Multisensory Development

Developmental disorders and multisensory perception

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Abstract and Keywords

This chapter presents a review of research concerning multisensory processing impairments in three developmental disorders: developmental coordination disorder (DCD), autism spectrum disorder (ASD), and developmental dyslexia (DD). By comparing multisensory processes across these three disorders, a number of similarities in sensory responses were noted (e.g., hypo- and hyper-sensitivity to sensory information, sensorimotor impairments). This chapter discusses whether multisensory processing abnormalities could represent a particular vulnerability or, perhaps more importantly, a particular risk factor in atypical development. Within and across disorders, possible developmental trajectories that may have led to such impairments are examined. Finally, the need for further multisensory research in developmental disorders is highlighted, and avenues for future research explored.

Keywords: developmental coordination disorder, autism spectrum disorder, developmental dyslexia, multisensory, developmental disorders, developmental trajectories, hyposensitivity, hypersensitivity, sensorimotor impairments, multisensory development

12.1 Introduction

Developmental disorders are disorders of the brain and nervous system that have their onset during early development, in contrast to disorders acquired in later life (Tager-Flusberg 1999). This difference in timing of onset has important implications for how we can characterize a disorder and its causal origins; if the

onset of a disorder is early in development, whether it is caused by aberrant genes or environment, the phenotypical outcome will be the result of an interaction between the initial atypical state of the individual and the subsequent ontogenetic developmental process (Bishop 1997; Karmiloff-Smith 1998, 2009). Across the study of developmental disorders, the developmental role of multisensory, and indeed unisensory, perceptual deficits has received only patchy consideration. Developmental dyslexia (DD), for example, was originally characterized as 'word-blindness', a visual perceptual impairment, with little consideration given to the developmental ontogeny of this impairment. Moreover, despite the multisensory basis of reading (in which we have to learn relations between auditory phonology and visual orthography), more recent perceptual accounts of DD have typically not focused on multisensory impairments. Similarly, despite the sensory abnormalities reported by Kanner (1943) and Asperger (1944/1991) in their first case study reports of autism, early formal diagnostic classifications (e.g. Wing and Gould's 1979, 'triad of impairments' in socialization, communication, and imagination), and later DSM classifications (where 'imagination' was replaced with repetitive or stereotyped behaviours) make no reference to perceptual/sensory abnormalities. This is despite the numerous reports of such abnormalities across multiple senses in this disorder (see below).

This less-than-central consideration given to the developmental impact of multisensory perceptual impairments seems somewhat neglectful. Our ability to perceive the environment in a coherent way, which is largely dependent upon an ability to integrate information across multiple sensory channels, is arguably among the first cognitive skills that the human infant seeks to master (e.g. Piaget 1952; see also Chapter 8 by Bahrick and Lickliter; Chapter 5 by Bremner *et al.*; Chapter 7 by Lewkowicz). If such processes are impaired this is likely to have significant downstream developmental effects. Furthermore, multisensory processing abnormalities are implicated across a wide range of developmental disorders and, as we shall discuss later, there also appear to be some surprising symptomatic commonalities between disorders in terms of multisensory processing. This raises the interesting possibility that multisensory processing abnormalities could represent a particular vulnerability or, perhaps more importantly, a particular risk factor in atypical development.

(p.274) 12.2 Why study atypical multisensory development?
12.2.1 The relation between unisensory and multisensory processing impairments in developmental disorders

While the majority of the chapters in this volume examine the typical development of multisensory processes, here we will review the literature pointing to multisensory processing abnormalities in atypically developing individuals. We will consider developmental disorders that are, for the most part, diagnosed on the basis of behavioural characteristics and are characterized by specific cognitive profiles. We will not consider neurological or genetic markers,

or a sensory receptor impairment—for coverage of atypical multisensory processing in individuals with congenital and late blindness see Chapter 13 by Röder. We will focus specifically on three developmental disorders, namely developmental coordination disorder (DCD), autism spectrum disorder (ASD), and developmental dyslexia (DD) in which abnormalities point most clearly towards a potential multisensory impairment. Because other disorders, such as attention deficit hyperactivity disorder (ADHD), are also suggestive of sensory processing impairments, we will refer to these where appropriate throughout.[1]

As already stated, there have been relatively few studies of multisensory impairments in developmental disorders. We believe that there are now a number of converging reasons to pursue the question of multisensory impairment in atypical development more seriously. The broad literature on multisensory perceptual processes (cf. Calvert *et al.* 2004; Spence and Driver 2004; Stein and Meredith 1993) demonstrates quite conclusively that our senses do not function in isolation, either at the level of basic perceptual processes or attention. Consequently, the growing literature investigating sensory processing impairments across various disorders will have to acknowledge, sooner or later, that, even if the ontogeny of a sensory processing problem is a basic unisensory deficit, difficulties with one sense will have important consequences for the way the other senses function, and importantly how multisensory development proceeds. Strong evidence for the developmental impact of a unisensory deficit on the development of multisensory processes can be seen from the literatures investigating sensory deprivation in developing animals (Chapter 14 by Wallace *et al.*) and in human infants and children with sensory loss (Chapter 13 by Röder). Similarly, a deficit with multisensory integration can have consequences for sensory processing when that is measured with respect to a single sensory modality.

12.2.2 A particular vulnerability of multisensory integration in developmental disorders: hyposensitivity, hypersensitivity, and sensorimotor problems

Another important reason to consider atypical multisensory development is that evidence concerning the development of multisensory integration (described throughout this volume) is increasingly indicating that the ability of typically developing individuals to optimally integrate the senses to speed and improve the accuracy of perceptual judgements or responses undergoes significant developments right across infancy, childhood, and into adolescence (e.g. Gori *et al.* 2008; Nardini *et al.* 2008; Neil *et al.* 2006; Tremblay *et al.* 2007). Given the extended development of multisensory integration and the fact that environmental input is considered to be an **(p.275)** important factor in integration (Chapter 14 by Wallace *et al.*; Gori *et al.* 2010), one can postulate that the typical emergence of such integrative processes could be particularly vulnerable to deviations from an atypical developmental trajectory. Indeed, as already stated, there are some striking commonalities across disorders in symptoms that are suggestive of a multisensory deficit. One particular example

of this is poor sensorimotor integration (or motor control), which appears to be apparent in almost all developmental disorders (e.g. DCD, ASD, specific language impairment; see, respectively, Sugden and Chambers 2005; Mari *et al.* 2003; Hill 2001). Sensorimotor control typically requires information about both the body and the relationship between the body and the external environment (Lee 1980). This information is provided via visual, somatosensory, proprioceptive, and even auditory inputs, all of which require integration for optimal performance (Chapter 6 by Nardini and Cowie; Ernst and Bülthoff 2004).

A further set of symptoms observed across disorders that may indicate multisensory deficits comes from reports of hyper- or hyposensitivity to perceptual information arising from one particular channel, such as hypersensitivity to sounds or tactile stimuli at the expense of other modalities (Grandin and Scariano 1986; O'Brien *et al.* 2008; O'Neill and Jones 1997; Williams 1992). As we shall explain later, one possible explanation for hyper- or hyposensitivity to sensory information arriving from a particular modality could be atypical integration of that sense with the others.

We now move on to describe DCD, ASD, and DD. We will cover the literature indicating multisensory processing abnormalities in each of these disorders, and will examine possible developmental trajectories which may have led to such impairments (see Table 12.1 for a summary of studies examining multisensory processing abnormalities in these disorders).

12.3 Developmental coordination disorder

Developmental coordination disorder (DCD) is diagnosed in children who experience movement difficulties out of proportion with their general development and in the absence of any medical condition (such as cerebral palsy) or identifiable neurological disease (American Psychiatric Association 2000, for full diagnostic criteria see Box 12.1). In daily life, this includes gross motor difficulties (e.g. poor posture, problems walking up/down stairs with alternate feet, poor sporting achievements) and fine motor difficulties (e.g. problems with buttoning, tying laces, handwriting, etc). Individuals with DCD can also be hyper- or hyposensitive to noise, touch, taste, temperature, or light (e.g. Dare and Gordon 1970), have difficulty with spatial awareness, display poor body awareness (e.g. Hill and Bishop 1998), and experience socio-emotional problems (e.g. Green *et al.* 2006; Skinner and Piek 2001; Wilson *et al.* 2000).

The general consensus, as well as the bulk of the empirical evidence, supports the view that sensorimotor difficulties are the core deficit in those with DCD. Given that multisensory integration is at the heart of sensorimotor processes (see Chapter 5 by Bremner *et al.* and Chapter 6 by Nardini and Cowie), this disorder is a prime candidate for the influence of multisensory processing abnormalities, with these impacting on, and being impacted by, atypical development from birth.

12.3.1 Multisensory processing abnormalities in DCD

We began this chapter by pointing out that we do not process information from our sensory modalities in isolation. For example, in controlling the movements of our body and limbs we make use of information from a variety of sensory inputs and, in particular, vision and proprioception (our sense of our body's layout with respect to itself arising from receptors in our limbs). Like vision, proprioception is a crucial source of input for efficient movement and thus engagement **(p. 276)**

Table 12.1. Summary of the key studies assessing multisensory integration in groups with DCD, ASD, or DD that are referred to in this chapter. The right hand column indicates whether the DCD, ASD, or DD group were impaired in multisensory integration, relative to any other groups. (VIQ = verbal IQ; PIQ = performance IQ; FSIQ = full scale IQ)

Reference	Diagnosis (ASD, DCD or DD)	Comparison Group	Age (years)	Matching criteria	Multisensory modalities assessed	Group impaired?
Hulme, Smart, and Moran (1982)	DCD	Typical children	11	Chronological age	Visual; Spatial	Yes
Lord and Hulme (1987)	DCD	Typical children	Mean age of both groups = 8.8 (SD = 1.5)	Chronological age, gender, VIQ, reading ability	Visual; Spatial	Yes
Mon-Williams, Wann, and Pascal (1999)	DCD	Typical children	5–7	Chronological age	Visual; Proprioceptive	Yes
Schoemaker *et al.* (2001)	DCD	Typical children	6–12	Chronological age, gender	Visual; Proprioceptive	Yes
Sigmundsson *et al.* (1999)	DCD	Typical children	6–8	Chronological age, gender	Visual; Proprioceptive	Yes
Smyth and Mason (1998)	DCD	Typical children	5–8	Chronological age, gender, IQ	Visual; Proprioceptive	Yes
Bonneh *et al.* (2008)	ASD	N.A. (case study)	13	N.A. (case study)	Visual; Auditory; Tactile	N.A. (case study)

Reference	Diagnosis (ASD, DCD or DD)	Comparison Group	Age (years)	Matching criteria	Multisensory modalities assessed	Group impaired?
Oberman and Ramachandran (2008)	ASD	Typical children	Mean age ASD group = 9.27 (SD = 1.3); Mean age control group = 9.27 (SD = 1.3)	Chronological age, VIQ, Matrices (boys only)	Visual; Auditory	Yes
Foss-Feig *et al.* (2010)	ASD	Typical children	8-17 (Mean age ASD group = 12.6, SD=2.6; Mean age control group = 12.09, SD=2.2)	Chronological age, gender, IQ	Visual; Auditory	Yes
Klin *et al.* (2009)	ASD	Typical infants,	2 (Mean age ASD group = 1.97, SE=.25; Mean age control group = 2.1, SE=.32)	Chronological age	Visual; Auditory	Yes
Smith and Bennetto (2007)	ASD	Typical children	12–19	Chronological age, gender, FSIQ, receptive language	Visual; Auditory	Yes

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Reference	Diagnosis (ASD, DCD or DD)	Comparison Group	Age (years)	Matching criteria	Multisensory modalities assessed	Group impaired?
Van der Smagt *et al.* (2007)	ASD	Typical adults	Mean age ASD group = 20.5 (SD = 3.2); Mean age control group = 20.7 (SD = 2.6)	Chronological age; gender; VIQ; PIQ; FSIQ	Visual; Auditory	No
Williams *et al.* (2004)	ASD	Typical children	5–13	Chronological age, gender	Visual; Auditory	No
Birch and Belmont (1964)	DD	Typical children	9–10	Chronological age (boys only)	Visual; Auditory	Yes
Birch and Belmont (1965)	DD	Typical children	9–10	Chronological age (boys only)	Visual; Auditory	Yes
Blau *et al.* (2009)	DD	Typical adults	Mean age DD group = 23.5 (SD = 3.7); Mean age control group = 26.8 (SD = 5.4)	Chronological age, educational level, handedness, IQ	Visual; Auditory	Yes
Hairston *et al.* (2005)	DD	Typical adults	21–57	Chronological age	Visual; Auditory	Yes

(p.277) (p.278) in activities of daily living, learning, and employment. Both static and dynamic (kinaesthetic) information is provided from the proprioceptors. As such, a number of studies of crossmodal processing of visual and proprioceptive information have been conducted in children with or without DCD.

