, as long as you drank lots of milk as a young woman, you can give the stuff up in your seventies – you are no longer building your bones (the developmental work of calcium is over). In terms of evolutionary time, interestingly, osteoporosis affecting postmenopausal women would probably have no causal effect – natural selection would not act to favour women who have strong bones after childbearing age since this would probably have no advantage in terms of reproductive success.

These examples may seem a long way from autism but, as will emerge in Chapter 6, psychological theories of autism can easily confuse developmental and processing causes.

Some facts and fiction

While the question “What is autism?” can be answered at a number of levels – as will be explored further in Chapters 3, 4, 5 and 6 – there are some statements which can be made concerning what autism is *not*. It may be useful at this early stage to clear away some of the myths and misunderstandings about autism.

Autism is *not* caused by “refrigerator parenting”.

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Autism *is* a biologically based disorder.

Autism is *not* confined to childhood.

Autism *is* a developmental disorder which lasts throughout life.

Autism is *not* always characterized by special, or “savant”, skills.

Autism *is* found at all IQ levels, but is commonly accompanied by general learning difficulties (mental handicap).

Autism is *not* just a “shell” within which a “normal” child is waiting to get out.

Autism *is* a severe disorder of communication, socialization and imagination.

Chapter 2

The history of autism

“He wandered about smiling, making stereotyped movements with his fingers, crossing them about in the air. He shook his head from side to side, whispering or humming the same three-note tune. He spun with great pleasure anything he could seize upon to spin . . . When taken into a room, he completely disregarded the people and instantly went for objects, preferably those that could be spun . . . He angrily shoved away the hand that was in his way or the foot that stepped on one of his blocks . . .” (Kanner 1943; reprinted in Kanner 1973: 3–5)

This description, of a five-year-old boy called Donald, was written over 50 years ago. Kanner saw Donald and made these observations in 1938, and they appear in his landmark paper “Autistic disturbances of affective contact”, published in 1943. Clinicians and teachers today remark on similar features. Autism itself, then, has changed little over the half century since its recognition. But what about the years before 1943? Is autism a new disorder? Probably not. Uta Frith (1989a) has speculated that we can find evidence of autism throughout history. She mentions the “Blessed Fools” of Old Russia, who were revered for their unworldiness. The apparent insensitivity to pain, bizarre behaviour, innocence, and lack of social awareness that these “Blessed Fools” showed, suggest that they may have had autism.

Almost certainly, autism has always existed. Folktales can be found in almost every culture which tell stories of naive or “simple” individuals with odd behaviour and a striking lack of common sense. The following folktales come from two very different cultures, but each centres on naive and over-literal understanding of communication – a very characteristic feature of high-functioning individuals with autism (see Chs 3 and 5). The first tale comes from India:

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One time Sheikh Chilli was hotly in love with a girl, and he said to his mother: "What is the best way of making a girl fond of one?" Said his mother: "The best plan is to sit by the well, and when she comes to draw water, just throw a pebble at her and smile."

The Sheikh went to the well, and when the girl appeared, he flung a big stone at her and broke her head. All the people turned out and were going to murder him, but when he explained matters, they agreed that he was the biggest fool in the world.

(From *Folktales of India*, Kang & Kang 1988)

The second folktale comes from Malta:

In a village there lived a boy called Gahan. It was Sunday and Gahan's mother wanted to go to church early. But Gahan didn't like getting up in the mornings, so he said he would stay in bed. When his mother was ready to go, she came into Gahan's room.

"I'm off to church now," she said. "When you get up, if you decide to come to the church, be sure and pull the door behind you."

"Don't worry, mother," said Gahan, "I won't forget."

After a while Gahan climbed out of bed, washed and dressed and was just about to leave when he remembered what his mother had said. He opened the front door, pulled it down, held it by the knocker and began to pull it along behind him.

. . . You can imagine how all the people laughed when they saw Gahan walking along the street dragging the door behind him. When he arrived at the church he walked straight in. But he made such a banging and clattering noise that everyone turned to see what was happening. They, too, thought that it was very funny, but Gahan's poor mother was very embarrassed.

"What on earth are you doing?" she asked.

"Well, mother," answered Gahan, "you asked me to pull the door behind me if I left the house, didn't you?"

(From *Folktales from Australia's children of the world*, Smith 1979)

These tales suggest that the odd behaviour and naivety of the person with autism have been recognized in many different cultures. It is interesting that the subjects of this sort of folktale are almost always male; autism is more than twice as common among men as among women (see Ch. 4).

Why did it take so long for autism to receive a name? Perhaps because autism is so rare (see Ch. 4). Perhaps because it is often accompanied by general learning difficulties, which have themselves become better under-

stood in this century. Although clinicians before Kanner had described children who we would now diagnose as suffering from autism, it was not until Kanner wrote about a group of 11 children with a puzzling but similar constellation of symptoms, that the syndrome of autism was really recognized. What was "autism" for Kanner?

Leo Kanner's autism

Kanner's first paper on autism highlights a set of features he perceived to be characteristic of all the children he saw. These features included the following:

"Extreme autistic aloneness" – the children failed to relate to people normally, and appeared to be happiest when left alone. This lack of social responsiveness appeared to Kanner to start very early in life, as shown by the autistic infant's failure to put out his arms to the parent who was about to pick him up, or to mould himself to the parent's body when held.

