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Face Perception

Tracey Ward[1] and Raphael Bernier[2]
[1]Simons Autism Family Collaboration University of Washington, Autism Research Center, Seattle, WA, USA
[2]Psychiatry and Behavioral Sciences, University of Washington, Seattle, WA, USA

Definition

Facial perception refers to the ability to rapidly recognize and understand information from faces. The ability to perceive faces and to use that information to guide and direct behavior plays a critical role in interpreting and forming representations from the social world and in the acquisition and understanding of reciprocal social interaction. Early facial recognition research suggests that humans innately attend to faces and are able to rapidly recognize faces remarkably early in life (Goren, Sarty, & Wu, 1975). Within 9 min after birth, infants turn their head to a 90° angle to preferentially attend to faces or face-like images. At 42 min after birth, infants can imitate facial expressions; and by 2 days old, infants can recognize the face of their mother. One-month-old infants show greater pupillary dilation to faces than to nonsocial stimuli suggesting greater interest and arousal in infants when looking at faces. By the end of the first year of life, infants are able to respond to directional gaze, emotional expression, and facial gestures. These early infant preferential face-directed behaviors mark the beginning of a highly protracted development in which repertoires for the face processing system become more sophisticated over time. The examination of facial perception in typical and atypical development, such as in autism spectrum disorders (ASD), has elucidated face perception processes across behavioral, anatomical, and neurophysiological domains and revealed differences in functioning in individuals with ASD and typical development in face perception.

Historical Background

Research with children with ASD has consistently revealed deficits in facial perception surfacing within the first 3 years of life. Children with ASD show impairments in social behaviors related to the perception and processing of information from faces. These impairments include reduced eye contact, joint attention, and social orienting, deficits in the imitation of faces and in face recognition, and attenuated responses to emotional displays of others (Dawson, Webb, & McPartland, 2005).

The retrospective examination of joint attention and gaze monitoring of toddlers through home videos of first birthday parties has provided insight into facial perception in ASD. In novel and uncertain situations, typically developing young children will seek affective cues from

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others' faces. Given that a child's first birthday can be a highly social, yet novel event, the setting provides the opportunity for a range of observable social behavior. Osterling and Dawson collected videotapes of 1st year birthday parties from children who were later diagnosed with ASD and from children with typical development. The tapes were rated by coders blind to the child's subsequent diagnostic status. The authors found that the children later diagnosed with ASD looked at and attended to faces significantly less than the typically developing children. The authors determined that the single most critical factor in recognizing early signs of ASD was a failure to look at others and attend to faces in a typical manner, at or before 12 months old (1994).

Current Knowledge

The dynamic processes related to facial perception can be examined from behavioral, neuroanatomical, and neurophysiological perspectives. Much of our understanding of facial perception has been derived from clinical work and research with individuals with ASD. These impressions suggest that individuals with autism, compared to typical peers, perform worse on face discrimination, recognition, and memory tasks when behaviorally assessed. Additionally, individuals with ASD display atypical development and anatomical activation of the brain structures involved in face processing and exhibit delayed and differential neurophysiological responses to faces.

Behavioral Perspectives on Face Perception

Very early in life, typically developing individuals show an attentional preference for faces and are equipped for the specialization of face processing. These specialized abilities involve recognizing upright faces better than inverted faces and interpreting complex interactions such as direction of gaze, emotional expressions, and facial gestures. Into middle childhood, the specialization for facial perception grows in complexity. Typically, individuals show better memory for faces than objects. They also derive meaningful social-emotional information from faces through preferential attention toward the eyes and perceive faces as a holistic unit rather than isolating individual facial features (Joseph & Tanaka, 2003). In contrast, individuals with ASD attend to inverted faces for the same amount of time as upright faces and recognize inverted faces better than typical individuals by focusing on isolated features of the face rather than the face as a whole (van der Geest, Kemner, Verbaten, & van Engeland, 2002). Further, when viewing faces, individuals with ASD fixate on the mouth region rather than the eyes. Accordingly, they exhibit better performance memory for the lower portion of the face than the upper portion of the face but show a decrease in attention to the entire internal region of the face. As a result, individuals with ASD perform in the below average range on tasks of overall face memory ability. Further, individuals with ASD show impairments in the ability to use information from faces to guide behavior, such as failing to monitor a gaze shift and follow a gaze to share attention.

Studies of facial discrimination and recognition using eye tracking suggest that individuals with ASD employ atypical strategies during facial perception tasks. First, individuals with ASD attend to other objects in the environment more often than the face. For example, a pioneering study using eye tracking found that when watching a videotape of the movie *Who's Afraid of Virginia Woolf*, individuals with ASD attended two times more to the actors' mouth, bodies, and objects in the scenes than to the actors' eyes (Klin, Jones, Schultz, Volkmar, & Cohen, 2002). This suggests that individuals with ASD do not seek social information and cues from naturalistic exchanges in the way that typical individuals seek social information. Subsequent studies have shown that individuals with ASD utilize atypical spatial distribution fixation strategies exemplified by fixating to, or avoiding specific regions of the face, focusing on the mouth, rather than the typical direction toward the eyes.

A second atypical face perception strategy that individuals with ASD appear to use involves

processing faces utilizing a piecemeal (i.e., feature-based) strategy (Chawarska & Shic, 2009). In contrast, typical individuals employ the piecemeal processing strategy when looking at objects but not faces. Typical individuals attend to faces holistically by utilizing a complex-looking strategy called configural processing (Dawson et al., 2005). Configural processing is the perception and encoding of faces as a single holistic unit by incorporating the major features (i.e., eyes, nose, mouth), in respect to other features throughout the face.

To explore the limitations of the configural strategy employed by the typical population, researchers alter the configuration of faces by inverting and partially obscuring the images.

Typical individuals are adept at distinguishing similarities between faces that are upright; however, when the face is inverted, it is more difficult to discern facial feature differences. Inversion appears to alter the perception of the face by challenging the relational facial features (i.e., eye, nose, mouth), which contests the configural processing looking strategy producing what is called the inversion effect. While inverting faces results in the inversion effect, altering a face by forcing one to focus on individual facial features results in the decomposition effect. Even though typical individuals are adept at perceiving faces, once the face is altered from its original composition (e.g., inverted or presented in sections), the accuracy in which the face is identified is challenged. By challenging the configural processing strategy in these two ways, typical individuals have to abandon a configural processing strategy when looking at faces and adopt a piecemeal strategy as if they were looking at an object. This concept provides support that face perception is a highly specialized and sensitive processing system, which when challenged, forces individuals to compensate using differential processing strategies and neuroanatomical mechanisms that are not specialized for face perception tasks.

From studies exploring the configural processing strategy, the decomposition effect and the inversion effect, researchers have found that typical individuals attend to inverted faces for longer lengths of time, are better at recognizing parts of the face when presented in the context of the entire face, and are better at recognition of the eyes compared to the mouth than their peers with ASD (van der Geest et al., 2002). When presented with the inversion face perception task, individuals with autism spend an equal amount of time looking at upright and inverted faces. This is similar to the way typical individuals attend to upright and inverted objects. By employing the object specific, piecemeal processing strategy, individuals with ASD are better at perceiving specific features of the face or partially exposed sections of the face with emphasis on local detail rather than global detail. In fact, individuals with ASD are in some cases better at feature-based matching, especially around the mouth, because they are not inhibited by the implementation of decomposition effect.

These behavioral-based face perception studies suggest that typically developing individuals use the configural processing strategy to perceive faces and the piecemeal processing to perceive objects while individuals with ASD appear to apply the piecemeal strategy to process both faces and objects. The benefits of configural processing strategies are reflected in better performance on face discrimination and recognition tasks and better memory for faces relative to objects for typical individuals. In contrast, children with ASD perform equally to typical peers and in some cases better on nonface memory recognition tasks that include objects such as buildings, animals, houses, and bicycles (Blair, Frith, Smith, Abell, & Cipolotti, 2002).

Neuroanatomical Perspectives on Face Perception

Much of our understanding of the neuroanatomical structures related to face perception has been driven by research with individuals with impairments in face perception. Studies of the brain in individuals with prosopagnosia, a disorder characterized by intact object recognition ability, but impairment in the ability to recognize faces, provide a model for face perception that can be applied to ASD. Neuroanatomical findings in individuals with prosopagnosia, who exhibit

behavioral face perception impairments similar to those found in ASD, indicate damage to particular cortical regions, such as the fusiform gyrus, indicating a critical role in face perception for this neural region (De Renzi, Perani, Cariesimo, Silveri, & Fazio, 1994). By utilizing functional magnetic resonance imaging (fMRI), structural magnetic resonance imaging (MRI), and positron-emission tomography (PET), neuroimaging research has provided further evidence that occipitotemporal cortical and other neural structures are functionally abnormal in individuals with ASD during facial perception. The regions identified as playing a primary role in face processing include the fusiform gyrus, the posterior superior temporal sulcus, and the amygdala. This dynamic human face processing system that is linked to the structural encoding of facial features, processing emotional expressions, and interpreting biological movement is critical to understanding the fundamental neural circuitry of the "social brain" (Pelphrey, Morris, McCarthy, & LaBar, 2007).

Fusiform Gyrus

Research over the last decade has verified a region of the fusiform gyrus that activates more strongly to faces than any other visual stimuli and appears to be specialized for face processing and facial expression discrimination. The fusiform face area (FFA), located in the medial-lateral region of the fusiform gyrus, responds almost twice as strongly to faces than wide varieties of nonface objects (Kanwisher, 2000). However, additional research adds complexity to the role of the FFA region. After finding that the FFA is activated while viewing objects that fall in an individual's area of expertise (e.g., car experts display FFA activation when viewing images of automobiles), Gauthier, Tarr, Anderson, and Skudlarski proposed that the FFA region is not only responsible for face perception but is responsible for the mediation of specific visual expertise. Gauthier and colleagues supported this conclusion by showing that training a group of college students to identify computer generated 3-D objects (called Greebles) results in an increase of activity of the FFA (1999).

Rather than increased activity in the fusiform gyrus when looking at faces as seen in typical development, neuroimaging findings suggest that individuals with ASD display reduced activation of the fusiform gyrus while looking at faces (Schultz et al., 2000). Further, individuals with ASD exhibit an increase in activation in the inferior temporal gyri, a surrounding anatomical region specialized for object recognition. While for typical individuals, viewing objects of expertise, such as faces, results in FFA activation, individuals with ASD show FFA activation only when looking at images of their specialized interest. While the fusiform gyrus is an area linked to facial perception in typical individuals, for individuals with ASD, this "social brain region" fails to activate to faces in the same manner.

Amygdala

While there is strong research to support the FFA role in the perception of faces, the amygdala plays a significant role in detecting emotional expressions on faces. The amygdala is a fast-responding structure that plays a critical role in notifying other brain regions of emotional arousal through a mediated reward system. This structure quickly and reliably reacts to salient environmental stimuli, especially fear, and plays a role in assigning significance and constructing social judgments, such as inferring personality characteristics from pictures of the face or facial features (Ledoux, 1996).

The allure of the amygdala in ASD research was initially recognized in postmortem cases of individuals with ASD who appeared to have limited dendritic tree development resulting in an amygdala that was small and densely packed compared to typical individuals (Bauman & Kemper, 1985). Adolphs found that individuals with ASD demonstrated similar impairments to those with focal lesions to limbic structures, including the amygdala. While individuals with ASD are able to form representations of faces and understand basic knowledge of emotions, they

are unable to link the perception of faces to social judgments that are often revealed by facial expressions. Neuroimaging studies of individuals with ASD have reported hypoactivation of the amygdala when viewing images of emotional and mental states of another individual. For example, Pelphrey and colleagues demonstrated that when emotional faces were presented naturalistically through a dynamic video display, all three components of the broader social brain – the amygdala, the superior temporal sulcus, and the fusiform gyrus – were hypoactive (2007). A study examining the perception of emotionally expressive faces in individuals with amygdala and hippocampal lesions found that there was a strong correlation between the extent of amygdala lesions and hypoactivation of the FFA, suggesting that the amygdala plays a direct role on the FFA when interpreting emotionally expressive face (Vuilleumier, Armony, Driver, & Dolan, 2004).

Superior Temporal Sulcus (STS)

The STS region of the brain is responsible for the detection and processing of biological motion (Pelphrey et al., 2007). In synchrony with the amygdala and fusiform gyrus, this neural network is important for linking structural encoding of facial features and emotions to biological motion. Perception of biological motion is thought to underlie impairments in attention and imitation, skills that are strongly linked to language and social development.

Since past research examined the face processing system through the display of emotional expressions using still image stimuli, Pelphrey and colleagues (2007) were interested in how presenting dynamic facial expressions affects the face processing system. They found that individuals with ASD display reduced activation in the STS, fusiform, and amygdala when faces contain dynamic information, such as movement, relative to static snapshots of the same emotional expressive face. These results were specific to the social brain and were not observed outside of the human face processing system (i.e., STS, fusiform gyrus, and amygdala).

The STS also plays a role in comprehending intentions and detecting "errors" in social situations (Pelphrey et al., 2007). This region modulates tasks involving the attribution of intention in the context of dynamic social situations. During situations in which an individual's actions do not fulfill the expectation (e.g., a character in a story is not looking in the direction that the viewer would think he/she should), there was an increase in blood flow to the STS in a typical observer.

However, for individuals with ASD, there was reduced activity in the STS region. The lack of observed activity in the STS during attribution tasks in ASD provides further evidence that the STS region plays a strong role in the social dysfunction in ASD.

Neurophysiological Perspectives on Facial Perception

By utilizing neurophysiological measures such as event-related potentials (ERPs), scientific studies have provided insight into the neuropsychological processes underlying face perception and face perception impairment in ASD. While fMRI studies have allowed researchers to efficiently investigate abnormal regional brain activity during facial performance tasks, electroencephalogram (EEG) and event-related potentials (ERPs) have allowed researchers to examine the temporal characteristics and strength of neural activity by assessing oscillatory activity in response to viewing faces. Unlike fMRIs, which cannot determine the timing of neuronal firing, EEG/ERPs are able to monitor the timing of the summation of a collection of neuronal postsynaptic potentials firing at the resolution of milliseconds. Most studies utilizing EEG data concentrate on the amplitude, scalp topography, and latency. Amplitude is considered a representation of the amount of neuronal resources recruited for a particular process. Scalp topography distribution differences are thought to represent the location of the originally generated neuronal activity, and the timing of information processing speed is represented through the wave-like display of troughs and peaks, known as latency. Because simply passive

viewing of stimuli is needed for EEG/ERP studies, valuable information can be gathered from children with significant social communication impairments through the use of EEG/ERP to effectively and noninvasively address fundamental questions about the neural basis of face processing in individuals with ASD.

Electrophysiology studies are particularly salient in face processing research because of the discovery of a face-sensitive component that occurs with a negative slope at approximately 170 milliseconds poststimuli exposure. The N170 represents one stage in a series of stages of face processing; the P100, a positive going peak around 100 milliseconds, reflects neural activity in basic visual processing; the N170 reflects structural encoding of the face; and the N250 reflects higher order recognition such as affect or identity recognition. The N170 is recorded over the posterior temporal lobe and is greater in the right hemisphere than the left hemisphere. The N170 responds quicker to face and eye stimuli alone, rather than objects or inverted faces. Slight changes in face composition or inversion alter the latency and amplitude of this component. In typical children, the N170 undergoes a prolonged period of development and does not reach full maturation until late adolescence. Between 4 and 15 years of age, the precursor to the N170, identified as the prN170, is measured from the same electrode configuration as the N170 in adults but is more positive (does not extend beyond the baseline) than the fully maturated N170 component in adults. The prN170 in children ages 3–6 years is shown to have a faster response to faces. Similar to adults, this component is shown to have preferential responses to faces than objects.