Box 12.1. A summary of DSM-IV (1994) diagnostic criteria for DCD

Criterion A:
Performance in daily activities that require motor coordination is substantially below that expected given the individual's chronological age and measured intelligence. This may be seen in marked delays in achieving motor milestones (such as walking, crawling, sitting), dropping things, "clumsiness", poor performance in sports, or poor handwriting.

Criterion B:
The disturbance in Criterion A significantly interferes with academic achievement or activities of daily living.

Criterion C:
The difficulties are not due to a general medical condition (such as cerebral palsy or hemiplegia) and the individual does not meet criteria for a Pervasive Developmental Disorder.

Criterion D:
If Mental Retardation is present, the motor difficulties are in excess of those usually associated with it.

N.B. Previously referred to as 'clumsy child syndrome' (Gubbay 1975, American Psychiatric Association 1980); now often referred to as 'dyspraxia'. DCD is estimated to affect 2-5% of individuals (Lingam *et al.* 2009, APA 2000, respectively). Like most developmental disorders, more males are affected than females (5:1, Henderson and Hall 1982).

Difficulties in using visual and proprioceptive inputs concurrently would be expected to have a particular impact on gross motor skills, including balancing, posture maintenance, and locomotion (e.g. walking). To balance and locomote efficiently we must process visual information about the body and external environment, proprioceptive information about limb and body position, and, on the basis of this, initiate an appropriate corrective response (see Chapter 6 by Nardini and Cowie). In addition to guiding proprioception and vision to produce accurate limb movements, the crossmodal calibration and integration of these sources of information is also a critical ingredient in balance and locomotion (Lee and Aronson 1974; Lee and Lishman 1975; Nardini *et al.* 2008).

Difficulties in balance are apparent in a significant proportion of children with DCD, evidenced in performance on standardized motor tests such as the Movement ABC, as well as from the outcomes of cluster analysis studies such as those reported by Dewey and Kaplan (1994), Hoare (1994), and Macnab *et al.* (2001). Visual information is important for accurate, effortless balance, and improvements in balance are observed over typical development. Interestingly, the reduction of sway/falls seen in the swinging room paradigm over time age in typical development suggest that proprioception becomes better able to override the misleading visual information present in that environment. Lee and Aronson (1974) have argued that this is due to the crossmodal tuning of the proprioceptors by vision across development.

(p.279) A small number of papers have considered posture and balance in DCD. All of these highlight important differences in performance compared to typically developing peers. Wann *et al.* (1998), for example, reported that children with DCD swayed more than typically developing peers when standing upright with their eyes open and showed particularly poor balance relative to controls when using proprioception alone. Wann *et al.* (1998) also examined responses to a moving visual environment in a swinging-room procedure (cf. Lee and Aronson 1974). Here, the children with DCD who had balance difficulties demonstrated greater postural responses to the swinging environment than controls. These data indicate that DCD children with balance problems may be over-reliant on visual information relative to their peers, and may also have difficulty in using vision to calibrate proprioceptive control of balance (resulting in the poor balance that Wann *et al.* observed when the DCD children's eyes were closed).

In a relatively recent study, Cherng *et al.* (2007) examined the effect on balance of varying the reliability of the proprioceptive/somatosensory input (this was achieved by varying the compliance of the standing surface). When there was one dominant sensory input (eyes open, or fixed foot support with eyes closed), those with DCD showed no differences in the stability of standing balanced in comparison to their peers, suggesting no significant difficulty in using the senses individually. Group differences were observed, however, when sensory inputs were altered by making the surface on which the children were standing less stable. As a result, Cherng *et al.* (2007) argued that individual sources of sensory input are adequate in DCD, but that children with DCD demonstrate a difficulty in retuning their sensorimotor control to cope with different reliabilities of those sensory signals.

Walking, another sensorimotor task that requires integration of visual and proprioceptive inputs, has also been studied in children with DCD. Deconinck *et al.* (2006) demonstrated that although a group of children with DCD walked at a similar speed as their typical peers in light conditions, they slowed their walking significantly in dark conditions (whereas typically developing children did not).

This again suggests that those with DCD have a stronger reliance than their peers on visual input—in this case for locomotion.

The largest group of studies to have investigated multisensory functioning in DCD have addressed the integration of proprioception and vision in reaching behaviours. They have used a paradigm developed by von Hofsten and Rösblad (1988). In von Hofsten and Rösblad's original study, participants were asked to stick a pin in the underside of the table to match the location of a spot that could be seen or felt on the table-top. This was achieved in three conditions in which available sensory information about the target location was varied:

1. visual—participants viewed the target location via the dot on the table top
2. proprioceptive—participants touched the dot on the table-top with their non-reaching hand with their eyes closed
3. visual and proprioceptive—participants viewed the target location while also touching the dot on the table-top with their eyes open.

Just as for typically developing children, those with DCD made significantly more errors in the proprioceptive-only condition, in line with studies demonstrating that vision provides more reliable information about location (Ernst and Banks 2002). However, in von Hofsten and Rösblad's study, and a number of other investigations (Mon-Williams *et al.* 1999; Schoemaker *et al.* 2001; Sigmundsson *et al.* 1999; Smyth and Mason 1998), those with DCD performed significantly less accurately than their peers across all conditions. Interestingly, however, a study by Mon-Williams *et al.* (1999) indicated that those with DCD experienced a particular difficulty relative to controls in the visual condition. In this condition, the children had to reach to a visual location using proprioceptive guidance alone (i.e. they were forced to match spatial locations across the modalities). **(p. 280)** One possibility is that the children with DCD find this task difficult because they have not appropriately calibrated proprioceptive guidance to a visual spatial frame of reference. Of course, this leaves open the question of whether impaired calibration of proprioception is due to upstream deficits in proprioception, vision, or the processes that allow them to interact. We shall discuss this question in more detail in the next section.

Overall, then, sensorimotor tasks requiring the integration of multisensory inputs (e.g. reaching, balance, and locomotion) constitute areas of difficulty for those with DCD, and this is predominantly the case in difficult or novel situations, including in the absence, or degradation, of particular sensory channels (vision/proprioception).

12.3.2 Unisensory origins of multisensory and sensorimotor impairments?

As we have already argued, reported unisensory impairments can both impact on multisensory functioning and arise as a result of multisensory impairments.

This relationship is likely to give rise to cascading developmental effects on perceptual functioning as sensory impairments have multiple downstream effects in other modalities and across modalities. It is now well accepted, for example, that the absence of a sensory channel will have a downstream influence on multisensory processing (Chapter 13 by Röder). Here, we review the research that has led scientists to argue that the difficulties with sensorimotor abilities (subserved by multisensory processes) observed in DCD are due to a concurrent unisensory impairment. Discussion of the relationship between unisensory impairments and sensorimotor functioning has not typically considered developmental relationships between unisensory and multisensory impairments. We shall argue that this is an important oversight.

One important unisensory account of the difficulties experienced by children with DCD has focused on deficits in proprioceptive/kinaesthetic perception. One paradigm that has typically identified a proprioceptive deficit is a non-visual arm placing task, which requires a participant to actively move their arm to match the posture of the other arm (placed by the experimenter) in a symmetrical arrangement across the body midline (see Fig. 12.1). This task has consistently highlighted substantial difficulties in those with DCD as compared to their peers (Cantell *et al.* 1994; Hill 1997; Pratt 2009; Smyth and Mason 1997).

Others have investigated the view that a kinaesthetic deficit (i.e. a deficit of dynamic proprioception) causes DCD. Using their 'kinaesthetic acuity test', Laszlo and colleagues reported that children with DCD were significantly less accurate in reporting which of their two arms was higher than the other, thus showing poor ability to discriminate unseen limb position following passive movement (Laszlo and Bairstow 1985; Laszlo *et al.* 1988; see Fig. 12.2). This paradigm has the benefit of not requiring a motor response. Thus, any deficit is unlikely to be due to concurrent difficulties with motor control. However, a number of replications using this test have yielded ambiguous results (Hoare and Larkin 1991; Lord and Hulme 1987; Piek and Coleman-Carman 1995).

Perhaps the main difficulty in arguing that the primary deficit in DCD relates to proprioception is that proprioceptive difficulties could be caused by developmentally earlier difficulties in sensorimotor functioning and in multisensory interactions. For instance, as we have already described, it is frequently argued that visual tuning of proprioception is required for proprioceptive sensitivity to develop fully (Lee and Aronson 1974). This interpretation is corroborated by data showing that people who have been blind from birth sway more when standing upright than sighted, but blindfolded individuals (Edwards 1946; see also Gori *et al.* 2010 regarding the visual tuning of haptics). Thus, proprioceptive deficits may well be due to developmentally prior difficulties with vision or with the crossmodal calibration process. **(p.281)**