"Anxiously obsessive desire for the preservation of sameness" – the children were extremely upset by changes of routine or surroundings. A different route to school, a rearrangement of furniture, would cause a tantrum, and the child could not be calmed until the familiar order was restored.

"Excellent rote memory" – the children Kanner saw showed an ability to memorize large amounts of effectively meaningless material (e.g. an encyclopaedia index page), which was out of line with their apparent severe learning difficulties or mental handicap in other respects.

"Delayed echolalia" – the children repeated language they heard, but failed to use words to communicate beyond their immediate needs. The echolalia probably explains the reversal of pronouns which Kanner remarked upon – that the children would use "you" when referring to themselves and "I" for the other person. This usage would follow from a direct repetition of the other speaker's remark. In the same way, children with autism commonly use the whole of a question as a request for the item which usually follows (e.g. "Do you want a sweet?" meaning "I want a sweet").

"Oversensitivity to stimuli" – Kanner noticed that many of the children he saw reacted strongly to certain noises and to objects such as vacuum cleaners, elevators and even the wind. Some also showed feeding problems or food fads.

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“Limitation in the variety of spontaneous activity” – shown in the children’s repetitious movements, verbalizations and interests. However, Kanner felt that the children showed a good relation to objects, often showing surprising dexterity in spinning things or completing jigsaw puzzles.

“Good cognitive potentialities” – Kanner believed that the outstanding memory and dexterity shown by some of his cases reflected a superior intelligence, despite the fact that many of the children had been considered to have severe learning difficulties. This strong impression of intelligence – that a child with autism *could* if only they *would* – is often felt by parents and teachers. The good memory in particular is tantalizing – leading one to feel that if only it could be turned to some practical use, the child might learn well. An impression of intelligence is also given by the lack of any physical stigmata in most cases of autism. Unlike children with many types of severe learning difficulties (e.g. Down’s syndrome), children with autism usually look “normal”. Kanner remarked on the “intelligent physiognomies” of his cases, and other authors have described children with autism as unusually beautiful.

“Highly intelligent families” – Kanner remarked that all his cases had intellectual parents. However, this is probably due simply to a referral bias – Kanner’s sample is unlikely to have been representative. Kanner also described the parents as cold, although in his first paper he was very far from a psychogenic theory. Instead he states, “these children have come into the world with innate inability to form the usual, biologically provided affective contact with people”.

In his later writing (Kanner & Eisenberg 1956) Kanner isolated just two of these many features as the key elements of autism: “extreme isolation and the obsessive insistence on the preservation of sameness”. The other symptoms he considered to be either secondary to and caused by these two elements (e.g. communicative impairments), or non-specific to autism (e.g. stereotypies). In Chapter 3, Kanner’s description of autism will be reassessed, and the issue of universality and specificity of symptoms will be discussed. Current diagnostic criteria will also be examined.

Hans Asperger

The history of autism is something like waiting for a bus – nothing for years and then two come along together! In 1944, just one year after Kanner

published his influential paper, an Austrian physician, Hans Asperger, published a dissertation concerning “autistic psychopathy” in childhood. It has taken nearly 50 years for Asperger’s original paper, “Die ‘Autistischen Psychopathen’ im Kindesalter”, to appear in translation in English (Frith 1991b). Hans Asperger deserves credit for some very striking insights into autism: some insights which Kanner (1943) lacked and which it has taken us many years of research to rediscover. Before considering these particular observations of Asperger’s, it is worth noting the many features on which the two physicians agreed.

Kanner’s and Asperger’s descriptions are surprisingly similar in many ways, especially when one remembers that each was unaware of the other’s ground-breaking paper. Their choice of the term “autistic” to label their patients is itself a striking coincidence. This choice reflects their common belief that the child’s social problems were the most important and characteristic feature of the disorder. The term “autistic” comes from Bleuler (1908), who used the word (from the Greek “autos” meaning “self”) to describe the social withdrawal seen in adults with schizophrenia. Both Kanner and Asperger believed the social handicap in autism to be innate (in Kanner’s words) or constitutional (as Asperger put it), and to persist through life into adulthood. In addition, Kanner and Asperger both noted the children’s poor eye contact, their stereotypies of word and movement, and their marked resistance to change. The two authors report the common finding of isolated special interests, often in bizarre and idiosyncratic objects or topics. Both seem to have been struck by the attractive appearance of the children they saw. Kanner and Asperger make a point of distinguishing the disorder they describe from schizophrenia, on the basis of three features: the improvement rather than deterioration in their patients, the absence of hallucinations, and the fact that these children appeared to be abnormal from their earliest years, rather than showing a decline in ability after initially good functioning. Lastly, both Kanner and Asperger believed that they had observed similar traits – of social withdrawal or incompetence, obsessive delight in routine, and the pursuit of special interests to the exclusion of all else – in the parents of many of their patients.

There are three main areas in which Asperger’s and Kanner’s reports disagree, if we believe that they were describing the same sort of child. The first and most striking of these is the child’s *language abilities*. Kanner reported that three of his 11 patients never spoke at all, and that the other children did not use what language they had to communicate: “As far as the communicative functions of speech are concerned, there is no fundamental dif-