McPartland, Dawson, Webb, and Panagiotides documented the first report of an altered N170 pattern in adolescents and adults with ASD (2004). Individuals with ASD showed a slower latency when looking at faces than furniture and failed to show the face inversion effect. Additionally, researchers observed a slower speed of information processing which disrupts early structural encoding. These differences in the timing of cortical processing suggest that individuals with ASD have abnormal circuitry or delayed neural processes that modulate the face processing system.

Children with ASD also show disruptions to this physiological measure of face processing. They demonstrate longer prN170 responses to faces than objects. By the age of 6, compared to typically developing peers who continue to show faster responses to faces over objects, children with ASD show no latency differences to faces versus objects (Webb, Dawson, Bernier, & Panagiotides, 2006). In addition, research indicates that children with ASD display structural encoding of faces that is disrupted and slowed. While typically developing children exhibit a faster prN170 response to faces than objects, individuals with ASD exhibit a slower prN170 response to faces than objects.

Differential activation patterns have been observed using ERP studies in typical children comparing familiar and unfamiliar stimuli (i.e., caregivers to strangers, familiar objects to unfamiliar objects) as early as 6 months. This suggests early preferential neurological responses to familiar over unfamiliar faces and objects (de Haan & Nelson, 1999). Typically developing infants and young children display ERP differences when viewing familiar and unfamiliar faces and objects at the P400 component recorded from posterior electrodes. Children with ASD, however, fail to show a differential P400 component when looking at familiar versus unfamiliar faces. Instead, children with ASD display an enhancement in the P400 component when looking at favorite objects versus unfamiliar objects. These results provide evidence that children with ASD have specific impairment in processing faces, but not objects.

Children with ASD also show abnormal electrophysiological responses when viewing emotional expressions. Compared to typical peers, children with ASD show a slower and more positive N300 wave in response to fear and exhibit abnormal scalp topography. This slower information processing for faces in the N300 for emotional stimuli is associated with greater severity in social domains such as joint attention and social orienting (Dawson et al., 2004).

Neurophysiological studies in early childhood provide insight into the structural and neurological mechanisms that are involved in the development of face processing. Face processing impairments examined at the neurophysiological level provide insight into social brain functioning and the face processing system in individuals with ASD from infancy and throughout development.

Future Directions

Faces hold special significance and convey valuable social information early in life. Face perception provides a foundation for the development of social cognitive skills such as sharing attention and understanding emotions. Given the prominent role of face perception in social cognition, the study of ASDs has provided insight into the behavioral, neuroanatomical, and neurophysiological components of face perception.

Behavioral studies of face perception in individuals with ASD have yielded insight into visual scanning, face processing strategies, and the role of expertise of processing information. Imaging studies have identified structures related to the processing of faces, such as the fusiform gyrus, superior temporal sulcus, and amygdala, and demonstrated that the structures of the "social brain system" involved in face perception appear to be functioning differently in ASD. Neurophysiological studies have elucidated face-specific brain waves and revealed temporal delays in the processing of faces in ASD.

The ability to process and gather information from facial expressions is crucial for successful social interactions. This unique ability allows humans to understand affective cues in uncertain situations, such as situations that may be dangerous and important for survival, and to accurately read the emotions of another individual. This ability provides attentional and directive focus through gaze monitoring of others and provides a catalyst to overtly and covertly mimic the facial expressions and affective states of others, such as states that offer the opportunity for bonding or shared reciprocal experience (Chawarska & Shic, 2009). While face perception provides immediate insight and interpretation of the social world, over time, and development, it is clear that early developing face processing skills set the foundation for the maintenance of exchanges and social relationships.

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Face Recognition

Cora Mukerji[1], Danielle Perszyk[1] and James C. McPartland[2]
[1]Yale Child Study Center, New Haven, CT, USA
[2]Yale Child Study Center, Yale University, New Haven, CT, USA

Definition

Face recognition is an important dimension of face processing in which specific information about identity is derived. The capacity to recognize individual faces is critical to socialization throughout development and adulthood, providing important social cues that guide interactions. While no scientific consensus exists on whether autistic populations show behavioral impairment in the ability to recognize faces, substantial evidence indicates that individuals with autism may use atypical visual and neural mechanisms to determine facial identity. By disrupting the extraction of important social information from the face, these abnormalities in processing facial recognition may contribute to the social deficits that characterize autism spectrum disorders (ASD).

Historical Background

Faces are highly relevant visual stimuli, conveying information critical to social understanding and effective communication. The ability to accurately recognize others' faces, which allows for quick identification of others and retrieval of information associated with them, is particularly critical to social function. Developmental studies indicate that face recognition facilitates engagement with others from infancy; from the first days of life, infants demonstrate the capacity to recognize their mothers' faces among others (Pascalis, de Schonen, Morton, Deruelle, & Fabre-Grenet, 1995). Further studies of face recognition ability in children have revealed that the basic capacity to differentiate faces matures over the course of development, following a developmental trajectory distinct from general pattern recognition or memory (Carey, Diamond, & Woods, 1980).

Neuropathological findings have emphasized the importance of face recognition over the lifespan, suggesting that circumscribed regions of the brain are specialized for differentiating faces. Patients with prosopagnosia, a disorder of face perception, exhibit normal recognition of common objects in spite of severe impairment in recognizing faces (Whiteley & Warrington, 1977), while individuals demonstrating severe impairment in recognizing common objects retain normal face recognition (Moscovitch & Winocur, 1997). Together, these findings suggest a double dissociation between face and object recognition, i.e., that face and object recognition

rely upon distinct neural networks in the brain. Subsequent neuroimaging studies have indicated that the region of the temporal lobe known as the fusiform gyrus is activated preferentially during face recognition tasks, suggesting the specialization of this brain region for face processing (e.g., Kanwisher, McDermott, & Chun, 1997). However, recent studies reveal that nonface objects with which an individual has expertise and novel face-like objects also animate this brain region, calling this interpretation into question (Grelotti et al., 2005).

In addition, early research into the effects of inversion upon recognition accuracy has indicated that face and object recognition rely upon distinct processing strategies. Yin (1969) discovered that inversion of faces impairs recognition in typical adults to a greater degree than does inversion of common objects; this robust phenomenon, known as the "inversion effect," suggests that adults use a different visual strategy to identify faces versus objects. Subsequent research indicates that, whereas object recognition depends upon discrimination of individual features, accurate face recognition relies more heavily upon the configuration of the face, which is disrupted during inversion. This processing strategy, however, may not be specific to faces but more generally applied to stimuli that an individual is expert at recognizing; individuals with expertise in nonface objects demonstrate an inversion effect for objects in their domain of expertise comparable to that for faces (Diamond & Carey, 1986).

The first experimental investigations of face recognition in autism were carried out by Langdell (1978) to assess whether disruption of the typical visual mechanisms underlying face recognition might contribute to the social impairment observed in children with autism; this research indicated that children and young adults with autism are proficient in recognizing faces but employ atypical visual strategies in order to do so. While children with autism did not exhibit behavioral deficits in recognizing photographs of their peers, they demonstrated a greater reliance on features located in the lower parts of the face (i.e., the mouth region) than did typical peers. In contrast, typically developing children relied more heavily on the upper part of the faces (i.e., the eye region) to recognize peers, replicating previous findings. Langdell (1978) also found that older children with autism failed to show an inversion effect for faces, while typically developing children did. Similarly, Hobson, Ouston, and Lee (1988) found that adolescents and young adults with autism were significantly better than typical peers at recognizing inverted faces, which they were able to identify as well as upright faces, suggesting that individuals with autism employ different processing strategies to determine facial identity than do typically developing individuals.

Current Knowledge

Initial studies of face recognition ability in autism indicated that children and adolescents with autism demonstrate behavioral proficiency in recognizing faces but atypical patterns of visual processing. Current research continues to explore the question of whether individuals with autism are impaired in face recognition. However, the focus of this line of investigation has largely shifted from reporting behavioral performance to clarifying the visual mechanisms and neural networks that underlie the distinct processing strategies for face recognition in ASD.

To date, no consensus exists as to whether individuals with autism demonstrate significant deficits in face recognition relative to typically developing peers matched on cognitive and verbal ability. While several recent studies report behavioral impairment in face recognition in children with ASD that is independent of overall functioning, many have failed to report such impairment (*see* Klin et al., 1999 *for a review*). Although most research to date indicates that recognition deficits in autism are specific to faces, some studies have reported more general impairment in processing both face and nonface stimuli (e.g., Davies, Bishop, Manstead, & Tantam, 1994). Klin and colleagues (1999) suggest that these inconsistencies in the literature are attributable to differences in age and functioning in the subject populations, control group criteria, and experimental tasks employed across

studies of face recognition in ASD; in addition, Klin and colleagues (1999) hypothesize that abnormalities in the way facial identity is determined in autism may not always translate into impaired performance because children with autism may develop compensatory strategies for face processing to overcome difficulties in the critical domain of face recognition.

While controversy exists as to whether individuals with autism demonstrate behavioral impairment in the specific domain of face recognition, an emerging body of research supports the hypothesis that atypical visual mechanisms underlie face recognition processing in ASD. Recent studies have provided direct evidence that the facial inversion effect observed in typical adults reflects the use of a configural visual strategy (i.e., one that relies upon the spatial relationship between individual features) for face recognition that is distinct from the feature-based strategies employed in object recognition (Freire, Lee, & Symons, 2000). Given the lack of inversion effect in autism, this suggests that facial recognition in autism relies less upon the configural strategies and more upon the feature-based strategies characteristic of object recognition in typical development. However, subsequent work has indicated that high-functioning children with ASD do employ holistic strategies for face recognition (i.e., evaluating the face as a whole rather than part by part) in instances where accurate face recognition depends upon the mouth region of the face (Joseph & Tanaka, 2003); this suggests that atypical patterns of face recognition in autism cannot be fully explained by impairment in configural processing of faces.

Current research confirms that individuals with autism rely upon different regions of the face to determine facial identity. While typically developing children are more proficient in recognizing faces based upon the eyes compared to the mouth, high-functioning children with ASD demonstrate the reverse pattern (Joseph & Tanaka, 2003). Eye-tracking studies have provided further evidence that, while observing and encoding faces, individuals with autism may preferentially attend to different face regions than do typically developing peers. Prior research has indicated that adults with high-functioning autism (HFA) spend more time scanning the external features of the face than core internal features (Pelphrey et al., 2002). Similarly, adults with HFA have been found to spend more time viewing mouths, bodies, and objects than eyes while viewing video clips of dynamic social interactions (Klin, Jones, Schultz, Volkmar, & Cohen, 2002). In comparison, typically developing subjects spend more time scanning the eye region of faces, indicating that abnormal patterns of viewing faces in autism, characterized by reduced attention to the socially informative eye region of faces and enhanced attention to mouths, may underlie atypical face recognition in ASD (Klin et al., 2002).

Neuroimaging techniques have recently been applied to shed light on the neural mechanisms that support the distinct processing of facial identity in autism. While brain imaging studies of typically developing individuals have consistently revealed activation in the fusiform face area (FFA) to viewing faces (e.g., Kanwisher et al., 1997), individuals with autism demonstrate atypical patterns of neural activation during face processing (Pierce, Muller, Ambrose, Allen, & Courchesne, 2001; Schultz et al., 2000). Work in this field has revealed evidence that face processing can occur outside the FFA, at individual-specific sites, in ASD (Pierce et al., 2001). This difference in where face processing occurs in the brain may be associated with distinct strategies for face recognition in this population. Furthermore, individuals with autism and Asperger disorder have shown weak activation in the FFA and heightened activation of the inferior temporal gyrus during a face discrimination task, the same pattern of brain activity observed when typically developing individuals performed a nonface object discrimination task (Schultz et al., 2000). This supports the idea that individuals with ASD may use feature-based processing strategies to discriminate among both faces and objects, showing a lack of typical specialization of face processing strategies in this population.

Further studies have revealed key differences in how the brain response to faces is mediated in real time in typical and atypical development,

suggesting that children with autism show impaired neural processing of faces at multiple stages of face recognition. Two distinct temporal markers of brain activity (the N400 and Pc) have been found to differentiate between familiar and unfamiliar faces and familiar and unfamiliar objects in typically developing 3- and 4-year-olds; in contrast, same-age peers with autism show differentiation between familiar and unfamiliar objects but not between familiar and unfamiliar faces at these two markers, suggesting that impairment in processing facial recognition emerges early in life (Dawson et al., 2002). Disruption of typical mechanisms for discriminating faces at this early age may derail subsequent brain and behavioral development by affecting how children with autism attend to faces in the world around them and how effective they are at gathering critical social information from faces (Dawson et al., 2002). Additional research has revealed that individuals with autism also show significant delay at a key temporal marker of face processing, the N170, compared to typically developing individuals, reflecting slowed neural speed of face processing in autism only 170 ms after viewing a face (McPartland, Dawson, Webb, Panagiotides, & Carver, 2004). Slowed speed of processing has been correlated with poorer performance on tests of facial recognition in autism; this suggests that greater delays in neural processing of faces may contribute to difficulties in face discrimination. While the N170 is delayed in typical development to inverted versus upright faces, this inversion effect is not observed in ASD, providing further support for the idea that individuals with autism may rely upon configural rather than typical holistic strategies for processing faces (McPartland et al., 2004).

Future Directions

While an emerging body of eye-tracking and neuroscientific research has provided evidence that individuals with autism employ atypical visual and neural mechanisms for discriminating faces, the exact nature of the relationship between these divergent mechanisms and social impairment in autism remains unknown. Atypical mechanisms for recognizing faces may serve as both a cause and an effect of social impairment in autism. For example, reduced attention to faces early in development may disrupt typical specialization of visual strategies and neural circuitry for discriminating faces; the resulting abnormalities in processing facial recognition may further deprive individuals of critical social inputs necessary for the development of typical patterns of social behavior (Dawson et al., 2002). Additional work is needed to clarify the connection between divergent patterns of visual and neural processing of facial identity and the social difficulties that characterize autism in order to design effective interventions.

See Also

- ▶ Face Perception
- ▶ Fusiform Face Area
- ▶ Fusiform Gyrus (FG)

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Face Tests

▶ Facial Recognition Tests

Face Validity

Ellen Johnson
Section of Social Work, Mayo Clinic, Rochester, MN, USA

Definition

Face validity refers to the extent to which a test appears to measure what it is intended to measure. A test in which most people would agree that the test items appear to measure what the test is intended to measure would have strong face validity. For example, a mathematical test consisting of problems in which the test taker has to add and subtract numbers may be considered to have strong face validity. The test items appear, at face value, to measure what one is seeking to measure.