(p.282) Charles Hulme and colleagues (Hulme *et al.* 1982; Lord and Hulme 1987) have argued that visual perceptual processing plays an important role in the difficulties experienced by those with DCD. They have shown that children with DCD demonstrate deficits in a range of visual tasks, including size constancy, visual length discrimination, and visual area discrimination. It is argued that the visual deficit is primary because deficits in these judgements remain even when children are not required to make a motor response in response to that information. Hulme and his colleagues' position has been further supported by a more recent meta-analysis, which indicates that visuospatial deficits are strongly implicated in DCD (Wilson and McKenzie 1998). Since visual perceptual ability is involved in most sensorimotor skills, dysfunction at this level of the sensorimotor control hierarchy may have a knock-on effect, both in terms of the sensory information available for on-line control and the crossmodal calibrations required for optimal sensorimotor control (see above). Such a difficulty would thus be predicted to have a developmental impact affecting a broad variety of sensorimotor and other areas of development over time (Bishop 1997; Hill 2010; Karmiloff-Smith 1998, 2009; Pratt 2009).
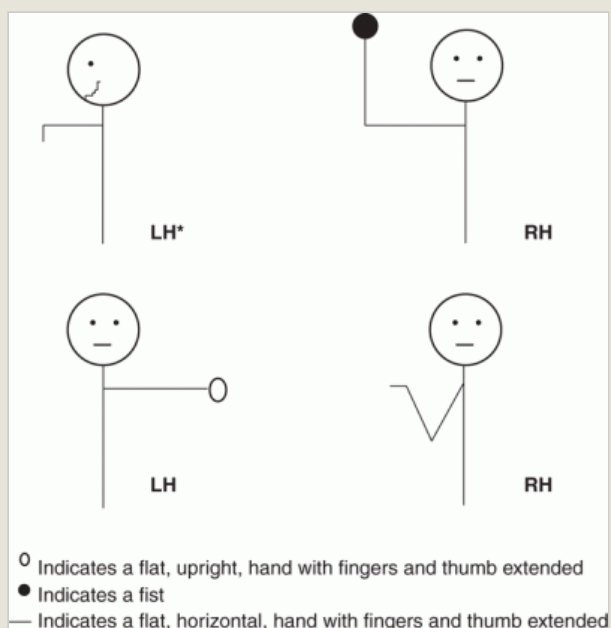
Fig. 12.1 Schematic representation of the positions used in the arm matching task (RH and LH indicate which arm was placed by the examiner). All postures are shown in forwards profile, except those marked with an asterisk, these are shown in sideways profile.
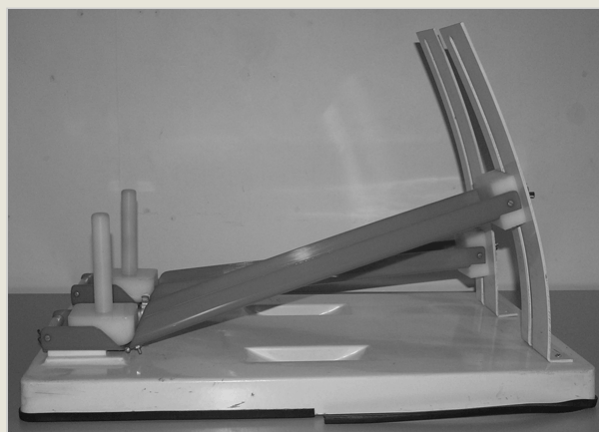
Fig. 12.2 Set-up of the kinaesthetic acuity task (Laszlo & Bairstow, 1985). The child grasps the pegs with his/her hands and must indicate which hand is higher after the hands are moved to the top of the ramps by the experimenter. The

According to this account, a unisensory deficit has an impact on the development of the multisensory processes involved in sensorimotor control. However, once again, there exist some concerns with this account. Not the least of which is that in a number of sensorimotor tasks, children with DCD appear to rely more heavily than their peers on visual input (e.g. Deconinck *et al.* 2006; Wann *et al.* 1998). There also remains the possibility that the visual discriminative impairments observed by Hulme and his colleagues (Hulme *et al.* 1982; Lord and Hulme 1987) may have arisen through impoverished tuning of vision in the context of atypical multisensory interactions earlier in development. Certainly, the atypical motor behaviours observed in DCD could give rise to such a context.

complete set-up (including child's hands) are covered throughout the task.

Thus unisensory accounts of sensorimotor impairments in DCD, be those in terms of a proprioceptive (e.g. Lazlo *et al.* 1988) or a visual deficit (e.g. Lord and Hulme 1987), suffer from a difficulty in tracing whether unisensory deficits themselves have their developmental origins in prior multisensory impairments of sensorimotor control or crossmodal calibration. However, some recent intervention studies have indicated that proprioceptive training can have significant, immediate, and sustained effects on static and dynamic balance tasks as well as manual dexterity and ball skills at a three-month follow-up (Sims *et al.* 1996a; see also Sims *et al.* 1996b; Sims and Morton 1998). This suggests that there may be something to the unisensory kinaesthetic deficit account.

In summary, research on visual and proprioceptive/kinaesthetic perception has highlighted deficits in children with DCD. However, unisensory deficit accounts of DCD suffer from an inability to rule out origins in terms of prior difficulties with sensorimotor control and multisensory integration and calibration. In order to examine the plausibility of such accounts more thoroughly, prospective longitudinal studies investigating the developmental primacy of sensory deficits will be important. Nonetheless, the research reported by Sims and colleagues (Sims *et al.* 1996a; see also Sims *et al.* 1996b; Sims and Morton 1998) highlights the importance of providing specific kinds of proprioceptive experience in ameliorating a wide range of difficulties in DCD.

12.4 Autism spectrum disorder

Autism spectrum disorder (or ASD) is a developmental disorder characterized by impaired social interaction and communication, as well as restricted, repetitive and stereotyped patterns of behaviour, interests, and activities (American Psychiatric Association 2000; World Health Organization 1993, for full diagnostic criteria see Box 12.2)[2]. There are wide variations in the **(p.283) (p.284)** presentation of autism and it is therefore commonly accepted that autism is a spectrum disorder that varies in severity between individuals.

Box 12.2. A summary of DSM-IV (1994) diagnostic criteria for Autistic Disorder and Asperger's Disorder

Autistic Disorder
Criterion A:

Impairments in social interaction; communication; evidence of restricted and repetitive behaviours – example behaviours for each of these include, but are not restricted to:

Social interaction

Lack of peer relationships; lack of spontaneous seeking to share enjoyment, interests etc with others; clear impairment in the use of nonverbal behaviours such as eye-to-eye gaze, facial expression or body postures.

Communication

Delayed development (or lack) of spoken language; difficulty initiating or sustaining conversations; stereotyped and repetitive use of language; lack of spontaneous pretend play.

Restricted, repetitive behaviours

High intensity preoccupation with one or more stereotyped and restricted patterns of interest; inflexible adherence to specific, non-functional routines or rituals; stereotyped and repetitive motor mannerisms (such as hand flapping).

Criterion B:

Onset before 3 years relating to delays or atypical functioning in one or more of: social interaction; language for social communication; symbolic or imaginative play.

Criterion C:

Behaviours above are not explained by Rett's Disorder or Childhood Disintegrative Disorder.

Asperger's Disorder
Criterion A:

Impairments in social interaction; seen for example through lack of peer relationships; lack of spontaneous seeking to share enjoyment, interests etc with others; clear impairment in the use of nonverbal behaviours such as eye-to-eye gaze, facial expression or body postures.

Criterion B:

Restricted repetitive behaviours; seen for example through a high intensity preoccupation with one or more stereotyped and restricted patterns of

interest; inflexible adherence to specific, non-functional routines or rituals; stereotyped and repetitive motor mannerisms (such as hand flapping).

Criterion C:
Behaviours have clinically significant impact in social, occupational, or other key aspects of functioning.

Criterion D:
Language development is not clinically delayed.

Criterion E:
Cognitive development and adaptive behaviours are not clinically delayed.

Criterion F:
Behaviours above are not explained by another specific Pervasive Developmental Disorder or Schizophrenia.

N.B. The autism spectrum can be referred to as autism, autistic disorder, autism spectrum disorder. ASD is estimated to affect 1% of the population (Baird *et al.* 2006). Like most developmental disorders, more males are affected than females. Learning disability (IQ‹70) is estimated to affect 25% of those diagnosed on the autism spectrum (*e.g.*, Friedman-Hill *et al.* 2010).

12.4.1 Hypothesizing 'core' multisensory deficits in ASD

Multisensory processing in ASD has gained interest recently, with a number of researchers suggesting that impairments in multisensory integration might be a core deficit in ASD (Bahrick 2010; Bahrick and Todd 2012; Foxe and Molholm 2009; Oberman and Ramachandran 2008). Foxe and Molholm (2009) have suggested that difficulties in integrating or binding disparate sensory elements (e.g. linking the movement of a bouncing ball with the sound of the ball hitting the ground) could lead to confusion in individuals with ASD, which may result in classic symptoms of the disorder such as social withdrawal. On the other hand, Bahrick and her colleagues (Bahrick 2010; Bahrick and Todd 2012) have put forward some more specific hypotheses regarding the multisensory origins of ASD suggesting that a deficit in sensitivity to intersensory redundancy and, in Bahrick and Todd (in press), a deficit in engaging and disengaging attention to intersensory redundancy in early infancy, could lead to a particular problem in orienting to social stimuli (which Bahrick argues are intrinsically multisensory), thus explaining the later difficulties children with ASD have with social interaction. This account makes use of the intersensory redundancy hypothesis (Chapter 8 by Bahrick and Lickliter), according to which the detection and attention to amodal multisensory information (information that is redundantly presented across more than one modality) is crucial for the typical development of perceptual and cognitive processes.

A rather different multisensory account of ASD has been put forward by Oberman and Ramachandran (2007, 2008). They suggest that an abnormality in the 'mirror neuron system' (Rizzolatti and Craighero 2004) is central to ASD. The mirror neuron system is a brain network **(p.285)** converting sensory stimuli concerning others' actions into similar (mirrored) sensorimotor representations in the observer. This system is believed, by some, to subserve understanding of thoughts, emotions, and actions in others, and to be necessary for the typical development of imitation, theory of mind, language, empathy, and recognition (Oberman and Ramachandran 2007). Interestingly from the point of view of the current review, Oberman and Ramachandran (2007, 2008) hint that a deficit in the mirror neuron system might be related to a multisensory deficit (stating that multisensory systems are 'mirror-neuron like'). Unfortunately this idea is not expanded upon and, crucially, Oberman and Ramachandran do not explain how and why a multisensory integration deficit would imply a deficit in the (sensorimotor) mirror neuron system, or vice versa. Perhaps more damaging for their account, there is now much debate about the existence of a mirror neuron system abnormality in ASD, with some authors demonstrating normal development of imitation in individuals with ASD (see Fan *et al.* 2010; Hamilton 2009; Southgate and Hamilton 2008). Nonetheless, Oberman and Ramachandran (2008) present some data which may be indicative of a multisensory impairment (see below).

One further account, which bears similarities to both of the aforementioned accounts, is offered by Gergely (2001), who argues that ASD represents a deficit in responding to 'imperfect' multisensory contingencies. Gergely (2001) focuses particularly on visual-proprioceptive contingencies, arguing that a typical process of developmental change in attention to such contingencies in infancy does not occur in ASD. Infants who are developing typically undergo a shift in attention from preferring perfect visual-proprioceptive contingencies, which arise when they see their own body moving (see Chapter 5 by Bremner *et al.*; Bahrick and Watson 1985), to preferring imperfect contingencies, which are apparent when another person moves in response to their own movements (Gergely and Watson 1999). This 'contingency switch', is argued to take place in typically developing children at around 3 months of age, and to be an important process underlying the emergence of social-orienting behaviour (see Csibra and Gergely 2011). Gergely (2001) proposes that the switch does not take place in individuals with ASD, explaining the emergence of atypical social orienting in this group.

12.4.2 Evidence of multisensory deficits in ASD

Despite the emergence of these recent theories concerning the multisensory origins of ASD, there is relatively little relevant empirical data so far, and certainly little evidence to distinguish between the accounts described above. Nonetheless, a number of recent studies indicate that multisensory impairments may represent a promising avenue of research in ASD (Bonneh *et al.* 2008; Foss-

Feig *et al.* 2010; Klin *et al.* 2009; Oberman and Ramachandran 2008; Smith and Bennetto 2007; Russo *et al.* 2010). First-hand accounts of individuals with ASD also support the idea of multisensory abnormalities in this group (e.g. O'Neill and Jones 1997). Some have argued that first-hand accounts converge on the opinion that autistic perception is 'monochannel' (Bonneh *et al.* 2008). In other words, in individuals with ASD, it is suggested that attention to one sensory modality can impair perception in another.

Unusual sensory responses suggestive of 'monochannel' perception (hyper- and/ or hyposensitivity to individual sensory channels) have been reported retrospectively by observers from as early as 6–12 months of age (Baranek 1999; Dawson *et al.* 2000; Freeman 1993) and are therefore one of the earliest indicators of the disorder (O'Neill and Jones 1997). These unusual responses appear to persist across the lifespan and have been reported in older children (Kientz and Dunn 1997; Leekam *et al.* 2007), adolescents (Jones *et al.* 2009), and adults (Baron-Cohen *et al.* 2009; Crane *et al.* 2009) with ASD. Such reports have also been confirmed by first-hand accounts of adults with ASD (Grandin and Scariano 1986, Williams 1992). However, unusual sensory processing is not included in the diagnostic criteria for ASD, primarily because similar reports are provided across a **(p.286)** range of developmental disorders (e.g. Miller *et al.* 2001; Rogers *et al.* 2003).[3] We will discuss the similarities in unusual sensory processing in ASD and DCD in the following section, and highlight how this pattern of processing may be indicative of developmental disorder more generally, rather than a specific indicator of a particular disorder. However, as noted above, the single case study reported by Bonneh *et al.* (2008), has examined the multisensory basis of 'mono-channel' perception in an individual with ASD, and we report that here.

Given that descriptions of autistic perception as 'monochannel' could indicate increased crossmodal extinction (or an exaggerated Colavita sensory dominance effect; see Colavita 1974; Koppen and Spence 2007; Sinnett *et al.* 2007), Bonneh *et al.* (2008) investigated this possibility in the context of a case-study of a 13-year-old boy ('AM') with a diagnosis of ASD. In their first study, AM was presented with either unimodal (tactile, visual, or auditory stimuli) or bimodal (visual-auditory, visual-tactile, or auditory-tactile) stimuli and was asked to identify how many and which modalities were presented on each trial. AM demonstrated a striking inability to report bimodal stimuli (in comparison to typical 8-year-olds who performed perfectly). Moreover, the child exhibited a hierarchy of extinction in which visual stimuli extinguished concurrent tactile stimuli, and auditory stimuli extinguished both visual and tactile stimuli. Further studies demonstrated that AM also had difficulty detecting whether bimodal stimuli originated from a single location or two locations (e.g. visual flash to the right and sound to the left), and also that he was unable to respond to a visual colour when an incongruent colour name was presented simultaneously. These

findings are in striking accordance with AM's report that 'when I hear, my vision shuts down' (Bonneh *et al.* 2008, p. 2).

The case study reported by Bonneh *et al.* (2008) is certainly intriguing, and points towards an important avenue of research into multisensory processing in individuals with ASD. However, it is important to recognize the limitations of this case study which demonstrated crossmodal extinction in only one, relatively low-functioning (in terms of daily-life functioning) individual. It will be important to determine whether this kind of crossmodal difficulty is also present in others with ASD. At present, there is some evidence to suggest that this is the case. Van der Smagt *et al.* (2007), and Foss-Feig *et al.* (2010), examined low-level influences of auditory information on visual perception (sounds presented concurrently with visual flashes inducing illusory perceptions of additional flashes, cf. Shams *et al.* 2000). Using this task, Van der Smagt *et al.* (2007) found similar influence of audition on reports of visual percepts between high-functioning adults with and without ASD. However, by varying parameters of the stimuli Foss-Feig *et al.* (2010) found that children with ASD were more likely than typically developing controls to show an influence of an auditory beep on their visual percepts across a wider temporal window.

Researchers have also started to investigate a potential multisensory deficit in language processing in ASD (see also the developmental dyslexia section below). Language processing can be seen as a multisensory integration process, combining auditory and visual information in perceiving speech and making links between audio–visual input, orthography and articulated speech sounds in reading. Here, again, the research has revealed some promising findings, but also a number of conflicting results. Using a speech-in-noise paradigm, which allowed the investigation of audiovisual processing and lip reading in adolescents with and without ASD, Smith and Bennetto (2007) reported that those with ASD were significantly poorer than their peers at lip reading, and gained less benefit from visual information in an audiovisual speech-perception task. They concluded that this performance might reflect a specific deficit in auditory–visual integration.

However, others have failed to find poor performance on similar audiovisual integration tasks. Using speech synthesizer software and a 'virtual' head, Williams *et al.* (2004) presented speech **(p.287)** stimuli to children with and without ASD in unimodal (visual, auditory) and bimodal conditions (requiring multisensory integration). A group difference was only identified in the unimodal conditions. An intervention study, along with computer modelling, led these authors to conclude that children with ASD show normal integration of visual and auditory information.

Working from their premise that the mirror neuron system is impaired in ASD (see above), Oberman and Ramachandran (2008) investigated the 'bouba-kiki effect' (Köhler 1929; Ramachandran and Hubbard 2001). This effect arises when participants are asked to pair nonsense words with shapes. Typical adults (and children) pair these stimuli up with remarkable agreement, suggesting that participants perceive synaesthetic correspondences between the visual and auditory stimuli (see Chapter 10 by Maurer *et al.* for research discussing the role of synaesthesia in development; see also Spence 2011). In contrast, children with ASD perform at almost chance levels on this task. Oberman and Ramachandran (2007, 2008) argue that this arises from poor multisensory integration (which they locate—perhaps controversially—to the mirror neuron system, see above).

Thus while a number of studies are suggestive of a deficit in multisensory integration in ASD (Bonneh *et al.* 2008; Foss-Feig *et al.* 2010; Klin *et al.* 2009; Oberman and Ramachandran 2008; Smith and Bennetto 2007, see Table 12.1), the number of negative results urges caution and further research before accepting a core deficit account of ASD in terms of multisensory processing. As with DCD, studies have yet to clarify the origins of multisensory atypicalities in terms of prior difficulties with unisensory processing, sensorimotor control, and multisensory integration and calibration.

However, at least one study has indicated atypicalities in multisensory processing in infants diagnosed with ASD (Klin *et al.* 2009). In an investigation of 2-year-olds' preferences to attend to biologial motion displays with an accompanying soundtrack, Klin *et al.* found that, whereas typically developing 2-year-olds' visual preferences were driven by the presence of appropriately oriented biological motion in the displays, 90% of the looking in the ASD group of 2-year-olds was driven by the amount of audiovisual (AV) synchrony present in the stimulus events; they preferred to look at AV synchrony. Such early manifestations of atypical multisensory processing in children with ASD indicates that future research into multisensory perception in ASD is certainly warranted. It is important to note, however, that Klin *et al.*'s (2009) data is not entirely consistent with current theories of multisensory disturbances in ASD (particularly those that implicate a difficulty in detection of and attention to intersensory redundancy; Bahrick 2010; Bahrick and Todd in press).

12.5 Links between DCD and ASD: hypersensitivity, hyposensitivity, and atypical multisensory processing

Reports of hyper-/hyposensitivity to individual sensory channels across developmental disorders led to a significant body of occupational therapists and other clinicians adopting a sensory integration approach to the remediation of DCD, ASD, and other disorders. This has stemmed predominantly from the work of Anna Jean Ayres (1979), who described 'sensory integration disorder' (later referred to as sensory processing disorder, SPD), arguing that individuals with

this disorder experience difficulty in dealing with cues from sensory sources and using these to both initiate and control behaviour. On this basis, Ayres and colleagues developed 'sensory integration therapy', which aimed to provide proprioceptive, kinaesthetic, tactile, and vestibular stimulation to those with DCD, ASD, and other disorders, with the aim of improving the sensory and sensory-integration deficits that Ayres and others believed were the cause of these conditions. While this approach remains popular in some areas of the world today, the small number of reports evaluating the success of this intervention approach suggest that it is, at best, no better than other therapeutic interventions (Hoehn and Baumeister 1994; Kaplan *et al.* 1993).

 (p.288) The disappointing results of sensory integration therapy might be partly due to a lack of a systematic study of the nature of the atypical sensory processes underlying hyper- and hyposensitivity to sensory stimulation. Until recently, there have been few attempts to identify what aberrant perceptual process may lead to these symptoms. However, there is the possibility that hypo- or hypersensitivity could reflect a difficulty with multisensory integration.

B.E. Stein *et al.* (2009) recently suggested that symptoms of hyper- and hyposensitivity to unisensory inputs may be due to atypical neural integration of separate sensory channels in the superior colliculus (SC). Drawing from their extensive body of research on the neurophysiological responses of neurons in cat SC (see Chapter 14 for a discussion of how multisensory integration develops in the SC), Stein *et al.* (2009) suggest the development of responses to multisensory inputs in SC as a model for examining sensory integration disorder, but stop short of explaining how an underdeveloped neural response in SC would lead to symptoms such as hyper- or hyposensitivity. One possibility is that a breakdown of multisensory integration may lead to an increased reliance on (or dominance/capture by) one sensory input, and corresponding extinction of responses to the other (see Bonneh *et al.* 2008, described above). It seems logical to predict that an increased reliance or capture by one sense would lead to hypersensitivity, whereas reduced reliance on another sense might lead to hyposensitivity to that modality of stimulation. Certainly, much more research is needed to examine sensory processing abnormalities across disorders. We suggest, specifically, that it will be informative to test whether such a relationship between crossmodal dominance, extinction, and specific patterns of hypo- and hypersensitivity exists in those with developmental disorders. Bonneh *et al.*'s (2008) investigation in an individual with ASD makes a start in this regard.

12.6 Developmental dyslexia
Developmental dyslexia (DD) is diagnosed in those who show a substantial discrepancy between their reading ability and intelligence despite adequate teaching. Individuals diagnosed with DD may experience difficulties such as an inability to learn the alphabet, problems in distinguishing between similar

sounding words, the presence of writing errors (e.g. letter reversal), and very poor spelling (see Box 12.3). DD has also been associated with deficits in oral and written language acquisition (dysphasia and dysgraphia, respectively), mathematics, visuospatial ability, **(p.289)** motor coordination, and attention (Habib 2000). Genetic effects are moderately important in DD (DeFries and Alarcon 1996) although environmental effects, such as orthography (the fact that different languages have different writing systems, some of which are more regular than others; e.g. Greek versus English) are also important (Furnes and Samuelsson 2010; Paulesu *et al.* 2001).

Box 12.3. A summary of DSM-IV (1994) diagnostic criteria for DD

Criterion A:
Reading achievement, as measured by individually administered standardized tests of reading accuracy or comprehension, is substantially below that expected given the individual's chronological age, measured intelligence and age-appropriate education.

Criterion B:
The disturbance in Criterion A significantly interferes with academic achievement or activities of daily living that require reading skills.

Criterion C:
If a sensory deficit is present, the reading difficulties are in excess of those usually associated with it.

N.B. DD is diagnosed in 3-6% of the population (Rutter *et al.* 2004), is found across cultures (Paulesu *et al. 2001)* and persists into adulthood (Rutter *et al.* 2006).

12.6.1 Multisensory processing difficulties in DD

Reading, the core deficit in DD, requires us to make rapid temporal parsings between (visual) written letters and speech sounds. The multisensory nature of this task prompted a number of early researchers of reading disorders to hypothesize and examine visual–auditory integration deficits in children with reading difficulties (e.g. Birch and Belmont 1964; Critchley 1970). For instance, Birch and Belmont (1964, 1965) reported that children with reading difficulties were impaired at identifying crossmodal equivalence between auditory tap patterns and visual patterns of dots in a line. They concluded that deficits in detecting equivalences between hearing and vision contribute to reading difficulties.

However, later researchers noticed some important problems with Birch and Belmont's test of auditory–visual equivalence detection (Tallal and Stark 1982; Zurif and Carson 1970). Primarily, it is possible that the children with reading difficulties were impaired on Birch and Belmont's task, not because of a difficulty with noticing equivalences across the modalities, but rather due to a difficulty in perceiving temporal patterns within one or other modality or across both modalities. Later studies have been equivocal in terms of supporting Birch and Belmont's (1965) interpretation. Snowling (1980) examined dyslexic children's ability to make grapheme–phoneme correspondences in the context of a recognition memory task involving pseudowords. She observed that, compared to reading-age-matched controls, children with DD showed deficits in recognizing across, but not within, modalities. On the other hand, Zurif and Carson (1970) found that children with dyslexia were impaired at matching temporal patterns both crossmodally and within modalities, and concluded that perceptual deficits in dyslexia could also be due to an amodal difficulty with temporal information. Temporal processing accounts of DD (e.g. J. Stein 2001; Tallal 1980; Temple 2002) have since become some of the more influential explanations of the disorder. We shall return to discuss these accounts in the next section.