See Also

▶ Validity

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Face-Processing Tests

► Facial Recognition Tests

Facial Imitation

► Oral-Facial Imitation

Facial Inversion Effect

► Upright/Inverted Figures

Facial Recognition Tests

Susan Faja
Department of Psychiatry and Behavioral Sciences, University of Washington, Seattle, WA, USA

Synonyms

Face-processing tests; Face tests

Abbreviations

BTFR	Benton test of facial recognition
CMS	Children's memory scales
K-ABC-II, FR	Kaufman assessment battery for children-II, face recognition subtest
LFI	Let's face it!
WMS	Wechsler memory scales

Description

A variety of tests have been developed to measure the ability to recognize faces. In order to recognize a face, one must have the basic perceptual ability to distinguish that face from others, often over time. Tests that measure this ability provide information about the accuracy with which the face is represented, recognized, and distinguished from others. A range of skills are critical for face recognition including the ability to analyze the face and its features, understanding the relationship between these features within the overall configuration of the face, perceiving the identity of the face across changes in viewpoint and expression, and explicitly recognizing the faces of familiar people.

Recognition of faces is a critical throughout social development because faces apart from any other physical aspect are used to identify others in social situations. The face provides a unique way to identify individuals and comes to serve as a representation of that person and their personal characteristics. Infants distinguish faces of familiar individuals shortly after they are born. Without the early ability to recognize and remember faces or to make use of the nonverbal information they contain, many other subsequent learning processes may be derailed, including the development of coordinated eye contact, joint referencing, and emotional reciprocity.

Impaired face recognition in individuals with autism spectrum disorders has been demonstrated with a wide range of experimental tests. On average, groups with autism spectrum disorders are less accurate in their ability to recognize the faces

of others. This is true for discrimination of facial identities, recognition of familiar faces, and immediate recognition of newly learned faces. Impairment in face recognition appears to be somewhat specific because individuals with autism are typically unimpaired in their recognition of objects. On an individual level, there appears to be variability in the ability of people with autism spectrum disorders to recognize faces. There is some evidence of an association between the degree of severity of face-processing impairments and the severity of other more general social symptoms. For this reason, tests of facial recognition are important in classifying the degree of difficulty individuals experience in this perceptual domain in clinical and research settings. Face recognition tests are also useful when describing group characteristics of a research sample because they provide a common way for experimental measures to be interpreted and compared across studies.

Historical Background

Early reports of individuals who experienced difficulty with recognizing faces were made in the late 1800s, though these difficulties were usually associated with other visual impairments. Bodamer (1947) coined the term "prosopagnosia" (i.e., "loss of knowledge of faces") to describe individuals who had greater impairments with faces than objects. In order to test these patients with frank impairments in face recognition, tests of face recognition began to be developed. For example, in the 1960s, Benton and Van Allen developed a face-matching test and De Renzi and colleagues developed a same-different face-matching test. The former became the Benton Facial Recognition Test (Benton, Sivan, Hamsher, Varney, & Spreen, 1983). Warrington (1984) developed a test that involved learning a set of new faces followed by a series of target-distracter face pairs to test recognition memory for faces, although this measure has not been widely used with autism and nonface information is contained in the pictures that may aid in the discrimination of targets. In more recent years, similar face recognition tests have been included with standardized batteries of memory and neuropsychological functioning. The Wechsler Memory Scales-III (Wechsler, 1997) includes the faces subtests, which are appropriate for adults. A very similar task is included in the Children's Memory Scales (Cohen, 1997) and is appropriate for children. Also appropriate for younger children are the face recognition subtest of the Kaufman Assessment Battery for Children (K-ABC-II; Kaufman & Kaufman, 1983) and the memory for faces subtest of the NEPSY-II (Korkman, Kirk, & Kemp, 2007). These measures along with experimental tasks have been used to assess face recognition in autism.

The face processing of individuals with autism has been studied extensively since Langdell (1978) first offered evidence of atypical strategies in the way young children with autism use information to identify familiar faces. Langdell proposed that face-processing deficits may explain the social symptoms associated with autism including impaired eye contact and reduced social reciprocity. Throughout the 1980s and 1990s, a series of studies followed that revealed impaired or peculiar patterns of face recognition in children, adolescents, and adults with autism. In many cases, these studies did not use standardized measures of face recognition and instead used tasks developed for the particular study being conducted.

Most investigations of face recognition have been limited by small sample size with a few exceptions, thus, limiting the ability to generalize conclusions about the integrity of the face recognition system in individuals with autism and ability to determine the expected range of function for individuals with autism. In an effort to address this problem, the face recognition subtest of the K-ABC was administered to over 100 children with autism, pervasive developmental disorder – not otherwise specified – and typically developing children (Klin, Sparrow, de Bildt, Cicchetti, Cohen, & Volkmar, 1999). Children with autism performed worse than the other two groups, even when matched on verbal and nonverbal mental age. Based on this investigation, it was concluded that children with autism exhibited impairments

in face recognition that could not be accounted for by more generalized cognitive impairment and that these difficulties were limited to children with autism, while children with PDD-NOS did not demonstrate difficulties.

Most studies have been also limited by use of a single measure of face processing or recognition. As a result, it has been difficult to compare results across studies, and the ability to determine the nature and relative contribution of various processing deficits is limited. Additionally, while standardized measures provide a way to compare performance across studies, they are limited by their sensitivity to the specific peculiarities of face processing exhibited by individuals with autism (i.e., a focus on features within the face rather than the face as a whole, biased processing of the lower half of the face, and reduced disruption in performance when faces are presented upside down). To address these limitations, the *Let's Face It!* Skills Battery was developed by Tanaka and Schultz. The *Let's Face It!* Skills Battery is a battery of computerized face recognition tasks aimed at children and includes measures of short-term memory for faces, assessments of feature processing and configural perception, and holistic processing. Many of the tasks involve matching and require children to recognize individual faces across changes in orientation and emotional expression and when certain regions of the face are obscured. The battery also includes tasks that measure processing and short-term memory for objects. Each test within the battery was administered to over 60 children, with the exception of the object memory task. The LFI Skills Battery was sensitive to autism-related impairments in recognition of faces across changes in orientation, expression, and feature (immediate memory face subtest, face-matching subtest). Performance on the part/whole subtest also revealed a processing advantage for the mouth region of the face in the autism spectrum group. However, like controls, individuals with autism spectrum disorders had an advantage processing whole faces rather than isolated features. Finally, performance for the object processing control conditions was unimpaired in the group with autism spectrum disorders, which is consistent with previous findings of a face-specific impairment in autism.

Psychometric Data

All tests presented are individually administered and are thought to measure face recognition with still photographs of unfamiliar faces.

The *Benton Test of Facial Recognition* (BTFR; Benton et al., 1983) is a measure of the ability to perceive and match unfamiliar faces without a memory component. Normative samples span a wide range of ages (6–74 years). Black and white photos are presented, with the target at the top of the page and six test faces at the bottom of the page. Photos vary based on orientation and lighting condition across three conditions: (1) an identical photograph, (2) the same face with different lighting, and (3) three-quarter views of the same face. Initially, there is only one target face, but in the latter portion of the task, three of the six faces have the same identity (i.e., there are three correct responses). Administration time typically ranges from 5 to 15 min, although the items are untimed. The Benton Test of Facial Recognition has a short and long form (i.e., a subset of the first 13 items versus the full set of items). Scores are obtained by a simple summation of correct responses. The psychometric properties of the long form are adequate at best, and the short form has poor psychometric properties. In particular, evidence challenging the construct validity comes from work by Duchaine and colleagues who found adults with typical development were able to score in the normal range when administered a version of the BTFR that presented only the eyebrows and hairline of faces. These authors concluded simultaneous presentation of targets, and test faces limited the test by allowing the possibility of feature-based strategies to be employed. The same group provided evidence challenging the predictive validity of the BTFR by reporting individuals with developmental prosopagnosia who scored within the normal range. Normative data is available for 286 adults and 229 children and was collected in Iowa in 1994 (Benton, Sivan, Hamsher, Varney, & Spreen, 1994).

The *Cambridge Face Memory Test* (Duchaine & Nakayama, 2006) is a nonstandardized assessment of face processing, recognition, and memory of unfamiliar faces. The test is appropriate for adults and was recently developed and tested with college students and older adults. However, it provides increasing difficulty levels and has been used with children with typical development and autism spectrum disorders (O'Hearn, Schroer, Minshew, & Luna, 2010). Administration time takes between 10 and 15 min. There are four phases of the task (practice with cartoons, introduction with the same images, novel images, and novel images with noise). During the same images phase, six male faces are first learned by viewing them from three angles (left, frontal, and right) for 3 s each. In this phase, three test items that are identical to the study images are presented for each face. In the novel images phase, a brief review of the study images is followed by presentation of 30 test items that vary by lighting or pose. Finally, in the novel images with noise phase, study images are again briefly reviewed followed by 24 test items with different level of Gaussian noise added to the image. The Cambridge Face Memory Test also assesses the effect of face inversion, which is thought to disrupt specialized face-processing mechanisms. Initial data are presented supporting the validity of the Cambridge Face Memory Test, but normative data and reliability measurements were not available. This measure is available for research.

The *Children's Memory Scales: faces immediate and faces delayed subtests* (Cohen, 1997) measure face recognition and memory with unfamiliar faces. The test is appropriate for children aged 5–16 years and can be administered in 10 min plus the 25–35-min delay. A series of color photographs of unfamiliar faces are presented one at a time for 2 s (either 12 or 16 faces depending on age). Immediately, a series of test faces are presented one at a time, and the child is asked to respond to each by indicating whether the face was viewed in the learning phase. After a delay, a second series of test photos is presented, and children are asked to respond in the same manner. Correct responses for the immediate and delayed faces are totaled to arrive at two scaled scores. The stability of the assessment across administrations is adequate. Normative data were obtained from a representative sample of the US population in the 1990s.

The *Kaufman Assessment Battery for Children-II: face recognition subtest* (K-ABC-II, Kaufman & Kaufman, 2004) is standardized for ages 3–18; however, psychometric data for the face recognition test is available only for ages 3–5 years. The test involves viewing and attending to one or two photographs of faces with a brief exposure. Then, children are asked to select the correct face or faces from a group. The task requires children to recognize the target face(s) across changes in pose. In the revision of the K-ABC, new items were added to the subtest, and distracting features in the background of photos were removed. The K-ABC-II face recognition subtest is standardized on a national sample of children and has excellent psychometric properties.

The *Let's Face It! Skills Battery* (Wolfe et al., 2008) is a battery of tasks designed to measure specific aspects of face processing as well as memory and is aimed at children with autism spectrum disorders. Administration of the battery occurred over 2 days in the initial report and requires more time for completion than other individual tests described in this section. The battery contains five facial identity tests, three emotion processing tests, and two object processing tests. Face recognition subtests include matching identity across expression, matching identity across masked features, featural and configural face discriminations, parts/whole identity, and immediate memory for faces. The LFI Skills Battery is not standardized but has been administered to a relatively large sample of children with autism spectrum disorders (85 total, with >60 responding to any given subtest) and typical development. The age range of participants was 5.1–20.1 years for the group with autism spectrum disorders (mean age = 11.58). The sample was high functioning, with a mean full scale IQ of 99.74 and included 71 males and 14 females. The effect sizes for individual subtests are moderate to large.

The largest effect sizes were for the matching identity across expression subtest (effect size = 1.00) and for the immediate memory for faces subtest (effect size = 1.00). Moderate effect sizes were reported for task conditions in which the eye region was tested (parts/whole identity, face dimensions).

The *NEPSY-II memory for faces subtest* (Korkman, Kirk, & Kemp, 2007) is a subtest is taken from a larger battery of neuropsychological assessments measuring attention, executive function, language, learning and memory, sensorimotor function, social perception, and visuospatial processing. Specifically, the memory for faces subtest is thought to measure encoding, discrimination, and recognition of unfamiliar faces, while the delayed memory subtest assesses long-term face memory. The task is standardized for ages 5–16 years. Normative data were collected between 2004 and 2006 from across the United States and included 1,200 children aged 3–16 years. The revision process also included clinical studies of children with autistic disorder and Asperger's disorder. The memory for faces task presents children with a series of photos of faces, during which the child is asked to identify the gender of the face as a way to improve attention. Immediately after presentation, the child selects the target faces from groups of three faces (a target and two distracters). Fifteen to twenty minutes later, the memory for faces delayed subtest may be administered, and children are asked to select the same set of targets from a new sets of three faces. The subtest yields three scores – memory for faces total score (immediate recognition), memory for faces delayed total score (delayed memory for faces), and memory for faces versus memory for faces delayed contrast scaled score (contrast between the first two). The validity of the subtest is supported by moderate correlations between the memory and delayed memory portions, between the NEPSY-II and NEPSY, between the NEPSY-II memory for faces and CMS faces subtests, and by diminished performance by children with autism spectrum disorders. Concurrent validity data suggest that processing speed and visual discrimination are also involved. Inattention, impulsivity, and repetitive behaviors may influence performance. The internal consistency of the NEPSY ranges from adequate to high and has adequate stability over time. Of interest, the NEPSY-II battery also includes other measures of social function including affect recognition and theory of mind subtests.

The *Wechsler Memory Scales-III faces subtest* (Wechsler, 1997) is a measure of face memory and recognition for use with individuals aged 16–89 years. As with the CMS, this subtest may be administered in 10 min plus the 25–35-min delay. Twenty-four photographs of unfamiliar faces are presented one at a time for 2 s, followed immediately by a test phase of 48 faces. The adult is asked to respond to each by indicating whether the face was viewed in the learning phase. After a delay, a second test series is presented in the same way. Correct responses for the immediate and delayed faces are totaled to arrive at two scaled scores (immediate and delayed). The scales were normed on a representative sample of over 1,000 individuals throughout the United States during the 1990s, with good reliability. The WMS has since been revised (WMS-IV), but the faces subtest was not included in the most recent version of the battery.

Clinical Uses

Measurement of face recognition in a clinical setting may provide useful information about social function as well as memory for social versus nonsocial information. Subtests within larger batteries provide a means for such comparisons and offer standardized scores for comparison with the population. For preschoolers, the K-ABC-II face recognition subtest provides a useful measure. Older children may be assessed with tests such as the NEPSY-II memory for faces subtest or Children's Memory Scales: faces immediate and delayed subtests. The Wechsler Memory Scales-III faces subtest provides a similar measure for adults. These tests provide a more global assessment of face recognition but may be influenced by motivation, attention, and repetitive or impulsive responding. As well, they

do not provide insight into why face recognition may be disrupted. An emerging research literature with tests such as the *Let's Face It!* Skills Battery and Cambridge Face Memory Test, which provide more focused assessment of face recognition and perception, suggests that such tests may ultimately be available.

See Also

- ▶ Face Perception
- ▶ Face Recognition
- ▶ Memory Assessment

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Facilitated Communication

Maureen Nevers
Augmentative Communication Consultant, Center on Disability & Community Inclusion, Burlington, VT, USA

Definition

Facilitated communication (FC) is a form of augmentative communication in which a person with a communication impairment expresses themselves by pointing (e.g., at pictures, letters, or objects) or typing (e.g., on a keyboard). Also called Facilitated Communication Training, this method involves a communication partner (facilitator) who may provide emotional, communication and physical supports to enable the person to point reliably to communicate (ICI Research Statement 2010).