In one more recent study addressing the relationship between visual and auditory temporal processing in DD, Hairston and colleagues (2005) have identified differences in multisensory interactions in processing temporal information between adults with DD and controls. Using a visual 'temporal order judgement' (TOJ) task in which participants were asked to judge which of two visual stimuli had appeared first on a screen (either above or below a fixation cross), Hairston *et al.* (2005) first examined the participants' threshold stimulus onset asynchrony for this task. Consistent with arguments for a temporal processing deficit (J. Stein 2001; Tallal 1980; Temple 2002), participants with DD had a much higher threshold for discriminating the temporal order of the stimuli (i.e. they required longer temporal gaps between the stimuli in order to respond appropriately to their temporal order). In a separate condition, the authors presented an interfering auditory stimulus (which was not spatially informative). When this was presented close enough in time to the visual stimuli, all participants showed an improvement in their TOJs with respect to the visual stimuli. This benefit is thought to be provided by a multisensory 'ventriloquism' of one of the visual cues to the auditory stimulus (Morein-Zamir *et al.* 2003). Hairston *et al.*'s (2005) key finding was that the facilitation conferred by the auditory stimulus was greater in the group with DD than in the control group. In addition, the temporal window over which this facilitation occurred was greater for the group with DD. Hairston *et al.* (2005) concluded that adults with DD have an expanded temporal window of multisensory integration for auditory and visual information. They suggest that this expanded temporal window for processing multisensory **(p.290)** information may lead to an increased number

of multisensory binding errors. More specifically, they argue that an increase in the time taken to link visual and auditory information together (e.g. when linking visual representations of orthography with corresponding auditory information during reading) may lead to a higher number of reading errors, as well as a general slowing in reading ability, in those with DD.

Thus, a few studies have indicated that multisensory impairments exist in individuals with DD. However, the argument for multisensory integration being an important contribution to reading disability (Birch and Belmont 1965) suffers from a paucity of empirical support, and also a lack of consistency among the findings of studies of multisensory processing in DD. Whilst Snowling (1980) has demonstrated, and Birch and Belmont (1965) have argued, that individuals with DD have a particular difficulty in making links between visual and auditory stimuli, more recent findings by Hairston and colleagues (2005) suggest that individuals with DD actually integrate auditory–visual information more (or across a longer time-span) than controls. While it is possible to see how both of these multisensory processing differences could lead to reading deficits, they do not offer a consistent explanation of DD in terms of a core multisensory deficit. Next we discuss the potential relationships between more accepted 'core deficit' accounts of DD and multisensory functioning.

12.6.2 Other accounts of developmental dyslexia and their developmental relationship to multisensory impairments

Over the last three decades there has been a great deal of debate as to what represents the 'core deficit' in DD. Some theories argue that the disorder represents a specific phonological deficit (e.g. Pennington *et al.* 1991), and others place it within a more broad perceptual and/or sensorimotor domain. John Stein (2001), for instance, suggests that DD is primarily due to difficulties in processing rapidly occurring sensory stimuli, irrespective of modality. Meanwhile, Nicholson and Fawcett (1990) have argued that sensorimotor difficulties substantiated in the cerebellum are at the heart of the disorder.

The magnocellular deficit theory of dyslexia (J. Stein 2001; J. Stein and Walsh 1997) was proposed as an explanation for reports of poor visual and auditory temporal processing in those with dyslexia. It has been argued that a unifying explanation of this performance profile relates to a specific biological atypicality in the magnocellular neurons in the sensory areas of the central nervous system (as distinct from parvocellular neurons). In the visual system, magnocellular neurons are particularly involved in the perception of visual movement, depth, and small differences in brightness (low-contrast black and white information). Although magnocellular neurons do not form a distinct pathway in the auditory system (as they do in the visual system), J. Stein (2001) also argues that a magnocellular deficit can explain auditory processing difficulties reported in some individuals with DD, including difficulties with rapid auditory temporal processing (Tallal and Piercy 1973) and auditory frequency discrimination (e.g.

Ahissar *et al.* 2000; McAnally and Stein 1996). It is claimed that this view explains additional differences in visual processing, binocular control, vergence, visual crowding, and visuospatial attention reported in groups of individuals with DD (e.g. Hari *et al.* 2001; Spinelli *et al.* 2002; J. Stein 2001; J. Stein and Fowler 1993; J. Stein and Walsh 1997). The deficits entailed by the magnocellular theory of DD could certainly go some way to explaining difficulties with the multisensory task of reading, as reading requires us to make rapid temporal translations between visual orthography and auditory phonology. A magnocellular impairment could also underlie some of the multisensory deficits described in the previous section. For instance, less rapid temporal processing of stimuli (auditory or visual) could result in multisensory integration of auditory and visual information occurring across a wider temporal window (Hairston *et al.* 2005).

(p.291) However, while there is some neurophysiological evidence to support the magnocellular deficit theory of DD in the form of structural and functional brain abnormalities (Eden *et al.* 1996; Livingstone *et al.* 1991), there is now a growing body of evidence against this view, perhaps most critical being those studies that show that whilst magnocellular system deficits are more prevalent in individuals with DD than the general population, such deficits are not nearly as consistent as phonological deficits (Ramus *et al.* 2003a,b).

An additional theory of DD, the 'automatization', or 'cerebellar deficit', theory was proposed by Nicolson and Fawcett (1990) following their observation that individuals with dyslexia experienced difficulty in dual tasks involving motor skills, such as balancing and counting at the same time. In this account, reading deficits are attributed to 'an inability to become completely fluent in cognitive and motor skills' (Fawcett and Nicolson 1992, p. 507), with the cerebellum and associated systems being specifically implicated. Further work reported by Nicolson, Fawcett, and colleagues has documented a broad range of motor deficits in children and adults with dyslexia (e.g. Fawcett *et al.* 1996; Stoodley *et al.* 2005, 2006), as well as suggesting atypicalities in the cerebellum in adults with dyslexia (Laycock *et al.* 2008). However, again this theory suffers from the limitation that motor difficulties only affect a small subset of individuals with DD (e.g. Ramus *et al.* 2003a,b).

It is a little difficult to see how the cerebellar deficit theory could explain multisensory abnormalities either specific to reading abilities or more generally. Indeed, there appears to be little evidence for a causal link between motor impairments and reading difficulties (Ramus *et al.* 2003b). However, an alternative possibility is that motor impairments and reading difficulties may arise as a result of a more general multisensory vulnerability. As we saw earlier, multisensory processes are at the heart of sensorimotor control and may play an important role in other disorders with a motor component, such as DCD. Indeed, Ramus *et al.* (2003b) make a parallel argument to this, suggesting that DD may

represent a general sensorimotor vulnerability, which manifests itself in different ways across the population of affected individuals.

However, the most accepted account of DD is the one that argues that phonological processing difficulties constitute the 'core deficit' of the disorder. According to this account, it is poor detection and/or discrimination of speech sounds that leads to the difficulties observed in dyslexia. These difficulties have a striking impact on reading and writing since these depend on good phonological processing (Muter *et al.* 2004), and the greatest functional impact is seen in those whose language has a greater degree of irregular orthography (conversion from sounds to the written word). However, even those whose native language has a regular orthography show a 'dyslexic' pattern of brain activation in phonological processing tasks (e.g. Paulesu *et al.* 2001). Furthermore, pre-readers at risk for dyslexia show poor phonological learning and awareness skills (Carroll and Snowling 2004) such as slow learning of rhymes.

While there is no doubt that more general sensory processing abnormalities and sensorimotor problems are evident in at least a subgroup of those with DD, the bulk of the evidence supports the view that a phonological deficit is sufficient to cause DD, while additional sensory and sensorimotor difficulties are seen in some individuals (Ramus *et al.* 2003a,b).

A recent neuroimaging study by Blau *et al.* (2009) has attempted to elucidate the relationship between the phonological deficits observed in DD and the multisensory impairments in making links between phonology and orthography when reading. Blau *et al.* (2009) used fMRI to examine brain activation in adults with DD in response to speech sounds, visual letters, and multisensory speech–letter combinations (congruent and incongruent). Replicating previous findings, they found that adults with DD showed underactivation (relative to controls) of the left superior temporal cortex in response to speech sounds. However, they also found an absence of an audiovisual congruency effect in the superior temporal cortex, such that, in contrast to controls, no difference **(p.292)** was observed between blood-oxygen-level-dependence responses to congruent and incongruent speech–letter combinations. The authors conclude that there is an observable neural impairment in audiovisual multisensory integration in adults with DD, and furthermore demonstrate a strong relationship between the multisensory impairment and the simple auditory processing impairment that was also observed. Whilst it is difficult to identify a direction of causality, there seems to be a strong relationship between phonological and multisensory impairments in adults with DD. Further developmental research will elucidate how these impairments arise and whether one or the other is the 'core' deficit.

While these findings suggest that examining a multisensory involvement in DD offers a promising avenue of research, there is much work to be done in determining the origin of multisensory impairments (in behaviour and the brain)

and DD. One particularly important question concerns whether atypical multisensory processing in DD is the cause or result of reading/phonological abnormalities. The majority of studies of multisensory processing in DD have involved adults, so we have a poor picture of the developmental relationship between multisensory processes and the symptoms of DD. Further research on multisensory integration in children with DD and pre-reading children with DD will help clarify whether multisensory integration is a central deficit in DD.

12.7 Conclusion

The study of atypical development is not only useful in terms of the treatment of developmental disorders, but because it can also provide a point of comparison from which to investigate the processes involved in the emergence of certain skills and behaviours in typical development. Furthermore, it can also help us to explain individual differences in the population (Elsabbagh and Johnson 2010). As we pointed out at the start of this chapter, it is surprising to note how little research has examined sensory and multisensory impairments in developmental disorders. The three disorders covered in this chapter—DCD, ASD, and DD— have all been proposed at one point or another to involve atypical sensory processing. Indeed, clinicians would, for the most part, argue strongly that sensory difficulties were central in at least the first two of the three disorders considered (i.e. DCD and ASD).

In conclusion, this review raises a number of points regarding our understanding of sensory processing in developmental disorders. Not least, it shows that there are some distinct similarities in sensory responses that exist across disorders. Hypo- and hypersensitivity to sensory information is found in ASD, DCD, ADHD, and Williams syndrome, among others. Such responses may be indicative of disordered multisensory integration (resulting in dominance or extinction of responses to particular sensory channels). Sensorimotor impairment has been identified across a similarly large range of developmental disorders (including all of those reported here). As we have argued earlier, much research on DCD points to an interpretation of sensorimotor impairments in terms of atypical interactions between the sensory modalities used to guide a range of different actions.

Thus it is our contention that further multisensory research in developmental disorders is much warranted. A large body of literature (see the other chapters in this volume) now indicates that multisensory processes have an extended development through infancy and well into childhood. Such abilities, if perturbed at an early stage in development, could lead to significant downstream impairments, not just in multisensory processes, but also in unisensory and cognitive abilities. It is certainly a limitation that most studies of sensory processing difficulties in children with developmental disorders examine processing in one sense at a time. Because unisensory processing is influenced by multisensory processes and vice versa, developmental disorders **(p.293)**

research requires studies to trace the trajectories of emergence of problems in processing the senses on their own and in combination. Such studies, we hope, will provide answers not just to the question of how sensory impairments emerge in atypical development, but also help us understand the ontogeny of multisensory perception more generally.

Acknowledgements

References

Bibliography references:

Ahissar, M., Protopapas, A., Reid, M., and Merzenich, M.M. (2000). Auditory processing parallels reading abilities in adults. *Proceedings of the National Academy of Sciences USA,* 97, 6832–37.

American Psychiatric Association (1980). *Diagnostic and statistical manual of mental disorders, 3rd edn (DSM-III)*. Washington, DC.

American Psychiatric Association (2000). *Diagnostic and statistical manual of mental disorders, text revision (DSM-IV-TR)*. Washingon, DC.

Ashwin, E., Ashwin, C., Tavassoli, T., Chakrabarti, B., and Baron-Cohen, S. (2009). Eagle-eyed visual acuity in autism. *Biological Psychiatry,* 66, e23–24.

Asperger, H. (1944/1991). 'Autistic psychopathy' in childhood. In *Autism and Asperger syndrome* (ed. U. Frith)*,* pp. 37–92. Cambridge University Press, Cambridge.

Ayres, A.J. (1979). *Sensory integration and the child*. Western Psychological Services, Los Angeles.

Bahrick, L.E. (2010). Intermodal perception and selective attention to intersensory redundancy: Implications for typical social developmental and autism. In *The Wiley-Blackwell handbook of infant development, 2 nd edn.* (eds. J.G. Bremner, and T.D. Wachs), pp. 120–66*.* Wiley-Blackwell, Oxford, UK.

Bahrick, L.E., and Watson, J.S. (1985). Detection of intermodal proprioceptive-visual contingency as a potential basis of self-perception in infancy. *Developmental Psychology,* 21, 963–73.

Bahrick, L.E., and Todd, J.T. (2012). Multisensory processing in autism spectrum disorders: Intersensory processing disturbance as a basis for atypical development. In *The new handbook of multisensory processes* (ed. B.E. Stein). MIT Press, Cambridge, MA.

Bahrick, L.E., Lickliter, R., Castellanos, I., and Vaillant-Molina, M. (2010). Increasing task difficulty enhances effects of intersensory redundancy: testing a new prediction of the Intersensory Redundancy Hypothesis. *Developmental Science,* 13, 731–37.

Baird, G., Simonoff, E., Pickles, A., Chandler, S., Loucas, T., Meldrum, D., and T.C. (2006). Prevalence of disorders of the autism spectrum in a population cohort of children in South Thames: The Special Needs and Autism Project (SNAP). *Lancet,* 368, 179–81.

Baranek, G.T. (1999). Autism during infancy: a retrospective video analysis of sensory-motor and social behaviors at 9–12 months of age. *Journal of Autism and Developmental Disorders,* 29, 213–24.

Baranek, G.T., David, F.J., Poe, M.D., Stone, W.L., and Watson, L.R. (2006). Sensory experiences questionnaire: discriminating sensory features in young children with autism, developmental delays, and typical development. *Journal of Child Psychology and Psychiatry,* 47, 591–601.

Baron-Cohen, S., Ashwin, E., Ashwin, C., Tavassoli, T., and Chakrabati, B. (2009). Talent in autism: hyper-systemizing, hyper-attention to detail and sensory hypersensitivity. *Philosophical Transactions of the Royal Society B: Biological Sciences,* 364, 1377–83.

Birch, H.G., and Belmont, L. (1964). Auditory visual integration in normal and retarded readers. *Annals of Dyslexia,* 15, 48–96.

Birch, H.G., and Belmont, L. (1965). Auditory-visual integration, intelligence and reading ability in school children. *Perceptual and Motor Skills,* 20, 295–305.

 (p.294) Bishop, D.V.M. (1997). *Uncommon understanding: development and disorders of language comprehension in children*. Psychology Press, Hove.

Blakemore, S.-J., Tavassoli, T., Calò, S., *et al.* (2006). Tactile sensitivity in Asperger syndrome. *Brain and Cognition,* 61, 5–13.

Blau, V., van Atteveldt, N., Ekkebus, M., Goebel, R., and Blomert, L. (2009). Reduced neural integration of letters and speech sounds links phonological and reading deficits in adult dyslexia. *Current Biology,* 19, 503–508.

Bonneh, Y.S., Belmonte, M.K., Pei, F., *et al*. (2008). Cross-modal extinction in a boy with severely autistic behaviour and high verbal intelligence. *Cognitive Neuropsychology,* 25, 635–52.

Calvert, G., Spence, C., and Stein, B.E. (2004). *The handbook of multisensory processes*. MIT Press, Cambridge, MA.

Cantell, M.H., Smyth, M.M., and Ahonen, T.K. (1994). Clumsiness in adolescence: educational, motor and social outcomes of motor delay detected at five years. *Adapted Physical Activity Quarterly,* 11, 115–29.

Carroll, J.M., and Snowling, M.J. (2004). Language and phonological skills in children at high risk of reading difficulties. *Journal of Child Psychology and Psychiatry,* 45, 631–40.

Cherng, R.J., Hsu, Y.W., Chen, Y.J., and Chen, J.Y. (2007). Standing balance of children with developmental coordination disorder under altered sensory conditions. *Human Movement Science,* 26, 13–26.

Colavita, F.B. (1974). Insular-temporal lesions and vibrotactile temporal pattern discrimination in cats. *Physiological Behaviour,* 12, 215–218.

Crane, L., Goddard, L., and Pring, L. (2009). Sensory processing in adults with autism spectrum disorders. *Autism: The International Journal of Research and Practice,* 13, 215–28.

Critchley, M. (1970). *The dyslexic child*. Heinemann, London.

Csibra, G., and Gergely, G. (2011). Natural pedagogy as evolutionary adaptation. *Philosophical Transactions of the Royal Society B,* 366, 1149–57.

Dare, M.T., and Gordon, N. (1970). Clumsy children: a disorder of perception and movement organisation. *Developmental Medicine and Child Neurology,* 12, 178–85.

Dawson, G., and Watling, R. (2000). Interventions to facilitate auditory, visual, and motor integration in autism: A review of the evidence. *Journal of Autism and Developmental Disorders,* 30, 415–21.

Dawson, G., Osterling, J., Meltzoff, A.N., and Kuhl, P. (2000). Case study of the development of an infant with autism from birth to 2 years of age. *Journal of Applied Developmental Psychology,* 21, 299–313.

Deconinck, F.J.A., De Clercq, D., Savelsbergh, G.J.P., *et al*. (2006). Visual contribution to walking in children with developmental coordination disorder. *Child: Care, Health and Development,* 32, 711–22.

DeFries, J.C., and Alarcon, M. (1996). Genetics of specific reading disability, *Mental Retardation and Developmental Disabilities Research Reviews,* 2, 39–47.

Dewey, D., and Kaplan, B.J. (1994). Subtyping of developmental motor deficits. *Developmental Neuropsychology,* 10, 265–84.

Dunn, W. (1999). *Sensory profile: user's manual .* The Psychological Corporation, San Antonio.

Dunn, W., and Bennett, D. (2002). Patterns of sensory processing in children with attention deficit hyperactivity disorder. *OTJR-Occupation Participation and Health,* 22, 4–15.

Eden, G.F., VanMeter, J.W., Rumsey, J., Maisog, J.M., Woods, R.P., and Zeffiro, T.A. (1996). Abnormal processing of visual motion in dyslexia revealed by functional brain imaging. *Nature,* 382, 66–69.

Edwards, A. (1946). Body sway and vision. *Journal of Experimental Psychology,* 36, 526–35.

Elsabbagh, M., and Johnson, M.H. (2010). Getting answers from babies about autism. *Trends in Cognitive Sciences,* 14, 81–87.

Ernst, M.O., and Banks, M.S. (2002). Humans integrate visual and haptic information in a statistically optimal fashion. *Nature,* 415, 429–33.

Ernst, M.O., and Bülthoff, H.H. (2004). Merging the senses into a robust percept. *Trends in Cognitive Sciences,* 8, 162–69.

 (p.295) Fan, Y.T., Decety, J., Yang, C.Y., Liu, J.L., and Cheng, Y. (2010). Unbroken mirror neurons in autism spectrum disorders. *Journal of Child Psychology and Psychiatry,* 51, 981–88.

Fawcett, A.J., and Nicolson, R.I. (1992). Automatisation deficits in balance for dyslexic children. *Perceptual Motor Skills,* 75, 507–29.

Fawcett, A.J., Nicolson, R.I., and Dean, P. (1996). Developmental dyslexia: the cerebellar deficit hypothesis. *Trends in Neurosciences,* 24, 508–511.

Filipek, P.A., Accardo, P.J., Baranek, G.T., *et al.* (1999). The screening and diagnosis of autistic spectrum disorders. *Journal of Autism and Developmental Disorders,* 29, 439–84.

Foss-Feig, J.H., Kwakye, L.D., Cascio, C.J., *et al.* (2010). An extended multisensory temporal binding window in autism spectrum disorders. *Experimental Brain Research,* 203, 381–89.

Foxe, J.J., and Molholm, S. (2009). Ten years at the Multisensory Forum: musings on the evolution of a field. *Brain Topography,* 21, 149–54.

Foxe, J.J., and Schroeder, C.E. (2005). The case for feedforward multisensory convergence during early cortical processing. *Neuroreport,* 16, 419–23.

Freeman, B. (1993). The syndrome of autism: update and guidelines for diagnosis. *Infants and Young Children,* 6, 1–11.

Friedman-Hill, S.R., Wagman, M.R., Gex, S.E., Pine, D.S., Leibenluft, E., and Ungerleider, L. G. (2010). What does distractibility in ADHD reveal about mechanisms for top-down attentional control? *Cognition,* 115, 93–103.

Frith, C. (2004). Is autism a disconnection disorder? *The Lancet Neurology,* 3, 577.

Furnes, B., and Samuelsson, S. (2010). Predicting reading and spelling difficulties in transparent and opaque orthographies: a comparison between Scandinavian and US/Australian children. *Dyslexia,* 16, 119–42.

Gergely, G. (2001). The object of desire: 'Nearly, but clearly not, like me': contingency preference in normal children versus children with autism. *Bulletin of the Menninger Clinic,* 65, 411–26.

Gergely, G., and Watson, J.S. (1999). Early socio-emotional development: contingency perception and social-biofeedback model. In *Early social cognition: Understanding others in the first months of life* (ed. P. Rochat), pp. 101–36. Lawrence Erlbaum Associates, Hillsdale, NJ.

Ghazanfar, A.A., and Schroeder, C.E. (2006). Is neocortex essentially multisensory? *Trends in Cognitive Sciences,* 10, 278–85.

Gordon, N. (1982). The problems of the clumsy child. *Health Visitor,* 55, 54–57.

Gori, M., Del Viva, M., Sandini, G., and Burr, D. (2008). Young children do not integrate visual and haptic information. *Current Biology,* 18, 694–98.

Gori, M., Sandini, G., Martinoli, C., and Burr, D. (2010). Poor haptic orientation discrimination in non-sighted children may reflect disruption of cross-sensory calibration. *Current Biology*, 20, 223–25.

Gothelf, D., Farber, N., Raveh, E., Apter, A., and Attias, J. (2006). Hyperacusis in Williams syndrome: characteristics and associated neuroaudiologic abnormalities. *Neurology,* 66, 390–95.

Grandin, T., and Scariano, M. (1986). *Emergence: Labeled Autistic*. Arena Press, Novato, CA.