Historical Background

FC was initially developed in Australia by Rosemary Crossley, who began using physical support to help nonspeaking individuals communicate in the 1970s. In 1989, Douglas Biklen of Syracuse University visited the DEAL Center in Australia and observed individuals using FC. In 1990,

Biklen published an account of his DEAL observations in the *Harvard Educational Review* article titled "Communication Unbound." Subsequently, the use of FC expanded rapidly in the United States, and in 1992, the Facilitated Communication Institute at Syracuse University was established. By 1993, controversy about the validity of FC had grown, with negative research findings and media reports. Throughout the 1990s, many studies tried unsuccessfully to establish the validity of this approach. In 2010, the focus of the Facilitated Communication Institute at Syracuse University was broadened, and it was renamed the Institute on Communication and Inclusion at Syracuse University.

Rationale or Underlying Theory

FC presumes that the user has communicative competence and will be able to produce meaningful, even complex messages with the proper support. Typing to communicate then promotes access to social interaction, academics, and participation in inclusive schools and communities.

Goals and Objectives

The Facilitated Communication Standards (FCTS, 2011) suggest two primary goals: (1) describing the elements of best practice in facilitated communication (FC) and (2) outlining a training process which promotes those best practices in the use of FC. The intention of the FCTS document was to develop consistently high-quality practice of facilitated communication across users and facilitators, increase the focus on working toward independence, and give assurance to agencies that the method is being applied correctly.

According to the FCTS, a primary, long-term goal of facilitated communication training is independent communication. This goal involves being able to access communication aids without physical support, being able to initiate the process of communication with others, and exercising control over the content of one's communication. Crossley (1994) described the factors that impact progress toward independence, including severity of movement and hand function difficulties, skill and experience level of facilitators, availability of facilitators, opportunities for regular use of facilitated communication, access to appropriate technology, types of activities and environments in which an individual is involved, and consistency of opportunities to practice more independent typing.

Treatment Participants

Use of FC is not restricted to any specific age or diagnostic group. People with a variety of diagnoses, including autism, Down syndrome, intellectual disability, cerebral palsy, and acquired brain damage, have used FC. The FCTS indicate that assessment is a key initial step in the process of identifying the potential benefit of FC for an individual. The following considerations are suggested for those attempting to engage individuals in a fluent conversational context: current communication strategies, current and past use of AAC strategies, independent pointing skills, history and current description of movement problems, and current and past use of support strategies. Individuals with limited or no speech should carefully consider what has been tried and what has been effective. It should compare independent skills with facilitated skills and have a rationale for the need for support through facilitation by the potential candidate. Other considerations include the use of other support strategies such as structuring communicative interactions, accommodating the environment, and using routines and scripts.

The FCTS also address the relationship between FC and AAC. In cases where other AAC strategies have been effective, facilitated communication training may be considered as an additional benefit. In cases where current communication strategies are ineffective and AAC strategies have not been tried, facilitated communication training may be recommended on the basis of significant and specific movement problems. "It is important to note that facilitated

communication training is not seen as a substitute for AAC approaches, but rather as a way of effectively and rapidly gaining access to a wider range of AAC than might otherwise be possible."

Treatment Procedures

Facilitated communication (FC) is a method designed to assist individuals with autism and related disabilities to communicate through the use of a typewriter, keyboard, or similar device. The technique involves a trained "facilitator" holding the disabled person's hand, arm, or shoulder while the person communicating types messages on the keyboard device.

Assessment guides intervention, and if it is determined that facilitated communication training (FCT) would benefit an individual, recommendations should be given through the assessment. Recommendations should include specific support strategies for dealing with hand function issues (e.g., the amount of resistance and pull back needed); specific support strategies for general movement issues (e.g., support for trunk positioning and eye gaze); minimum effective level of physical support (i.e., degree of fading achieved while allowing for reasonably accurate pointing); optimal positioning of the candidate and the device relative to each other; description of observed literacy skills; device options, with recommendations addressing both short-range and longer-range access issues; other supports necessary for successful communication (e.g., the commitment of the support team to create a positive environment, give consistent access, and allow time to practice); strategies that would enable the user to focus his or her attention on the task at hand; and plans for a continuing training process, including scheduling designed to optimize opportunities for effective practice for the candidate and his or her facilitators.

The facilitator can be a teacher, other professional, a family member, or a friend. The facilitator may offer physical, emotional, and/or communication support to the person using FC, with the type and amount of help needed varying from person to person. A facilitator's *physical support* may include isolating the index finger; stabilizing the arm to overcome tremor; providing backward resistance on the arm to slow the pace of pointing or to overcome impulsiveness; a touch of the forearm, elbow, or shoulder to help the person initiate typing; or pulling back on the arm or wrist to help the person not strike a target repetitively. Providing encouragement and motivation as the person types or points to communicate *are emotional supports that may be provided by the facilitator*. The facilitator's communicative support assists the FC user to stay focused on the communication interaction, provides feedback on the content of the message, and assists in clarifying unclear messages.

Facilitated communication training involves developing communication skills through pointing at pictures or (e.g., typing or pointing at pictures or letters) with a partner or facilitator. The facilitator provides physical support (e.g., holding the person's wrist or forearm during pointing, providing backward resistance as the person tries to move the arm forward to point, placing a hand on the shoulder as the person points or types). This support is helpful in overcoming such movement-related difficulties as tremor, impulsivity, low muscle tone, poor eye-hand coordination, and/or difficulty in forefinger isolation (Crossley, 1994).

"The goal of all programs designed to train facilitators is the same: to produce qualified, competent facilitators. This is accomplished through: an introductory information session; resource and information exchange; a continuum of beginner, intermediate and advanced training; direct supervision leading to indirect supervision; continuing education, and ongoing technical assistance" (FCTS).

Efficacy Information

Controversy about the validity of FC has accompanied the technique since the early 1990s, with negative research findings and media reports. Throughout the 1990s, many studies tried unsuccessfully to establish the validity of this

approach. One of the most basic concerns was over the authorship of the messages and how it is possible to determine the true source of a message that is constructed with the support of a facilitator. In fact, in most cases, researchers could not establish that the facilitator was not influencing the content of the message. Studies showed that "a facilitator's physical touch or support of the typist's hand or arm could negatively influence the person's pointing, even to the point of controlling the output" (Biklen). As a result, many professional organizations in the United States adopted formal position statements indicating that FC was not a valid method to support communication for individuals with disabilities. The American Academy of Pediatrics notes, "the potential for harm does exist, particularly if unsubstantiated allegations of abuse occur using FC. Many families incur substantial expense pursuing these treatments, and spend time and resources that could be used more productively on behavioral and educational interventions" (1998, p. 342). The Academy further advises "when controversial or unproven treatments are being considered by a family, the pediatrician should provide guidance and assistance in obtaining and reviewing information. The pediatrician should ensure that the child's health and safety, and the family's financial and emotional resources are not compromised" (1998, p. 342). In 1994, Sarah Blackstone concluded "People and institutions charged with protecting individuals with disabilities now have no alternative but to consider FC an experimental procedure."

In 2010, Mostert summarized the state of the literature related to FC. First, claims of FC efficacy were made under conditions where alternate explanations could not be ruled out. Second, despite empiricists' conclusion that FC was ineffective as an intervention, advocates dismiss these empirical investigations as either irrelevant or unnecessary and persist in their claims and beliefs that FC worked. Third, researchers have moved on to new topics rather than repeatedly replicating solid evidence around a settled empirical question. Fourth, newer pro-FC studies operate from the premise that FC works and is a legitimate practice. FC is presented as a valid intervention to readers who are unaware of the empirical dismissal of the intervention and who might not be skilled in distinguishing solid from suspect research. FC will continue to reinforce the assumptions of efficacy among parents and practitioners. Lastly, newer literature has been published in scholarly journals, which seems to enhance the credibility of FC and may be interpreted as valid research indicating that FC works. Mostert (2010) concludes by saying "Optimistically perhaps, empiricists assume that when any intervention is clearly demonstrated to be ineffective (or even harmful) and that its ineffectiveness is clearly communicated to the field, practitioners will usually implement more effective interventions. However, as with other suspect approaches throughout the history of special education, this is often a false assumption and certainly a problematic assumption regarding FC."

Beukelman and Mirenda (1998) do acknowledge that there is "a small group of people around the world who began communicating through FC and are now able to type either independently or with minimal, hand-on-shoulder support. There can be no doubt that, for them, FC 'worked,' in that it opened the door to communication for the first time."

Outcome Measurement

Documenting the progress of facilitated communication users and their facilitators over time is considered an important part of the intervention and is recommended as a part of the support plan (FCTS). The FCTS suggest that the following indicators be used to document validity of the intervention:

- Similar patterns of spelling and typographical errors across facilitators
- Typing of similar topics and themes across facilitators
- Consistent style of typing across facilitators
- Instances of independent and/or initiated communication
- Self-correction of mistakes

- Effective use of protest strategies
- Sharing of information not known to the facilitator
- Successful participation in message-passing exercises
- Behavior or actions that confirm typed communication
- Speech that correlates with typed communication
- Consistent physical style of typing across facilitators
- Physical attention to the typing (e.g., eye contact with the communication device)

The FCTS also outline Biklen and Cardinal's (1997) conditions for increasing the likelihood that facilitated communication users will be able to demonstrate their authorship:

- Extensive experience with facilitation by both facilitator and facilitated communication user
- Practice using multiple trials
- Consultation with facilitated communication user on test and format
- Familiar facilitators
- Monitoring for facilitated communication user's style
- No-risk or low-risk testing
- Building of confidence, with limits on opportunities to fail
- Naturally controlled conditions
- Ongoing feedback on performance
- Minimization of word retrieval tasks
- Presentation of information through multiple modalities
- Age appropriate content
- Personally relevant content
- Extensive time to respond to questions

The Institute on Communication and Inclusion at Syracuse University outlines certain methods for verifying the authorship of typed communication:

- *Video eye tracking* of the FC users' eye gaze to verify the individual's targets before any movement of the hand toward a target (Emerson, Grayson, & Griffiths, 2001).
- *Linguistic analysis* of individuals' typing, demonstrating that the individuals with disabilities employ significantly different patterns of word use and sentence construction than their facilitators and that they were different from each other even when they shared the same facilitator (Niemi & Karna-Lin, 2002; Tuzzi, 2009; Tuzzi, Cemin, & Castagna, 2004; Zanobini & Scopesi, 2001).
- *Evidence of speech before and during typing* (Broderick & Kasa-Hendrickson, 2001; Kasa-Hendrickson & Broderick, 2009).
- *Message passing* (Cardinal, Hanson, & Wakeham, 1996; Sheehan & Matuozzi, 1996; Weiss, Wagner, & Bauman, 1996). Each of these message-passing studies, where individuals demonstrate authorship, involves multiple sessions, with the possible effect of allowing participants to be desensitized to anxiety over the course of the study.

Qualifications of Treatment Providers

In 2000, the Facilitated Communication Training Standards were published. These standards contained the first comprehensive set of guidelines to outline best practices in FC, the framework for training and technical assistance, and facilitator competencies.

Potential facilitators must learn and understand the particular physical problems FC is intended to address. Practice is critical as facilitators need to learn how to monitor a person's eyes on the target, how to teach finger isolation, ways to pace skill learning toward independent typing, and how to fade physical support. It is important for facilitators to foster the individual's control over his or her typing and to avoid facilitator influence such as physical or verbal influence (e.g., completing the person's sentences, anticipating letters and words).

Sarah Blackstone (1994) identified some guidelines that she felt would help promote safe practices when using FC. The first recommendation was to get and give informed consent, whereby the facilitator acknowledges that the technique is controversial and identifies the specific types of risks (e.g., unfounded allegations of abuse). Secondly, she suggests that the user first have an augmentative and alternative

communication assessment and that other access options be considered. Thirdly, all facilitators should be trained and supervised. Fourth, establish and use a validation procedure to help ensure authorship of the message. And lastly, she recommends a balanced approach that considers all sides of the discussion and access to a range of related literature.

See Also

- ▶ Augmentative and Assistive Technology
- ▶ Nonverbal Communication
- ▶ Visual Supports

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Faciogenital Dysplasia

► Aarskog Syndrome

Fact Memory

► Explicit Memory

Factor Analysis

Thomas Frazier
Research, Center for Autism, The Cleveland Clinic, Cleveland, OH, USA

Definition

Factor analysis refers to statistical methods that attempt to represent a set of correlated observed variables (questionnaire items, autism symptoms, etc.) in terms of a smaller number of unobserved (latent) variables, also called factors or dimensions. The ultimate goal is to determine the number and composition of dimensions that can parsimoniously represent a larger set of variables.

Historical Background

Factor analytic methods began with Charles Spearman's observation that specific measures of children's academic performance and cognitive skills were correlated. He developed a theory that the correlations between different measures of ability were due to a single common factor or dimension that he called "g" or general intelligence. Subsequent uses of factor analysis attempted to identify whether multiple components of intelligence can be identified, such as verbal and nonverbal abilities. As factor analytic techniques have become more popular, use of these methods expanded to other sets of variables, including psychiatric symptoms. Factor analytic examinations of psychiatric symptoms have been particularly helpful, as they have assisted researchers in identifying the boundaries of psychiatric conditions and separating important core components of the disorders from minor or associated symptoms.

One of the earliest uses of factor analysis in autism was a study by Freeman and colleagues (1980) examining the factor structure of a behavior observation scale. Since, numerous studies have used factor analysis methods in samples of individuals with autism. A search of the term "factor analysis" in PubMed yield more than 250 hits and a search on the Journal of Autism and Developmental Disorder website yield more than 1,100 articles. The majority of studies examine autism and/or other behavioral symptoms, often using specific measures designed to assist in screening, diagnosing, or tracking of symptoms in individuals with autism. A recent example is a study by Pandolfi and colleagues (2010) examining a measure of Asperger's syndrome symptoms.

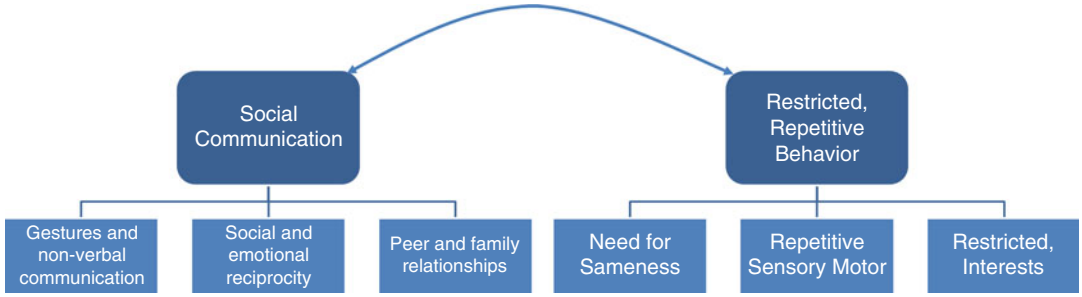
Examining the use of factor analysis in autism, it has become clear that the primary implementation has been to better understand which autism symptoms cluster together to form broader symptom domains. The other major use has been to evaluate how many dimensions are evaluated by a specific instrument, typically a measure of autism symptoms. Most measures do not include all autism symptoms or include some items that are not considered core parts of autism (e.g., self-injurious behavior on the Repetitive Behavior Scale). Thus, results of factor analytic studies examining specific instruments are mainly useful for scoring the instrument rather than understanding the core symptom domains to be considered in autism. The exceptions are factor analyses of measures which comprehensively cover autism symptoms, such as the Autism Diagnostic Interview-Revised. One other major observation when reviewing the use of factor analysis in autism is the wide variation in factor analytic procedures used across studies.