Green, D., Baird, G., and Sugden, D. (2006). A pilot study of psychopathology in developmental coordination disorder. *Child: Care, Health and Development,* 32, 741–50.

Gubbay, S. (1975). *The clumsy child—a study of developmental apraxia and agnosic ataxia*. W.B. Saunders, London.

Habib, M. (2000). The neurological basis of developmental dyslexia: an overview and working hypothesis. *Brain,* 123, 2373–99.

Hairston, W.D., Burdette, J.H., Flowers, D.L., Wood, F.B., and Wallace, M.T. (2005). Altered temporal profile of visual-auditory multisensory interactions in dyslexia. *Experimental Brain Research,* 166, 474–80.

Hamilton, A. F. (2009). Goals, intentions and mental states: challenges for theories of autism. *Journal of Child Psychology and Psychiatry,* 50, 881–92.

Happé, F., and Frith, U. (1994). Autism: Beyond theory of mind. *Cognition,* 50, 115–32.

 (p.296) Happé, F., and Frith, U. (2006). The weak coherence account: detail-focused cognitive style in autism spectrum disorders. *Journal of Autism and Developmental Disorders,* 36, 5–25.

Hari, R., Renvall, H., and Tanskanen, T. (2001). Left minineglect in dyslexic adults. *Brain,* 124, 1373–80.

Heath, S.M., Bishop, D.V.M., Hogben, J.H., and Roach, N.W. (2006). Psychophysical indices of perceptual functioning in dyslexia: a psychometric analysis. *Cognitive Neuropsychology,* 23, 905–29.

Henderson, S., and Hall, D. (1982). Concomitants of clumsiness in young school-children. *Developmental Medicine and Child Neurology,* 24, 448–60.

Hill, E.L. (1997). *An investigation of the motor deficits in developmental coordination disorder and specific language impairment*. Unpublished thesis (PhD), University of Cambridge.

Hill, E.L. (2001). The nonspecific nature of specific language impairment: a review of the literature with regard to concomitant motor impairments. *International Journal of Language and Communication Disorders,* 36, 149–71.

Hill, E.L. (2010). Motor difficulties in specific language impairment: evidence for the Iverson account? – a commentary on Iverson's 'Developing language in a developing body: The relationship between motor development and language development'. *Journal of Child Language,* 37, 229-61.

Hill, E.L., and Bishop, D.V.M. (1998). A reaching test reveals weak hand preference in specific language impairment and developmental coordination disorder. *Laterality,* 3, 295–310.

Hill, E.L., and Frith, U. (2003). Understanding autism: insights from mind and brain. *Philosophical Transactions of the Royal Society B: Biological Sciences,* 358, 281–89.

Hoare, D. (1994). Subtypes of developmental coordination disorder. *Adapted Physical Activity Quarterly,* 11, 158–69.

Hoare, D., and Larkin, D. (1991). Kinaesthetic abilities of clumsy children. *Developmental Medicine and Child Neurology,* 33, 671–78.

Hobson, J. A., and Minshew, N. (2008). Sensory sensitivities and performance on sensory perceptual tasks in high-functioning individuals with autism. *Journal of Autism and Developmental Disorders,* 38, 1485–98.

Hoehn, T.P., and Baumeister, A.A. (1994). A critique of the application of sensory integration therapy to children with learning disablities. *Journal of Learning Disabilities,* 27, 338–50.

Hulme, C., Smart, A., and Moran, G. (1982). Visual perceptual deficits in clumsy children. *Neuropsychologia,* 20, 475–81.

Johnson, M. H. (2005). *Developmental Cognitive Neuroscience .* Wiley-Blackwell, Oxford.

Jones, C., Happé, F., Baird, G., *et al.* (2009). Auditory discrimination and auditory sensory behaviours in autism spectrum disorders. *Neuropsychologia,* 47, 2850–58.

Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child,* 2, 217–50.

Kaplan, B.J., Polatajko, H.J., Wilson, B.N., and Faris, P.D. (1993). Reexamination of sensory integration treatment. *Journal of Learning Disabilities,* 26, 342–47.

Karmiloff-Smith, A. (1998). Development itself is the key to understanding developmental disorders. *Trends in Cognitive Sciences,* 2, 389–98.

Karmiloff-Smith, A. (2009). Nativism versus neuroconstructivism: Rethinking the study of developmental disorders. *Developmental Psychology,* 45, 56–63.

Kientz, M.A., and Dunn, W. (1997). A comparison of the performance of children with and without autism on the sensory profile. *American Journal of Occupational Therapy,* 51, 530–37.

Klin, A., Lin, D.J., Gorrindo, P., Ramsay, G., and Jones, W. (2009). Two-year-olds with autism orient to non-social contingencies rather than biological motion. *Nature,* 459, 257–61.

Köhler, W. (1929). *Gestalt psychology.* Liveright, New York.

Koppen, C., and Spence, C. (2007). Audiovisual asynchrony modulates the Colavita visual dominance effect. *Brain Research,* 1186, 224–32.

Kronbichler, M., Hutzler, F., and Wimmer, H. (2002). Dyslexia: verbal impairments in the absence of magnocellular impairments. *Neuroreport,* 13, 617–20.

 (p.297) Laszlo, J.I., and Bairstow, P.J. (1985). *Test of kinaesthetic sensitivity .* Holt, Rinehart and Winston Ltd., Eastbourne.

Laszlo, J.I., Bairstow, P.J., Bartrip, J., and Rolfe, U.T. (1988). Clumsiness or perceptuo-motor dysfunction? In *Cognition and action in skilled behaviour* (eds. A. Colley and J. Beech), pp. 293–309. Elsevier Science Publishers B.V., North Holland.

Laufer, Y., Ashkenazi, T., and Josman, N. (2008). The effects of a concurrent cognitive task on the postural control of young children with and without developmental coordination disorder. *Gait and Posture,* 27, 347–51.

Laycock, S.K., Wilkinson, I.D., Wallis, L.I., *et al.* (2008). Cerebellar volume and cerebellar metabolic characteristics in adults with dyslexia. *Annals of the New York Academy of Sciences,* 1145, 222–36.

Lee, D.N. (1980). The optic flow field: the foundation of vision. *Philosophical Transactions of the Royal Society London B,* 290, 169–79.

Lee, D.N., and Aronson, E. (1974). Visual proprioceptive control of standing in human infants. *Perception and Psychophysics,* 15, 529–32.

Lee, D.N., and Lishman, J.R. (1975). Visual proprioceptive control of stance. *Journal of Human Movement Studies,* 1, 87–95.

Leekam, S.R., Nieto, C., Libby, S.J., Wing, L., and Gould, J. (2007). Describing the sensory abnormalities of children and adults with autism. *Journal of Autism and Developmental Disorders,* 37, 894–910.

Lingam, R., Hunt, L., Golding, J., Jongmans, M., and Emond, A. (2009). Prevalence of developmental coordination disorder using the DSM-IV at 7 years of age: a UK population-based study. *Pediatrics,* 123, e693–e700.

Livingstone, M.S., Rosen, G.D., Drislane, F.W., and Galaburda, A.M. (1991). Physiological and anatomical evidence for a magnocellular defect in developmental dyslexia. *Proceedings of the National Academy of Sciences USA,* 88, 7943–47.

Lord, R., and Hulme, C. (1987). Kinaesthetic sensitivity in normal and clumsy children. *Developmental Medicine and Child Neurology,* 29, 720–25.

Macnab, J.J., Miller, L.T., and Polatajko, H.J. (2001). The search for subtypes of DCD: Is cluster analysis the answer? *Human Movement Science,* 20, 49–72.

Mari, M., Castiello, U., Marks, D., Marraffa, C., and Prior, M. (2003). The reach-to-grasp movement in children with autism spectrum disorder. *Philosophical Transactions of the Royal Society Series B,* 358, 393–404.

McAnally, K.I., and Stein, J.F. (1996). Auditory temporal coding in dyslexia. *Proceedings of the National Academy of Sciences USA,* 263, 961–65.

Miller, L J., Reisman, J.E., McIntosh, D.N., and Simon, J. (2001). An ecological model of sensory modulation: performance of children with fragile X syndrome, autistic disorder, attention-deficit/hyperactivity disorder, and sensory modulation dysfunction. In *Understanding the nature of sensory integration with diverse populations* (eds. S. Smith-Roley, E. Blanche, and R. Schaaf), pp. 57–88. Therapy Skill Builders, San Antonio, TX.

Milne, E., Swettenham, J., Hansen, P., Campbell, R., Jeffries, H., and Plaisted, K. (2002). High motion coherence thresholds in children with autism. *Journal of Child Psychology and Psychiatry,* 43, 255–63.

Mon-Williams, M., Wann, J.P., and Pascal, E. (1999). The integrity of visual-proprioceptive mapping in developmental coordination disorder. *Developmental Medicine and Child Neurology,* 41, 247–54.

Morein-Zamir, S., Soto-Faraco, S., and Kingstone, A. (2003). Auditory capture of vision: examining temporal ventriloquism. *Brain Research: Cogition Brain Research,* 17, 154–63.

Mottron, L., Dawson, M., Soulières, I., Hubert, B., and Burack, J. (2006). Enhanced perceptual functioning in autism: an update, and eight principles of autistic perception. *Journal of Autism and Developmental Disorders,* 36, 27–43.

Muter, V., Hulme, C., Snowling, M.J., and Stevenson, J. (2004). Phonemes, rimes, vocabulary, and grammatical skills as foundations of early reading development: evidence from a longitudinal study. *Developmental Psychology,* 40, 665–81.

Nardini, M., Jones, P., Bedford, R., and Braddick, O. (2008). Development of cue integration in human navigation. *Current Biology,* 18, 689–93.

(p.298) Neil, P.A., Chee-Ruiter, C., Scheier, C., Lewkowicz, D.J., and Shimojo, S. (2006). Development of multisensory spatial integration and perception in humans. *Developmental Science,* 9, 454–64.

Nicolson, R.I., and Fawcett, A.J. (1990). Automaticity: a new framework for dyslexia research? *Cognition,* 30, 159–82.

Oberman, L.M., and Ramachandran, V.S. (2007). The simulating social mind: the role of the mirror neuron system and simulation in the social and communicative deficits of autism spectrum disorders. *Psychological Bulletin,* 133, 310–27.

Oberman, L.S., and Ramachandran, V.S. (2008). Preliminary evidence for deficits in multisensory integration in autism spectrum disorders: the mirror neuron hypothesis. *Social Neuroscience,* 3, 348–55.

O'Brien, J., Tsermentseli, S., Cummins, O., Happé, F., Heaton, P., and Spencer, J. (2009). Discriminating children with autism from children with learning difficulties with an adaptation of the short sensory profile. *Early Child Development and Care,* 179, 383–94.

O'Neill, M., and Jones, R.S.P. (1997). Sensory-perceptual abnormalities in autism: a case for more research? *Journal of Autism and Developmental Disorders,* 27, 283–93.

Ornitz, E.M. (1989). Autism at the interface between sensory and information processing. In *Autism: nature, diagnosis and treatment* (ed. G. Dawson)*,* pp. 174–207. The Guilford Press, New York.

Paulesu, E., Demonet, J.F., Fazio, F., *et al.* (2001). Dyslexia: cultural diversity and biological unity. *Science,* 291, 2165–67.

Pennington, B.F., Gilger, J.W., Pauls, D., Smith, S.A., Smith, S.D., and DeFries, J.C. (1991). Evidence for major gene transmission of developmental dyslexia. *Journal of the American Medical Association,* 266, 1527–34.

Piaget, J. (1952). *The origins of intelligence in children .* International University Press, New York.