There are two general categories of factor analyses: exploratory and confirmatory factor analyses. The main difference in these categories is that, in exploratory analyses, variables are submitted with no pre-conceived notion of which variables will measure which hypothetical factor. In confirmatory factor analysis, prior to the analysis, the researcher specifies which variables will measure which factors and whether factors will be correlated. Researchers can be even more specific by explicitly fixing the level an item or symptom will load on a factor. Thus, confirmatory methods are more rigorous in that a researcher must have a hypothesis prior to conducting the analysis. Confirmatory methods are also useful in that multiple models can be specified that correspond to different hypotheses or theories. Direct comparisons of the fit of these models provide evidence of the relative plausibility of the hypotheses or theories. However, because not all possible models can be testing in confirmatory factor analyses, the better fitting model is not viewed as the truth but rather a better approximation of reality.

Both exploratory and confirmatory approaches consist of multiple methods for estimating the factor structure, including estimating model fit to the observed variables. Norris and Lecavelier (2010) provide an excellent overview of exploratory factor analytic methods and recommendations for their use in autism and other developmental disorders. Tabachnick and Fidell (2007) provide a very accessible introduction to both exploratory and confirmatory methods. Although principal components analysis is not strictly a factor method, results are often highly similar and this method is included when discussing previous factor analytic studies of autism.

Current Knowledge

Factor analytic methods have helped to clarify the nature of autism symptoms. Although there is not perfect agreement across studies, a consensus has emerged as to the number and composition of autism symptom dimensions. A useful starting place is to examine the DSM-IV-TR criteria for Autistic Disorder. These criteria include three symptom domains – social interaction; communication; and restricted, repetitive, stereotyped patterns of behavior. Most empirically sophisticated studies have identified two major dimensions of autism symptoms – social communication difficulties and restricted, repetitive behavior (see Fig. 1)



Factor Analysis, Fig. 1 The two primary symptom domains measured by more specific facets of social, communication, and stereotypic behavior are shown. The *double headed arrow* indicates that greater social communication difficulties tend to be associated with greater restricted, repetitive behavior

(Frazier, Youngstrom, Kubu, Sinclair, & Rezai, 2008; Snow, Lecavalier, & Houts, 2009; van Lang, 2006). These analyses have focused on DSM-IV-TR symptoms using the Autism Diagnostic Interview-Revised. The social communication dimension merges social interaction and communication symptoms and is more parsimonious than positing separate factors. In hindsight, this is not surprising because social symptoms (deficits in social reciprocity, relationships, shared enjoyment, etc.) are highly correlated with communication symptoms (deficits in nonverbal communication, reciprocal conversation, etc.). The second dimension, restricted/repetitive behavior, includes all of the symptoms previously identified in this domain in the DSM-IV-TR but also includes repetitive vocal behavior (idiosyncratic speech/neologisms, echolalia, inappropriate questions, etc.).

A few studies examining the Social Responsiveness Scale, a quantitative trait measure of autism symptoms, have identified only a single autism symptom factor with predominantly social difficulties represented (Constantino et al., 2004; Constantino & Gruber, 2005). This discrepancy between a single symptom severity model and the model in Fig. 1 is primarily due to the predominance of social indicators examined on the Social Responsiveness Scale and the focus on population samples rather than mixed clinical or autism-only samples. Specifically, the discrepancy between a single autism factor and separate social communication and restricted/repetitive dimensions is at least partly due to the fact that, in population samples, the majority of healthy individuals will show less variation in symptom levels while a small minority of mostly affected individuals will show very large variations in symptom levels. This pattern exaggerates correlations among social communication and restricted/repetitive behavior symptoms, resulting in a single symptom severity factor. Furthermore, the Social Responsiveness Scale has only a single scale (12 items) evaluating restricted/repetitive behaviors, decreasing the ability to identify a second dimension. However, it should be noted that, in spite of these differences, factor analyses have still tended to identify a smaller repetitive behavior factor in addition to the major social interaction and communication dimension.

Still other studies have found more than two factors. However, these additional factors are often minor and age- or sample- specific. For example, in a study by Frazier and colleagues (2008), a third factor that represented peer relationships and play was identified. This factor accounted for very little variance in the observed symptoms and did not improve fit beyond a two-factor model such as that in Fig. 1 above. Other studies have identified specific factors related to social motivation, unusual language, or level of functioning (Tadevosyan-Leyfer et al., 2003; Szatmari et al., 2002), but these specific factors are often highly correlated with broader social communication or restricted/repetitive behavior factors or are measured by a few items.

Only a few studies have examined whether sub-factors might exist within broad autism symptom dimensions. These studies have identified three major sub-domains of restricted/repetitive behavior, including repetitive sensory motor behavior, need for sameness, and restricted or circumscribed interests (Lam et al., 2007; Lam, Bodfish, & Piven, 2008; Cuccaro et al., 2003). However, these investigations have typically excluded sensory sensitivity symptoms, which may represent an additional sub-dimension of restricted/repetitive behavior.

Because factor analyses assume one or more latent dimensions, they cannot examine whether a set of autism symptoms are best represented by subgroups of autism cases. A related set of latent variable models and procedures are able to examine this question. These models include taxometric procedures, cluster analyses, and latent class analyses. A small number of studies have applied these procedures. A recent study used taxometric, latent factor, and latent class analyses to examine the structure of autism symptoms (Frazier et al., 2010). This study found that autism was a qualitatively distinct category that encompassed all of the DSM-IV-TR disorders and had a broad range of symptom severity from moderate to very high symptom levels.

Other studies have examined whether subgroups of autism cases can be identified using taxometric, cluster, and latent class analyses (Ingram, Takahashi, & Miles, 2008; Prior et al., 1998; Stevens et al., 2000; Verte et al., 2006). Two of these studies found three clusters that included high symptom levels, moderate symptom levels, and low symptom levels, roughly corresponding to the clinical conceptualizations of Autism, PDD NOS, and Asperger's disorder. However, both studies consisted of small sample sizes and were limited by the use of cluster analyses which lack well-validated decision rules for determining the number and composition of clusters. Additionally, the composition of the clusters suggested arbitrary grading of symptom severity rather than qualitatively distinct groups. Similarly, a third study using cluster analysis found only two groups differing in symptom severity. Finally, a recent study using taxometric procedures identified a subgroup of autism characterized by physical abnormalities such as large head circumference (Ingram et al., 2008). This study also found a distinction between autism cases based on social communication difficulties, but it was not clear if this identified a unique subgroup of autism or actually distinguished autism from non-autism cases.

Factor analytic methods have been helpful for reducing the large number of specific symptoms seen in individuals with autism to a smaller number of core dimensions. Related latent variable models and other procedures have been helpful for determining whether a specific category or subcategories of autism exist. Answers to these questions can and have guided screening and assessment and assisted in developing parsimonious diagnostic criteria that capture the core behaviors observed in autism-affected individuals.

Future Directions

Prospective factor analytic studies are needed that examine the hierarchical structure of the newly developed DSM-5 criteria. Factor analytic methods could also be expanded to better understand underlying neurobiological abnormalities. For example, factor analytic methods may be useful for reducing complex EEG signals to a smaller set of dimensions that correspond with autism symptoms and behaviors. Additionally, studies using latent methods for examining categories may be helpful for determining whether autism subgroups can be identified. Newly developed factor mixture models may be particularly powerful in this regard because they permit simultaneously including factors and categories.

Future work using factor analyses and related methods would benefit from using empirically supported approaches for determining the number and composition of dimensions. It is clear that a large number of previous studies did not use empirically recommended approaches to conducting analyses. In future work, it will be important that researchers follow general

recommendations for implementing both exploratory and confirmatory methods (McCutcheon, 2002; Muthén, 2001; Norris et al., 2010; Ruscio, Haslam, & Ruscio, 2006).

See Also

- ▶ Latent Variable Modeling
- ▶ Statistical Approaches to Subtyping

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Factor Mixture Modeling

► Latent Variable Modeling

Factors Affecting Outcome

► Course of Development

Factors Affecting Outcomes

Patricia Howlin and Sarah Savage
Institute of Psychiatry, King's College of London, London, UK

Definition

The outcome for individuals with autism is very variable and depends not only on the characteristics of the individual but on family factors and external support systems. Childhood factors that are associated with later outcome include early language development, IQ, severity of autism symptoms, and access to early, autism-specific intervention. In adolescence and adulthood, the onset of comorbid mental health problems and lack of adequate support and interventions are associated with poorer outcomes.

Historical Background

It has been evident since the very earliest outcome studies of Kanner (1973), Eisenberg (1956), Rutter, Greenfeld, and Lockyer (1967), Lockyer and Rutter (1969), and others that outcome in autism is highly variable. Many individuals remain highly dependent on support throughout their lives; others are able to live independently and some go on to make significant achievements. Asperger (1944) was particularly impressed by the wide variation in outcome noting that "One might expect. . .that social integration of autistic people is extremely difficult if not impossible. . .this bleak expectation, however, is born out only in a minority of cases and, in particular, almost exclusively in those people with considerable intellectual retardation in addition to autism. . .This is not so with intellectually intact autistic individuals." Indeed, he suggested that among this latter group, "able autistic individuals can rise to eminent positions and perform with such outstanding success that one may even conclude that *only* such people are capable of certain achievements."

The factors affecting outcome and the reasons why some individuals are able to make very good progress while others continue to have significant problems throughout their lives has been the focus of considerable research over the years.

Current Knowledge

Although many follow-up studies have indicated a relatively poor outcome for individuals with autism, the results of such research cannot be interpreted without detailed information on the characteristics of the individuals involved. Kanner (1943) and Asperger (1944) were among the first to note a possible association between higher intellectual ability and greater achievements in adulthood. Subsequent follow-up studies have confirmed that childhood IQ is highly correlated with adult outcome. For example, in a series of studies conducted in the UK in the late 1960s and early 1970s, Rutter and colleagues (Lockyer & Rutter, 1969; Rutter & Lockyer, 1967; Rutter et al., 1967) found that individuals with an IQ below 60 were far more likely to have a "poor" or "very poor" outcome than those with an IQ above 60. Individuals with an IQ of at least 70 (i.e., at the lower end of the normal range) tended to be rated as having a "fair" outcome, while those with a "good outcome" had an average IQ above 80. More recent studies, involving individuals across the IQ range (i.e., from severe cognitive impairment to superior intelligence) have also noted a strong association between IQ and outcome, with very few individuals with a childhood IQ

below 75 living independently as adults (e.g., Eaves & Ho, 2008; Farley et al., 2009; Gillberg & Steffenberg, 1987; Howlin, Goode, Hutton, & Rutter, 2004). However, childhood IQ alone is not the only factor affecting outcome, and many individuals with a high IQ in childhood still do poorly in adulthood. Thus, it seems that while *only* individuals with a childhood IQ of at least 70–75 IQ do well in adulthood, even among those with an IQ above 70, outcome can still vary markedly (Howlin et al., 2004). The range of early cognitive abilities may also be important, in that children who are able to score only on nonverbal tests, or on a very limited range of subtests – even if their scores on these assessments are within the normal range – tend to have a poorer prognosis than those able to score on both verbal and nonverbal tests in early childhood.

Another crucial indicator of later outcome is early language development. Ever since the earliest follow-up studies of Rutter and his colleagues, it has been well established that there is a strong link between early language abilities and subsequent development (Howlin, Mawhood, & Rutter, 2000, 2004; Szatmari, Bartolucci, Bremner, Bond, & Rich, 1989; Venter, Lord, & Schopler, 1992). Thus, the majority of individuals who go on to do well as adults have usually developed at least some useful speech by the age of 5 years. Individuals who do not develop speech until after 5 years of age tend to have a much poorer outcome. However, there are exceptions to this, and some children who show significant delays in language do later catch up and may make good progress more generally. Overall, however, language impairments, particularly if coupled with low IQ, are the factors most strongly associated with a poorer adult outcome. In adulthood, individuals with good verbal comprehension, good spoken language, and a verbal IQ in the normal range are significantly more likely to be functioning well socially than those who are impaired in these areas.

There also appears to be an association between the severity of early autistic symptomatology and later outcome, although findings are somewhat contradictory. Thus, while some researchers have found that *total* symptom severity scores are predictive of later functioning (e.g., Rutter et al., 1967; DeMyer et al., 1973), others suggest that it is the severity of early repetitive and stereotyped behaviors that is crucial (e.g., Venter et al., 1992) and still others indicate that the level of impairment in the social domain is the strongest prognostic indicator (Kamp-Becker, Ghahreman, Smidt, & Remschmidt, 2009). Although a number of studies now confirm that the severity of autistic symptoms tends to reduce with age, the overall level of autistic symptomatology in adulthood continues to affect social and economic independence and several researchers have noted that inappropriate and/or ritualistic and stereotyped behaviors are negatively correlated with employment status, the ability to develop close relationships, and general level of social functioning (e.g., Howlin et al., 2004; Rumsey, Rapoport, & Sceery, 1985).

The presence of mental health problems can also have a very negative impact on outcome. Several studies have confirmed the high rates of mental health problems in individuals with autism from adolescence onward. It is estimated that at least one third of individuals with autism suffer from emotional and psychiatric problems, mainly related to anxiety and depression, and these can have a very negative impact on general functioning as well as proving difficult to treat effectively (Ghaziuddin & Zafar, 2008; Lainhart, 1999; Stewart, Barnard, Pearson, Hasan, & O'Brien, 2006).

The presence of an additional comorbid condition, such as ADHD, is also associated with increased social and behavioral difficulties. Medical disorders, particularly epilepsy, also have a significant impact on outcome. Epilepsy occurs in around 20% of individuals with autism, but tends to be more common in individuals with lower IQ and poor language skills, factors that, of themselves, are also linked to poor outcome, so the impact of the epilepsy, per se, is difficult to judge (Bolton et al., 2011).

The association between outcome and age has still to be systematically explored. Although it is evident that overall severity of autism symptoms tends to reduce between early childhood and

early adulthood, there are no studies of the trajectories of change in older individuals. Thus, we have almost no information on whether this diminution in symptom continues across the life span or whether there may be increases in difficulties at some later stage of life.

In the above discussion, the focus has been on the impact of internal factors associated with outcome. However, external, environmental factors will also play a significant role throughout life. It is evident, for example, that the onset of mental health difficulties is strongly associated with environmental disturbance, such as the transition from school, difficulties in coping with college work or employment, family disruption and loss, change and unpredictability in daily life, and a host of other emotional stressors (Hutton, Goode, Murphy, Le Couteur, & Rutter, 2008). It is well established that social support networks, especially for individuals of higher IQ who are not eligible for help from Intellectual Disability Services, are often grossly inadequate and there is some evidence that lack of appropriate support in adulthood has a much greater impact on outcome than factors such as the individual's IQ (Venter et al., 1992).

It is also highly likely that the quality of early intervention, access to appropriate education programs, and support for families will have a significant impact on later life. Indeed, some proponents of highly intensive behavioral therapies suggest that the initial high cost of treating preschool children will be readily recouped as these children and their families will subsequently require no additional specialist help (Jacobson, Mulick, & Green, 1998). Unfortunately, there is no evidence for the long-term effects of early intervention programs, as the follow-up period for such studies is limited to 2–3 years at most. Thus, although they may have a significant short-term impact, the longer-term impact remains unknown.

Future Directions

Long-term follow-up studies over the past 50 years have taught us a great deal about the factors that are associated with outcome in adulthood. However, many issues still remain to be resolved. Thus, although there is good evidence that language, IQ, and severity of autism are all linked to longer-term prognosis, we still know relatively little about how these factors interact, or how far environmental, genetic, family, social, racial, or other variables may ameliorate or exacerbate their impact. The role of temperament and personality is almost unexplored, and almost nothing is known about factors that influence functioning in the later years of life.

Research into effective treatments has led to major improvements in early intervention for many children. However, if and for how long the effects of such interventions endure remains unknown, and much longer-term follow-up studies are needed if we are to judge the true benefit of these (often very costly) programs.

In summary, although research into children with autism has made significant strides forward over recent years, leading to major advances in diagnosis and understanding of causes, research into adulthood still has a long way to go. Follow-up studies of individuals identified and fully assessed as children and followed up periodically from decade to decade are essential if we are to understand the factors that influence prognosis and how these factors exert their effects.

See Also

- ▶ Adulthood, Transition to
- ▶ Asperger Syndrome Follow-Up Studies
- ▶ Employment
- ▶ Employment in Adult Life
- ▶ Factors Affecting Outcome
- ▶ Living Arrangements in Adulthood
- ▶ Psychotic Disorder

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Fading

Thomas Zane[1], Traci Lanner[2] and Michelle Myers[2]
[1]The Institute for Behavioral Studies, Endicott College, Beverly, MA, USA
[2]The School at Springbrook, Oneonta, NY, USA

Synonyms

Prompt fading; Stimulus fading

Definition

Fading is the process of reducing assistance (i.e., cues, prompts, supports) until no longer needed so that the skill or response being taught is exhibited independently. In behavioral instruction, fading generally refers to *gradually* removing supports put in place during training programs so that the target behavior eventually occurs independently. For example, when

teaching a child with autism how to spell his name, the teacher starts with stating each letter out loud and having the child repeat them until he does so without error. The adult then instructs the child to spell his name and starts by saying only the first letter out loud and then only "mouthing" (silently moving lips) the remaining letters using no voice. Eventually, the teacher asks the child to spell his name, and he does so without additional prompts. The removal of the verbal prompts illustrates the process of fading.

See Also

- ▶ Prompt Fading
- ▶ Prompts

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FAFSA

Ernst VanBergeijk
Vocational Independence Program, New York Institute of Technology, Central Islip, NY, USA

Synonyms

Financial aid; Scholarships

Definition

FAFSA is an acronym which stands for Free Application for Federal Student Aid. FAFSA is the mechanism by which the government distributes grants, loans, and student work study funds. More specifically, it is the gateway by which college students and their families access Federal Pell Grants, Stafford and Perkins Loans, Federal Supplemental Education Opportunity Grants, and Federal Work-Study program funds. By completing the FAFSA form, families learn what their expected family contribution (EFC) will be. The EFC is the amount of money the family is expected to contribute to the student's college education. Prior to 2008, only students enrolled full time in a degree-bearing program at an Institution of Higher Education (IHE) were eligible to complete the FAFSA application.

With the passage of the Higher Education Opportunity Act (HEOA) of 2008 (P.L. 110–315), the regulations governing financial aid and the FAFSA process changed to make it easier for students with intellectual disabilities (ID) to gain access to postsecondary education. The term ID is broadly defined and can include students with autism spectrum disorders or cognitive impairments. According to HEOA, a student with an intellectual disability is one who has mental retardation or a significant cognitive impairment *and* is or was eligible for a Free and Appropriate Public Education (FAPE) under the Individuals with Disabilities Education Act (IDEA). This can include students who were home schooled or attended private schools. The college is responsible for the determination and supporting documentation of the student's ID (Bergeron, Murray, & Shanley, 2010).

In order for a student with an ID to be eligible, he or she must be enrolled in an approved Comprehensive Transition and Postsecondary Program (CTP). He or she also must meet all of the general student eligibility requirements *except*: (1) he or she does *not* have to be enrolled for the purpose of obtaining a degree or certificate; (2) he or she is *not* required to have a high school diploma or have passed an ability-to-benefit

test; and (3) he or she must maintain satisfactory academic progress under school's policy for students in the CTP (Finkel, Anderson, Daley, & Shanley, 2010).

After a public commentary period, the U.S. Department of Education began accepting applications from IHE, for approval of their Comprehensive Transition Programs (CTP) in the latter part of 2010. An eligible CTP must be offered by a college that already has an established financial aid program for its general student body. It also must be designed to support students with intellectual disabilities (ID) and include an advising and curriculum structure. The CTP must require students with ID to participate in courses and activities with students without disabilities (Bergeron et al., 2010).

Students with ID will be eligible for Federal Pell Grants, Federal Supplemental Education Opportunity Grants, and Federal Work-Study programs funds only (Finkel et al., 2010). It is important to note that students with ID currently are not eligible for Perkins or Stafford loans.

U.S. DOE approved CTPs are listed at the National Coordinating Center and include the Consortium for Postsecondary Education for Individuals with Developmental Disabilities and the Center for Postsecondary Education for Individuals with Intellectual Disabilities.

See Also

- ▶ Comprehensive Transition Program
- ▶ Free Appropriate Public Education
- ▶ Individuals with Disabilities Education Act (IDEA)
- ▶ Transition Planning

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Free Application for Federal Student Aid. http://www.fafsa.gov

Heath Resource Center: Online clearinghouse on post secondary education for individuals with disabilities. New postsecondary programs for students with intellectual disabilities.

Student Aid on the Web (http://www.studentaid.ed.gov, www.studentaid.ed.gov) – The U.S. Department of Education's federal student aid information website.

http://www.heath.gwu.edu/

False Positive

Samantha Huestis
Yale Child Study Center, New Haven, CT, USA

Synonyms

Alpha (α) error; Type I error; Wrong decision

Definition

A false positive occurs when the null hypothesis is true and rejected erroneously. That is, a difference is observed when there is no true difference. An individual may be diagnosed with a condition, for example, that he does not have. A test that produces a substantial number of false positives (i.e., Type 1 Errors) is unable to systematically rule out disorders and diagnoses, and consequently has poor specificity.

See Also

- ▶ Sensitivity and Specificity

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False-Belief Task

Nirit Bauminger-Zviely
School of Education, Bar-Illan University, Ramat-Gan, Israel

Definition

False-belief task is based on false-belief understanding which is the understanding that an individual's belief or representation about the world may contrast with reality. False-belief task is a frequently used methodology to examine theory of mind (i.e., child's ability to construct people in terms of internal mental states such as their beliefs, Wellman, 1993). It is considered as litmus test of theory of mind, in that in such cases, it becomes possible to distinguish unambiguously between the child's (true) belief and the child's awareness of someone else's different (false) belief (Dennett, 1978). First-order false-belief tasks involve attribution about other's false belief with regard to real events; whereas, second-order false-belief tasks are related with what people think about other people's thoughts. In second-order false-belief tasks, the child is required to attribute the false belief of one person based on the thoughts of another (Perner & Wimmer, 1985). An example of a commonly used first-order false-belief task is the "Smarties," in which the child is required to predict another child's perception about the content of a box of candies (that actually includes a pencil) (Gopnik & Astington, 1988). A commonly used second-order false-belief task is the Perner and Wimmer (1985) "ice-cream van story" (or John and Marry tasks). In this story, Marry is asked to predict John's thoughts about the location of the ice-cream man. Only Marry knows the actual location (in the school), but John knows about his former intention to stay in the park. Thus, Marry needs to take into consideration John's thoughts about the location of the ice-cream man, based on the ice-cream man's original intentions.

See Also

▶ Theory of Mind

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Family Burden

Peter Doehring
Foundations Behavioral Health, Doylestown, PA, USA

Definition

The physical, emotional, and economic impact directly associated with the responsibility of

caring for a person with ASD, including the secondary impact on all family members. These can include: (a) the economic cost of care (Montes & Halterman, 2008), including lost opportunities for income (Jabrink, 2007); (b) quality of life (Lee, Harrington, Louie, & Newschaffer, 2008; Khanna, Madhavan, Smith, Patrick, Tworek, & Becker-Cottrill, 2010); and (c) the impact on siblings (Harris & Glasberg, 2003). Unlike the typical costs associated with child rearing, this burden often extends – and may increase – into adulthood, for those families who elect guardianship. It is reasonable to expect that this burden may vary as a function of the family's race, ethnicity, income, and education, given other findings suggesting disparities in accessing health services, and cultural differences in attitudes toward disability. It is generally believed that various types of supports, including parent training, respite care, and wraparound services, can mitigate some aspects of the family burden.

See Also

- ▶ Family-Centered Care
- ▶ Guardianship
- ▶ Health Disparities
- ▶ Parent Training
- ▶ Respite Care
- ▶ Wraparound Services

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Family Therapy

Guus van Voorst
Clinical Psychology, Center for Autistic Disorders, GGZ Centraal, Amersfoort, Amersfoort, Netherlands

Definition

Family-based treatments attempt to decrease interactions between family members that contribute to psychiatric disorders and to increase interactions that protect them from these problems. The psychoeducational model is most commonly used in conjunction with psychiatric disorders. Family life is all about relationships and communication. Autism spectrum disorders are about communication challenges, misunderstanding of social cues, and lack of emotional understanding, thus affecting relationships in families. The family burden for parents and siblings is very high in families with a member on the spectrum, and the divorce rate is also high in families with children who have autism. In marriage, if one of the partners is on the spectrum, there will be more problems than just the normal marriage conflicts.

Collaborative family work is directed to meet the needs of people with autism and their families and should address problems in each developmental phase of the family life cycle. Parents of a young autistic child are perplexed when they realize that the child is not developing properly and may feel rejected or unimportant when the child does not seek their attention. Getting a diagnosis and services may be stressful, as well as the process of adaptation: accepting the diagnosis and informing other

family members and friends. At school age, the family has to adapt in many ways: confrontation with outside world, dealing with aversive reactions of peers, specific ways of rearing, arranging for child care, and dealing with professionals. In adolescence, families have to cope with the chronicity of the child's disability, with issues of aggression, odd behavior, sexuality, and addiction. Parents have to delay the developmental phase of launching and moving on and are confronted with the perspective of endless care.

Marriages with an autistic member have a serious risk of running into trouble. The spouse with ASD misses relational skills, there will be an asymmetry in the relation, and the non-ASD spouse will feel lonely and neglected.

A diagnosis of an autism spectrum disorder is likely to be experienced as ambiguous loss in a family, because resolution of the situation is not possible and the outcome is not predictable, especially when the individual with ASD may give an outward appearance of health, thus raising too high expectations for his functioning.

Sometimes, families have several autistic members, for instance, one or two siblings, or a father and a son both have the condition, complicating family function and rearing practices even more.

Family interventions have been less effective in reducing core ASD symptoms, yet they do contribute to reducing the comorbid family and behavior problems associated with the disorder in children and adolescents. A biopsychosocial approach with a treatment package (e.g., family treatment, behavioral therapy, and pharmacology) is common in most clinical settings. Some models and schools of family therapy should not be applied in families with an autistic member, because they focus primarily on context, meaning, and mentalizing processes. Moreover, family and couples' therapists should adapt their style, taking into account that family members may have problems with imagination, generalizing, and they need basic knowledge about ASD in order to work effectively with these families.

Historical Background

For decades following Kanner's description of the disorder, it was wrongly believed that the parents, especially the mother, were responsible for causing the disorder by emotional neglect. Family psychoeducation started in the 1970s of last century in the treatment of schizophrenia. It was based on the premise that the patient has a brain disorder and that families need to be supported in their care of the mentally ill person. The emphasis on the biological aspect of the illness is intended to correct that families somehow cause the illness.

Family psychoeducation nowadays is commonly used in various chronic childhood and adult illnesses.

Current Knowledge

Nowadays, the association of genetic factors in the etiology of autism spectrum disorders is well established. Problems in parenting and family dysfunction may be a response to the child and adolescent psychopathology. A psychoeducational approach in families is the treatment of choice in clinical practice. It is defined as the systematic administration of information to both patient and family (significant others) about symptoms, diagnosis, treatment, and prognosis. Its aim is behavioral change and not just teaching for the sake of increasing knowledge. It must be given in doses, as the recipients are able to hear it and given repetitively over time. Other goals are improving family skills, positive communication development, and increased social involvement for the family. Family psychoeducation includes the provision of emotional support and resources during periods of transition and crisis.

Future Directions

Early diagnosis and treatment of the child and its family may have positive effects for some

children. There is no cure for ASD, yet various treatments are used to treat the effects of the disorder.

See Also

▶ Family Burden

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Family-Centered Care

Kelly Macy
Department of Communication Sciences, The University of Vermont, Burlington, VT, USA

Definition

Family-centered care is a set of beliefs and practices recognizing that the family is a child's primary source of strength and support and that the values and beliefs of the family are an important component of clinical and educational decision making. It is an approach to health care and education that shapes policies and programs, requiring collaboration among children, families, therapists, medical professionals, and educators to best support the education and health care of the child. The key elements of family-centered care were not formally defined until the late 1980s (Shelton & Stepanek, 1994). While it is important for professionals to appreciate that all families look and function differently, it is also important to remember that all families have a range of strengths, beliefs, emotions, and goals beyond their need for specialized health-care and educational services.

Family-centered care is especially relevant to children with autism and other special health needs since the family is the constant in the child's life and can provide important perspectives and information to the numerous team members and providers that transition in and out of a child's life. In order for professionals to make informed and appropriate decisions regarding assessment and intervention, they need to foster a relationship with the family and recognize the

dynamic connection between the needs of the child and the culture and values of the family. When professionals can recognize and describe the families' strengths, they can help to support effective service delivery for a child with autism (Prelock & Beatson, 2006).

Research has shown that the implementation of family-centered care results in improved outcomes for families of children with special needs and disabilities, which include better health outcomes, better allocation of resources, decreased costs, and increased family involvement, satisfaction, and coping (American Academy of Pediatrics Committee on Hospital Care, 2003).

See Also

- ▶ Culture and Autism
- ▶ Interdisciplinary Team

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Family-Centered Programming

- ▶ Parent-Professional Partnership

Fanatrex

- ▶ Gabapentin

FAST

- ▶ Functional Analysis Screening Tool
- ▶ Functional Assessment Screening Tool (FAST)

FAS-Test

- ▶ Verbal Fluency

FazaClo

- ▶ Clozapine

FBA

- ▶ Functional Behavior Assessment

Fear

- ▶ Anxiety

Febrile Convulsions

Jennifer M. Kwon
Department of Neurology & Pediatrics (SMD), University of Rochester, School of Medicine and Dentistry, Rochester, NY, USA

Synonyms

Febrile seizures

Definition

Febrile convulsions are a common type of "provoked" seizure seen in children between the ages of 6 months and 6 years. (Other kinds of provoked seizures include seizures following head trauma or withdrawal from medication.) Febrile seizures occur in the setting of a fever (temperature >38°C) but with no evidence of encephalitis or meningitis or prior history of afebrile seizures. Children who have febrile seizures typically have an acute intercurrent viral or bacterial illness such as an ear infection or sinus infection. The seizures are not associated with any systemic metabolic abnormalities such as hypoglycemia.