Piek, J., and Coleman-Carman, R. (1995) Kinaesthetic sensitivity and motor performance of children with developmental coordination disorder. *Developmental Medicine and Child Neurology,* 37, 976–84.

Pratt, M.L. (2009). *Profiling patterns of movement disturbance and their relationship to cognition and emotional wellbeing in two developmental disorders.* Upublished thesis (PhD)., University of London.

Raberger, T., and Wimmer, H. (2003). On the automaticity/cerebellar deficit hypothesis of dyslexia: balancing and continuous rapid naming in dyslexic and ADHD children. *Neuropsychologia,* 41, 1493–97.

Rajendran, G., and Mitchell, P. (2007). Cognitive theories of autism. *Developmental Review,* 27, 224–60.

Ramachandran, V.S., and Hubbard, E.M., (2001). Synaesthesia: a window into perception, thought and language. *Journal of Consciousness Studies*, 8, 3–34.

Ramus, F., and Szenkovits, G. (2008). What phonological deficit? *Quarterly Journal of Experimental Psychology,* 61, 129–41.

Ramus, F., Pidgeon, E., and Frith, U. (2003a). The relationship between motor control and phonology in dyslexic children. *Journal of Child Psychology and Psychiatry,* 44, 712–22.

Ramus, F., Rosen, G.D., Dakin, S.C., *et al.* (2003b). Theories of developmental dyslexia: Insights from a multiple case study of dyslexic adults. *Brain,* 126, 841–65.

Rizzolatti G., and Craighero, L. (2004). The mirror neuron system. *Annual Review of Neuroscience,* 27, 169–92.

Rochelle, K.S., and Talcott, J.B. (2006). Impaired balance in developmental dyslexia? A meta-analysis of the contending evidence. *Journal of Child Psychology and Psychiatry,* 47, 1159–66.

Rochelle, K.S., Witton, C., and Talcott, J.B. (2009). Symptoms of hyperactivity and inattention can mediate deficits of postural stability in developmental dyslexia. *Experimental Brain Research,* 192, 627–33.

Rogers, S.J., Hepburn, S., and Wehner, E. (2003). Parent reports of sensory symptoms in toddlers with autism and those with other developmental disorders. *Journal of Autism and Developmental Disorders,* 33, 631–42.

Rutter, M., Caspi, A., Fergusson, D., *et al.* (2004). Sex differences in developmental reading disability: new findings from 4 epidemiological studies. *Journal of the American Medical Association,* 291, 2007–2012.

Rutter, M., Kim-Cohen, J., and Maughan, B. (2006). Continuities and discontinuities in psychopathology between childhood and adult life. *Journal of Child Psychology and Psychiatry,* 47, 276–95.

 (p.299) Russo, N., Foxe, J.J., Brandwein, A.B., Altschuler, T., Gomes, H., and Molholm, S. (2010). Multisensory processing in children with autism: high-

density electrical mapping of auditory-somatosensory integration. *Autism Research,* 3, 253–67.

Schoemaker, M.M., van der Wees, M., Flapper, B., Verheij-Jansen, N., Scholten-Jaegers, S., and Geuze, R.H. (2001). Perceptual skills of children with developmental coordination disorder. *Human Movement Science,* 20, 111–33.

Shams, L., Kamitani, Y., and Shimojo, S. (2000). Illusions. What you see is what you hear. *Nature*, 408, 788.

Sigmundsson, H., Whiting, H.T.A., and Ingvaldsen, R.P. (1999). Proximal versus distal control in proprioceptively guided movements of motor-impaired children. *Behavioral Brain Research,* 106, 47–54.

Sims, K., and Morton, J. (1998). Modelling the training effects of kinaesthetic acuity measurement in children. *Journal of Child Psychology and Psychiatry,* 39, 731–46.

Sims, K., Henderson, S., Hulme, C., and Morton, J. (1996a). The remediation of clumsiness. I: An evaluation of Laszlo's kinaesthetic approach. *Developmental Medicine and Child Neurology,* 38, 976–87.

Sims, K., Henderson, S.E., Morton, J., and Hulme, C. (1996b). The remediation of clumsiness. II: Is kinaesthesis the answer? *Developmental Medicine and Child Neurology,* 38, 989–97.

Sinnett, S., Spence, C., and Soto-Faraco, S. (2007). Visual dominance and attention: the Colavita effect revisited. *Perception & Psychophysics,* 69, 673–86.

Skinner, R.A., and Piek, J.P. (2001). Psychosocial implications of poor motor coordination in children and adolescents. *Human Movement Science,* 20, 73–94.

Smith, E.G., and Bennetto, L. (2007). Audiovisual speech integration and lipreading in autism. *Journal of Child Psychology and Psychiatry* 48, 813–21.

Smyth, M.M., and Mason, U.C. (1997). Planning and execution of action in children with and without developmental coordination disorder. *Journal of Child Psychology and Psychiatry,* 8, 1023–37.

Smyth, M.M., and Mason, U.C. (1998). Direction of response in aiming to visual and proprioceptive targets in children with and without developmental coordination disorder. *Human Movement Science,* 17, 515–39.

Snowling, M.J. (1980). The development of grapheme-phoneme correspondence in normal and dyslexic readers. *Journal of Experimental Child Psychology*, 29, 294–305.

Southgate, V., and Hamilton, A.F. (2008). Unbroken mirrors: challenging a theory of autism. *Trends in Cognitive Sciences,* 12, 225–29.

Spence, C. (2011). Crossmodal correspondences: A tutorial review. *Attention, Perception, and Psychophysics*, 73, 971–95.

Spence, C., and Driver, J. (2004). *Crossmodal space and crossmodal attention*. Oxford University Press, Oxford.

Spinelli, D., De Luca, M., Judica, A., and Zoccolotti, P. (2002). Crowding effects on word identification in developmental dyslexia. *Cortex,* 38, 179–200.

Stein, J.F. (2001). The magnocellar theory of developmental dyslexia. *Dyslexia,* 7, 12–36.

Stein, J.F., and Fowler, M.S. (1993). Unstable binocular control in children with specific reading retardation. *Journal of Research in Reading,* 16, 30–45.

Stein, B.E., and Meredith, M.A. (1993). *The merging of the senses*. MIT Press, Cambridge, MA.

Stein, J.F., and Walsh, V. (1997). To see but not to read; the magnocellular theory of dyslexia. *Trends in Neurosciences,* 20, 147–52.

Stein, B.E., Perrault, T.J., Jr., Stanford, T.R., and Rowland, B.A. (2009). Postnatal experiences influence how the brain integrates information from different senses. *Frontiers in Integrative Neuroscience*, 3, 21.

Stoodley, C.J., Fawcett, A.J., Nicolson, R.I., and Stein, J.F. (2005). Impaired balancing ability in dyslexic children. *Experimental Brain Research,* 167, 370–80.

Stoodley, C.J., Fawcett, A.J., Nicolson, R.I., and Stein, J.F. (2006). Balancing and pointing tasks in dyslexic and control adults. *Dyslexia,* 12, 276–88.

Sugden, D., and Chambers, M., eds. (2005). *Children with developmental coordination disorder .* Whurr Publishers, London.

 (p.300) Tager-Flusberg, H. (1999). An introduction to research on neurodevelopmental disorders from a cognitive neuroscience perspective. In *Neurodevelopmental disorders* (ed. H. Tager-Flusberg), pp. 3–24. MIT Press, Cambridge, MA.

Tallal, P. (1980). Auditory temporal perception, phonics, and reading disabilities in children. *Brain and Language,* 9, 182–98.

Tallal, P., and Piercy, M. (1973). Developmental aphasia: Impaired rate of non-verbal processing as a function of sensory modality. *Neuropsychologia,* 11, 389–98.

Tallal, P., and Stark, R.E. (1982). Perceptual/motor profiles of reading impaired children with or without concomitant oral language deficits. *Annals of Dyslexia,* 32, 163–76.

Tecchio, F., Benassi, F., Zappasodi, F., *et al.* (2003). Auditory sensory processing in autism: a magnetocephalographic study. *Biological Psychiatry,* 54, 647–54.

Temple, E., Poldrack, R.A., Protopapas, A., *et al.* (2002). Disruption of the neural response to rapid acoustic stimuli in dyslexia: evidence from functional MRI. *Proceedings of the National Academy of Sciences,* 97, 13907–13912.

Tremblay, C., Champoux, F., Voss, P., Bacon, B.A., Lepore, F., and Théoret, H. (2007). Speech and non-speech audio-visual illusions: a developmental study. *PLoS One,* 2, e742.

van der Leij, A., and van Daal, V.H. (1999). Automatization aspects of dyslexia: speed limitations in word identification, sensitivity to increasing task demands, and orthographic compensation. *Journal of Learning Disabilities,* 32, 417–28.

van der Smagt, M.J., van Engeland, H., and Kemner, C. (2007). Brief report: Can you see what is not there? Low-level auditory-visual integration in autism spectrum disorder. *Journal of Autism and Developmental Disorders,* 37, 2014–2019.

von Hofsten, C., and Rösblad, B. (1988). The integration of sensory information in the development of precise manual pointing. *Neuropsychologia,* 26, 805–21.

Wann, J. P., Mon-Williams, M., and Rushton, K. (1998). Postural control and coordination disorders: the swinging room revisited. *Human Movement Science,* 17, 491–513.

White, S., O'Reilly, H., and Frith, U. (2009). Big heads, small details and autism. *Neuropsychologia,* 47, 1274–81.

Williams, D. (1992). *Nobody nowhere: the remarkable autobiography of an autistic girl*. Jessica Kingsley Publishers, London.

Williams, J.H., Massaro, D.W., Peel, N.J., Bosseler, A., and Suddendorf, T. (2004). Visual-auditory integration dring speech imitation in autism. *Research in Developmental Disabilities,* 25, 559–75.

Wilson, B.N., Kaplan, B.J., Crawford, S., Campbell, A., and Dewey, D. (2000). Reliability and validity of a parent questionnaire on childhood motor skills. *The American Journal of Occupational Therapy,* 54, 484–93.

Wilson, P.H., and McKenzie, B.E. (1998). Information processing deficits associated with developmental coordination disorder: a meta-analysis of research findings. *Journal of Child Psychology and Psychiatry,* 39, 829–40.

Wimmer, H., Mayringer, H., and Raberger, T. (1999). Reading and dual-task balancing: evidence against the automatization deficit explanation of developmental dyslexia. *Journal of Learning Disabilities,* 32, 473–78.

Wing, L. (1996). *The autistic spectrum: a guide for parents and professionals*. Constable and Robinson, London.

Wing, L., and Gould, J. (1979). Severe impairments of social interaction and associated abnormalities in children: epidemiology and classification. *Journal of Autism and Developmental Disorders,* 9, 11–29.

World Health Organisation (1993). *The ICD-10 classification for mental and behavioural disorders: Diagnostic criteria for research*. World Health Organisation, Geneva.

Yap, R.L., and van der Leij, A. (1994). Testing the automatization deficit hypothesis of dyslexia via a dual-task paradigm. *Journal of Learning Disabilities,* 27, 660–65.

Zurif, E.B., and Carson, G. (1970). Dyslexia in relation to cerebral dominance and temporal analysis. *Neuropsychologia*, 8, 351–61.

Notes:

([1]) We have not devoted an entire section to ADHD as it is as yet unclear whether reported atypical responses to sensory information arise from sensory processing abnormalities per se or from difficulties with the top-down control processes implicated in the hyperactive and inattentive difficulties found in this disorder (Friedman-Hill *et al.* 2010).

([2]) Note changes will be seen in the diagnosis of ASD in DSM-V (2013).

([3]) The importance of sensory symptoms in ASD will be recognised in DSM-V (2013).

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