Febrile seizures are commonly categorized as simple or complex. Simple febrile seizures last less than 15 min and have no focal features. Two or more simple febrile seizures can occur in succession, but the total duration of the seizures should be less than 30 min. A "complex" febrile seizure is one that has focal features, may last more than 15 min, or, when they occur in a series, may last more than 30 min.

See Also

- ▶ Electroencephalogram (EEG)
- ▶ Epilepsy
- ▶ Seizures

References and Readings

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Febrile Seizures

▶ Febrile Convulsions

Fecal Impaction

▶ Constipation

Fecal Incontinence

▶ Encopresis

Federal Rules of Evidence

Edward McNulty
Quinnipiac University School of Law, Hamden, CT, USA

Definition

In General

The Federal Rules of Evidence are the rules governing the admissibility of evidence at civil and criminal trials in federal courts. The Federal Rules of Evidence generally comprise a set of restraints that the courts place on lawyers in an attempt to manage the risks associated with the adversarial process in a trial setting. The Federal Rules of Evidence were adopted in 1975, and as of 2009, 42 states had adopted codes based on the federal model.

Admission of Relevant Evidence

Federal Rule of Evidence 402 provides for the admissibility of relevant evidence unless the rule provides otherwise. Federal Rule of Evidence 403 gives the trial judge discretion to exclude relevant evidence where its probative value is substantially outweighed by the danger of unfair prejudice, confusion of the issues, or misleading the jury, or by considerations of undue delay, waste of time, or needless presentation of cumulative evidence. Because exclusion occurs only where the probative value is "substantially outweighed," the rule favors admissibility.

Witness

Federal Rule of Evidence 601 serves the purpose of removing a considerable number of the common law grounds for disqualifying witnesses. In particular, a section of Federal Rule of Evidence 601 states that "[e]very person is competent to be a witness except as otherwise provided in these rules." Federal Rule of Evidence 602 requires that before a witness can testify, evidence must support that the witness has personal knowledge and memory of the matter. Thus, one may not testify to knowledge obtained from what others told them.

Expert Witnesses

Under Federal Rule of Evidence 702, only experts may testify on matters that are scientific, technical, or specialized in nature. In *Daubert v. Merrell Down Pharmaceuticals*, the Supreme Court held that (1) "general acceptance" is not necessary precondition to admissibility of scientific evidence under Federal Rule of Evidence and (2) under the Federal Rules of Evidence, the trial judge is assigned the task of ensuring that any and all scientific testimony or evidence admitted is not only relevant but reliable. In *Daubert* and similar cases, the United States Supreme Court set forth a number of factors to consider in determining whether to admit expert testimony under Federal Rule of Evidence 702. These factors are neither exclusive nor dispositive. For instance, the US Supreme Court stated in *Daubert* that courts may consider whether the theory or technique employed by the expert is generally accepted in the scientific community, whether it has been subjected to peer review and publication, whether it can be and has been tested, whether the known or potential rate of error is acceptable, and the existence and maintenance of standards and controls.

Sources

Daubert v. Merrell Dow Pharmaceuticals, Inc., 509 U.S. 579 (1993).

Federal Rules of Evidence (As amended to December 1, 2011)

Federal Rule of Evidence 402. General Admissibility of Relevant Evidence

Relevant evidence is admissible unless any of the following provides otherwise

- the United States Constitution
- a federal statute
- these rules; or
- other rules
- prescribed by the Supreme Court.

Federal Rule of Evidence 403. Excluding Relevant Evidence for Prejudice, Confusion, Waste of Time, or Other Reasons

The court may exclude relevant evidence if its probative value is substantially outweighed by the danger of one or more of the following: unfair prejudice, confusing the issues, misleading the jury, undue delay, wasting time, or needlessly presentating of cumulative evidence.

Federal Rule of Evidence 601. Competency to Testify in General

Every person is competent to be a witness unless these rules provide otherwise. But in a civil case, state law governs the witness's competency regarding a claim or defense for which state law supplies the rule of decision.

Federal Rule of Evidence 602. Need for Personal Knowledge

A witness may testify to a matter only if evidence is introduced sufficient to support a finding that the witness has personal knowledge of the matter. Evidence to prove personal knowledge may, consist of the witness' own testimony. This rules does not apply to a witness's export testimony under rule 703

Federal Rule of Evidence 702. Testimony by Expert Witnesses

A witness who is qualified as an expert by knowledge, skill, experience, training, or education may testify in the form of an opinion or otherwise, if: (a) the expert's scientific, technical, or other specialized knowledge will help the trier of fact to understand the evidence or to determine a fact in issue (b) the testimony is based on sufficient facts or data; (c) the testimony is the product of reliable principles and methods; and (d) the expert has reasonably applied the principles and methods to the facts of the case.

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Feedback Error-Related Negativity (fERN)

▶ Feedback-Related Negativity

Feedback on Provider Work Performance

S. Michael Chapman
TEACCH Autism Program, University of North Carolina Chapel Hill, Chapel Hill, NC, USA

Synonyms

Employee development; Employee performance; Performance reviews for direct care staff; Staff training and development

Definition

Refers to the various methods used to encourage personnel working with individuals with autism spectrum disorders to improve the strategies they use in their everyday interactions, teachings, and supports they provide. Though various methods have been tested (Brown, Willis, & Reid, 1981; Quilitch, 1975), it was found that the most successful method for increasing work performance was a combination of feedback and praise based upon the ability of the staff member to assist the individual under their care meet their specific goals/outcomes (Kreitner, Reif, & Morris, 1977; Montegar, Reid, Madsen, & Ewell, 1977).

See Also

▶ Individuals with Disabilities Education Act (IDEA)

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Feedback-Related Negativity

Michael J. Crowley
Developmental Electrophysiology Laboratory, Yale Child Study Center, New Haven, CT, USA

Synonyms

Feedback error-related negativity (fERN); Medial frontal negativity (MFN)

Definition

The feedback-related negativity or FRN is an electrical brain signal measured with an electroencephalogram. Detectable at the scalp via the event-related potential (ERP), the FRN occurs when an individual receives external feedback (visual, auditory) indicating that performance is worse than expected in a given context. Such contexts include monetary loss and feedback about performance in simple games. The FRN occurs approximately 250 ms after feedback and is typically observed at central to frontal-central scalp regions. The most likely neural generator of the FRN is the anterior cingulate cortex (Gehring & Willoughby, 2002; Luu, Tucker, Derryberry, Reed, & Poulsen, 2003).

See Also

- ▶ Anterior Cingulate
- ▶ Cingulate Cortex
- ▶ ERN
- ▶ Error-Related Negativity

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Feeding Disorder

- ▶ Pica

Feeding Problems

Allison Bean
Speech and Hearing Science, The Ohio State University, Columbus, OH, USA

Definition

Feeding problems are delays and/or disorders in the development of eating and drinking skills including disordered placement of food in the mouth, difficulty in appropriately manipulating food when it is in the mouth, difficulty chewing, and/or difficulty swallowing (American Speech-Language-Hearing Association, 2001). Feeding problems also include deficits in "any aspect of taking nutritional elements that result in under nutrition, poor growth or stressful mealtimes for children and their caregivers".

Historical Background

In the early 1900s, doctors were cognizant of the fact that behaviors such as vomiting could be a symptom of feeding problems including overfeeding or too frequent feeding, medical conditions such as pyloric obstruction, and feeding of improper foods (Lowenburg, 1912). By 1935, clinicians were addressing feeding problems that occurred as the result of structural anomalies with equipment such as the modified Asepto syringe and medicine dropper. In fact, the Brecht feeder was specifically developed to assist in feeding infants with structural abnormalities and/or physical and mental handicaps (Hess, 1946). Feeding problems began to appear in physicians' descriptions of developmental disabilities, such as cerebral palsy in the 1950s (Ingram, 1954; Perlstein & Barnett, 1952). During this time, it was suggested that feeding problems be classified into one of two groups: organic feeding problems and feeding problems in children who are emotionally disturbed and show tension and anxiety (Shwartz, 1958).

By the 1970s, reports suggested that feeding problems occurred frequently in preschool handicapped children and were a "significant clinical entity" (Palmer, Thompson, & Linscheid, 1975). However, no system was consistently being used to classify these difficulties; a problem that continues to exist today. Feeding problems during this time were attributed to neuromotor dysfunction or physical abnormalities. In cases where neuromotor dysfunction and physical abnormalities were ruled out, the feeding problems were determined to have a behavioral cause (Thompson & Palmer, 1974). Interventions to address feeding problems included techniques designed to treat feeding problems caused by mechanical difficulties (as cited by Thompson & Palmer). These techniques often included positioning recommendations aimed at facilitating movement and/or prescribed exercises to normalize tone and oral sensitivity. Treatment of behavioral feeding problems often included psychotherapy, using hunger as a means to motivate the child to eat a specific food and gradually increasing texture as a means to introduce solid foods into a child's diet, and/or behavioral modification strategies (Benotvin, 1970; Bernal, 1972; Jones, 1982; Thompson & Palmer, 1974; Palmer et al., 1975).

Diagnosis and treatment of feeding problems is still in its infancy. The treatment strategies used during the 1960s and 1970s are similar to those used now (for a review of early strategies, see Palmer, Thomspon, & Linscheid, 1975). One of the most significant changes in treatment of feeding problems since the 1970s is awareness that feeding is a give-and-take experience that relies on both the child and caregiver. Thus, treatment today often includes consideration of child-caregiver interactions during mealtimes.

Current Knowledge

Successful feeding involves (1) acceptance of a wide variety of developmentally appropriate foods and (2) development of motor skills that enable efficient and safe sucking, chewing, propelling, and swallowing. However, normal feeding development is not restricted to the physical act of eating. It also includes the successful integration of a range of physical functions and interpersonal relationships. Both physical factors (e.g., integration of swallowing and respiration, hand-eye coordination, normal posture, and tone development) and social factors (e.g., cultural patterns and social factors within a family) influence feeding development (Arvedson & Brodsky, 2002). Disruption in one or more of these areas can result in a feeding problem (Bryant-Waugh, Markham, Kreipe, & Walsh, 2010).

Feeding problems are defined as delays and/or disorders in the development of eating and drinking skills. This includes the motor aspects of eating (e.g., difficulty chewing and difficulty swallowing) as well as deficits in taking nutritional elements, which causes poor growth or stressful mealtimes (American Speech-Language-Hearing Association, 2001). Feeding problems manifest themselves in a variety of ways, ranging from an inability to swallow without choking to restricted diets. Approximately 25–40% of infants and toddlers are reported to have feeding problems (Reau, Senturia, Lebailly, & Christofell, 1996). Many early feeding problems are transient and resolve without significant clinical intervention (Bryant-Waugh et al., 2010). However, others fail to resolve without intervention. These feeding disorders often have multifactorial causes, which may include structural abnormalities, and a substantial behavioral component (Arvedson & Brodsky, 2002; Bernard-Bonnin, 2006).

A consistent classification system is not yet used for feeding problems. The Diagnostic and Statistical Manual of Mental Disorder-4th Edition (DSM-IV) classifies feeding disorders into three diagnostic categories: feeding disorders, pica, and rumination disorders. However, a number of eating disorders commonly seen in early childhood have no place in the DSM-IV classification system (Bryant-Waugh et al., 2010). The organic and nonorganic dichotomy has also been used to classify feeding problems. Organic feeding problems occur as a result of structural anomalies, neuromuscular issues, or other known physiological causes (e.g., gastroesophageal reflux), whereas nonorganic feeding

problems occur due to disruptive environments or have emotional underpinnings. While this dichotomy is helpful, feeding problems often have more than one cause (for a review, see Burklow et al., 1998). The American Speech-Language-Hearing Association has adopted a more in-depth classification system (American Speech-Language-Hearing Association, 2007). While this classification may prove to be more useful than the organic/nonorganic dichotomy, it does not address the issue that most children with feeding problems often have multiple diagnoses (Arvedson & Brodsky, 2002) and, as such, may fall under more than one category. For example, organic factors may disrupt the typical development of eating which results in the development of maladaptive behavioral patterns by the child and/or the caregiver (Burklow et al., 1998).

Given the complexity of many feeding problems, an interdisciplinary approach to addressing these problems is recommended. Interdisciplinary feeding teams may include a speech-language pathologists, developmental pediatrician, otolaryngologist, gastroenterologist, nutritionist/dietitian, occupational therapist, and social worker or nurse. Speech-language pathologists conduct feeding and swallowing evaluations and develop feeding plans. Developmental pediatricians provide diagnoses of pediatric and neurodevelopmental disabilities and manage overall patient care. Otolaryngologists examine the integrity of the structural mechanisms of swallowing and manage medical and surgical treatment of issues related to drooling, aspiration, and gastroesophageal reflux. Gastroenterologists conduct gastrointestinal evaluations and manage gastrointestinal diseases. The nutritionist/dietitian assesses and manages the child's nutritional needs, and the occupational therapist evaluates the child's posture, tone, and sensory system. Social workers or nurses coordinate home care and other appointments (Arvedson & Brodsky, 2002).

Regardless of who is on the team, diagnosis and management of feeding problems follow the same steps: (1) define the feeding and/or swallowing problem, (2) identify etiology(ies), (3) determine appropriate diagnostic tests, (4) plan approach to patient/family, (5) teach about problem, (6) implement treatment, monitor progress, and (7) evaluate progress (Arvedson & Brodsky, 2002). Determining the underlying etiology is a critical aspect of any successful intervention program because disturbances in feeding and eating behavior with similar clinical presentations may differ in their underlying etiologies and require different interventions (Bryant-Waugh et al., 2010). Certain medical conditions and developmental disabilities are associated with specific feeding problems. Neurological conditions and anatomical anomalies are most often associated with skills deficits such as oral motor delays, whereas medical conditions such as gastroesophageal reflux are most often associated with food refusal among children with Down syndrome, autism, and cerebral palsy (Field, Garland, & Williams, 2003; Williams, Bridget, & Schreck, 2005).

Feeding programs often have multiple components. For example, programs may address oral sensorimotor issues and posture and positioning during feeding. Oral sensorimotor programs are designed to address deficits in the structures that support feeding and swallowing. Goals of these programs may include improving jaw, lip, and/or tongue movements; developing coordinated movements of the mouth; and normalizing sensory oral experiences during mealtimes. Therapy may also be aimed at developing transitional feeding skills including spoon-feeding, cup-drinking, straw-drinking, and chewing. In addition to targeting skills that directly support feeding, interventionists may also address positioning and posture. Positioning is especially important for children with abnormal muscle tone. Management of behavioral feeding problems may focus on weight gain, initiation of oral feedings, weaning from tube feeds, increased oral intake, improved cooperation and reduced stress during mealtimes, and/or acceptance of a wider variety of flavors and textures (Arvedson & Brodsky, 2002). Because mealtimes offer the opportunity for parent-child interactions, it is important that management of feeding and swallowing problems occurs within the context of social and communicative

activities associated with mealtime for a parent and child (Arvedson & Brodsky, 2002).

Future Directions

The field of feeding problems is still in its infancy, especially with regard to feeding and swallowing problems in children (Arvedson & Brodsky, 2002). Although research in the area of feeding and typical swallowing and swallowing disorders has increased, continued research is needed. In the field of feeding problems, there are still inconsistencies in terminology and the detail of description of a number of eating problems, which hampers research. Thus, it is critical that an internationally recognized and accepted classification is established to inform clinical interventions for particular disorders and move the field forward (Bryant-Waugh et al., 2010).

To date, few research studies have attempted to examine prognosis, course, outcome, and treatment response in feeding disorders using a formal, widely accepted classification system. There is little evidence to guide clinical determination of what constitutes a clinically significant feeding problem and to help clinicians distinguish feeding problems that are more serious from those that are likely to be short lived. In order to better assess prognosis and outcome, researchers should employ a set of consistently used standardized assessment measures (Bryant-Waugh et al., 2010). In addition to directly examining feeding problems, research should continue to examine the outcomes of feeding assessment and intervention in schools and the overall impact on education. Finally, there needs to be an expansion of education programs focused on delivery of feeding and swallowing services in the school (American Speech-Language-Hearing Association, 2007).

See Also

- ▶ Gastrointestinal Disorders and Autism
- ▶ Nutritional Interventions
- ▶ PICA

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Feingold Diet

Susan Hyman
Division of Neurodevelopmental and Behavioral Pediatrics, University of Rochester Golisano Children's Hospital, Rochester, NY, USA

Definition

Feingold diet is a dietary intervention popularized for symptoms of inattention, impulsivity, and hyperactivity by Benjamin Feingold, M.D., a pediatric allergist who clinically observed improvement in behavior in his patients for whom he prescribed dietary restrictions for management of symptoms of allergy. It calls for the elimination of synthetic food colorings and synthetic preservatives made from petroleum-based products, synthetic sweeteners, and foods containing high levels of natural salicylates (related to the chemical compound in aspirin).

The Feingold diet eliminates:

- Synthetic food coloring made from petroleum products (e.g., Red #40; Yellow #5)
- Synthetic food preservatives made from petroleum products (e.g., BHT-butylated hydroxytoluene)
- Foods that may be high in natural salicylates (e.g., apples, almonds, berries, cucumbers, grapes, and oranges, among other fruits and vegetables)
- Artificial sweeteners (e.g., aspartame)

Foods could be reintroduced as tolerated. Later modifications to the diet included removal of the preservatives Sodium Benzoate and BHA (butylated hydroyanisole).

While additional dietary approaches to hyperactivity have become popular since the initial dissemination of the Feingold diet, assertions that sugar, yeast, milk, or other foods result in behavioral change were not part of the Feingold diet.

Historical Background

It was initially reported in the 1940s that some foods high in natural salicylates and FD and C Yellow #5 (a food coloring approved for food, drug, and cosmetics) produced similar allergic symptoms. Dr. Feingold was the chief of pediatrics of Cedar Sinai Medical Center in Los Angeles and subsequently the Chief of Allergy for Kaiser Permanente Medical Center in San Francisco. In 1965, Dr. Feingold prescribed a diet low in foods containing salicylates to a patient with hives whose psychiatric symptoms subsequently improved (Feingold, 1985). While he initially called the regimen for management of hyperactivity the K-P Program standing for Kaiser Permanente, it became known in the press as the Feingold diet or program. He clinically observed that the dietary regimen of removal of foods he believed contained high levels of natural salicylates and contained synthetic food dyes and sweeteners resulted in subjective behavioral improvement in up to 30–50% of his patients. By 1979, he further recommended elimination of synthetic preservatives with subjective reports of improvement in 60–70% of his patients. While Dr. Feingold recommended limiting sweets, he at no time recommended elimination of sugar in the diet. His concern was about the additives in processed foods and recommended that families make treats and sweets from scratch that should be provided as part of a healthy diet.

He presented his observations regarding food and food additives and behavior to the American Medical Association in 1973. Much controversy ensued, with small clinical trials both reporting improvement in children on the restricted diet

and other studies not being able to confirm these findings (Mattes & Gittelman, 1981). The National Institutes of Health convened a consensus development conference in 1983 to examine the data concerning defined diets and hyperactivity. The expert panel concluded that the data was insufficient to recommend defined diets for the treatment of hyperactivity but allowed that a diet trial could be considered by families after appropriate neurobehavioral evaluation and diagnosis took place with consideration of evidence-based options (National Institutes of Health Consensus Development Panel, 1983). Since that time, additional studies have been undertaken examining the behavioral effects of component parts of the Feingold diet. Wolraich et al. demonstrated that the artificial sweetener aspartame (Wolraich et al. 1994) does not impact attention. Studies on the effects of synthetic food coloring have been more controversial (reviewed in Schab & Trinh, 2004). Synthetic food coloring use is restricted by European countries but the scientific data has been interpreted to allow for continued use in the USA (Advisory Committee (March 30–1, 2011)).

Dr. Feingold's book, *Why is Your Child Hyperactive?*, was last published in 1985. The Feingold Foundation maintains an active society to support families who elect to pursue this dietary intervention (www.feingold.org/overview.php).

Rationale or Underlying Theory

The original theory proposed for behavioral improvement with the Feingold diet was related to the elimination of allergens leading to improved attention, learning, and behavior. Proponents point toward the introduction of increasing amounts of artificial flavors and colors in the diets of children as paralleling the increase in diagnosis of Attention Deficit Hyperactivity Disorder (ADHD). A hypothesis related to inhibition of neurotransmitter uptake of dopamine by FDC Red #3 was proposed but not confirmed in subsequent animal and human studies (National Institutes of Health Consensus Development Panel, 1983). The lack of documentation of benefit in clinical trials and biologic mechanisms to explain clinical observations impacted the FDA decision to continue to allow synthetic food coloring (Advisory Committee (March 30–1, 2011).

Goals and Objectives

The Feingold diet eliminates specific types of foods in the diet reported to be high in natural salicylates and processed foods containing synthetic food colorings, sweeteners, and preservatives to promote behavioral improvement in attention, hyperactivity, irritability, and learning. Dr. Feingold managed over 600 of his patients with this approach and reported that up to two thirds of the children improved. He reported that many children could be taken off of stimulant medications used to treat symptoms of ADHD once the diet was initiated. Dr. Feingold advised Conners, Goyette, Southwick, Lees, and Andralous (1976) in the first clinical trial testing his hypotheses. Prospective studies attempted to examine the behavioral effects of the Feingold diet, artificial sweeteners, and/or food dyes and preservatives on the behavior of children in the learning laboratory, the classroom, and the home. Studies were designed to examine both behavioral change with implementation of the diet and the response to double-blind placebo-controlled challenges of specific agents. The studies are difficult to compare because of varied design features and the different goals and outcome measures of the individual studies. Some address only components of the diet (e.g., aspartame, specific food dyes). There is no consistency to the dosage of exposure of synthetic food coloring across studies.

Treatment Participants

None of the published studies specifically evaluates the utility of the Feingold diet for children with ASD. The studies evaluate children with

ADHD, children with symptoms of hyperactivity, and/or community samples. There are several different sets of studies that examined components of the Feingold program that all approached identification of the study population in different ways. Many selectively recruited children whose families made the observation that their child improved behaviorally on the dietary intervention (Schab & Trinh, 2004). Because of the difficulty in maintaining a restricted diet and cost of clinical trials, the double-blind placebo-controlled studies were all of relatively small size.

Studies that set out to evaluate the effect of the Feingold diet including all four components included the original trial by Conners et al. (1976) that compared 15 children with ADHD to themselves in a 1 month crossover design. A crossover design was used to assess 36 children with hyperactivity without medication by Harley, Ray, Tomasi, Eichman, Matthews, Crun, Cleelard & Transmar, (1978). While not the original Feingold diet, oligoantigenic diets where a few nonprocessed foods are initially permitted then other foods are sequentially added back have been studied more recently (Pelser, Frankena, Buitelaar & Rommelse, 2010) in children with behavioral symptoms.

Other studies examined the effects of sucrose or artificial sweeteners like aspartame on behavior. Wolraich, Lindgren, Stumbo, et al. (1994) demonstrated no effect on cognition or attention from double-blind placebo-controlled challenges of aspartame in 25 typical preschool children and 23 school-aged children who were reported to have behavioral effects with sugar. In another report, Wolraich, Wilson, & White, (1995) summarized studies examining the effect of sugar an attention that included a total of 535 children. No significant effect was sustained across studies.

A third set of studies had as their goal the assessment of the behavioral effects of food dyes and preservatives on children. Mattes & Gittelman, (1981) reviewed the literature and reported on 11 children with symptoms of ADHD who were recruited as responders to the Feingold diet. A meta-analysis of studies including 136 participants in randomized double-blind placebo-controlled studies that examined the effects of artificial food coloring was published by Schab and Trinh (2004). McCann, Barrett, Cooper, et al. (2007) examined the behavioral response to two different concentrations of synthetic food dyes and sodium benzoate in two groups of children: 153 three-year-olds and 144 eight- and nine-year-olds drawn from a diverse community sample and not selected for hyperactivity.

A fourth type of study investigating the effect of increasing the nutritional value of foods provided a large population of public school students that included provision of foods with fewer synthetic ingredients (Schoenthaler, Doraz, & Wakefield, 1986).

Treatment Procedures

The study procedures are varied.

Diet introduction and maintenance: Most studies introduced the diet as part of the study or evaluated children already on the diet. Harley et al. (1978) provided all the foods to the families for the child under study to ensure that the diet was stringently followed. Some studies restricted other medications or supplements the children ingested. None formally analyzed and reported on the dietary sufficiency of the diet consumed or looked for other nutritional effects on behavior, although several commented on the overall sufficiency of the diet.

Dietary exposure to challenges: The studies that used a double-blind placebo-controlled approach to examine the behavioral effect of exposures typically provided a snack or beverage that contained synthetic food coloring, preservative, and/or aspartame at a standard time or times through the day. Some provided for repeat daily exposure for a week or more; others provided single challenges. At least one study examined the effect of time of day. Allowance for washout was not typically addressed and might impact the results of otherwise carefully done trials (Schab & Trinh, 2004).

Measurement of change in behavior: Outcome measures will be discussed below. The study designs included subjective rating scales from parents, teachers, and research team members; laboratory tests of attention and learning; and objective measures of behavior largely in class settings.

Efficacy Information

The report of the NIH consensus development panel in 1982 was an accurate summary of the evidence available at that time and remains an appropriate summary of the efficacy information accrued over the subsequent 29 years:

- There are clinical reports of children whose parents observe that dietary restriction improved symptoms of hyperactivity.
- The scientific literature overall does not demonstrate a statistically significant improvement in attention or hyperactivity as reported by laboratory tasks of inattention and impulsivity, clinician ratings or teacher ratings with the defined diets that eliminate food dyes and food preservatives or alter the fruit and vegetable composition of the diet.
- There may be an impact on behavior in parent rating scales related to food dye exposure. It is unknown if this may be related to specific food dyes and preservatives, exposure, and/or age.
- Aspartame does not impact attention or hyperactivity.
- A defined diet should only be considered after diagnostic evaluation for ADHD and the implementation of educational and behavioral interventions. Families considering a trial of dietary intervention should have adequate information to weigh the potential benefit from conventional medications and should discuss their plan with their health-care provider. Data collection regarding change in target behaviors will help families determine if their behavioral goals are met by this intervention.

No controlled trials on the effects of the Feingold Diet have been completed in children with autism spectrum disorders.

Outcome Measurement

The response dietary interventions across studies are difficult to compare because different measures were used that capture different aspects of behavioral response.

Subjective response as reported by parents: Studies used a variety of parent report measures designed to capture the core symptoms of ADHD: impulsivity, inattention, and motor hyperactivity. The Conners scales have been validated and widely used by researchers studying hyperactivity (Schab & Trinh, 2004). Other investigators compromised the interpretation of their studies by developing their own measures that were not well validated (reviewed in Schab & Trinh).

Subjective response as reported by teachers: Teacher reports using the Conner's scales were common to many studies. This questionnaire is well validated and was widely used in the time period that many of these studies were completed. Schab and Trinh (2004) point out that teachers often rate children as more hyperactive than parents because of the tasks required during the school day.

Laboratory measures of attention and activity: Laboratory measures of vigilance and attention were used in several studies. Tests such as the Conner's Continuous Performance Test II were used by investigators to provide data on sustained attention and response inhibition on standard tasks that are impacted by inattention, impulsivity, and hyperactivity in children with ADHD.

Combination of outcome measures: While most investigators reported on individual measures by examiner, McCann et al. (2007) created a Global Hyperactivity Index to capture the results of the laboratory attention task, classroom observation and rating, and parent rating in a single score. The participants in their study were not selected for symptoms of hyperactivity and represented a community sample.

Measurement of behaviors other than ADHD: Few studies examined behaviors other than those related to attention and hyperactivity. Rowe & Rowe (1994) attempted to capture parent rating

of irritability and sleep difficulties which were elevated in their report on exposure to food coloring. When parent report data is separated from the data collected by schools and clinicians, exposure to synthetic food coloring may be associated with increased behavioral symptoms (McCann et al., 2007; Schab & Trinh, 2004). It may be that behavioral effects other than ADHD core symptoms are captured in the parent report data.

Qualifications of Treatment Providers

The advocates of this diet suggest that there is no harm in the removal of synthetic food colorings and preservatives and artificial sweeteners by the provision of a diet containing fewer processed foods. The Feingold Foundation publishes a list of common products that meet their specifications, so implementation of the diet should be neither expensive nor onerous. Removal of a large number of the fruits and vegetables consumed in the American diet might have a nutritional impact. This has not been investigated. The diet recommends substitution of other foods such as bananas, cashews, and pears that the Feingold Foundation suggests are lower in natural salicylates. Reading food labels for processed foods for the presence of food dyes and preservatives may be challenging. Families who are considering a trial of dietary intervention should consult with their primary health-care provider. It may be helpful for families to be referred to a Registered Dietitian, a licensed professional with training in the nutritional content of foods, for counseling regarding diet change and how to provide an adequate diet if supplemented, processed foods are eliminated. Not all fruits and vegetables are nutritionally equal, so children with food selectivity might require targeted supplementation.

A family considering a dietary intervention might also consult with their child's educational or behavioral providers to collect data on the behaviors they wish to change. This may be very helpful in determining if there is a behavioral effect that is clinically significant enough to continue intervention. Since the dietary intervention targets symptoms of ADHD, formal rating scales for ADHD in use in the school setting could be considered.

See Also

- ▶ Aberrant Behavior Checklist
- ▶ Gluten-Free Diet

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http://www.feingold.org/overview.php

Fenfluramine

Lawrence David Scahill
Nursing & Child Psychiatry, Yale University School of Nursing, Yale Child Study Center, New Haven, CT, USA

Synonyms

Pondimin

Definition

Fenfluramine is a drug with an incompletely understood mechanism of action. It was originally introduced for the treatment of obesity. It promotes the release of serotonin acutely, but may have a serotonin-depleting effect over time.

Fenfluramine was originally used for the treatment of obesity. Given the replicated finding that at least a subgroup of children with autism are *hyperserotonergic*, investigators considered fenfluramine for the treatment of autism. Side effects were noteworthy. A randomized trial conducted in the mid-1980s showed that fenfluramine was no better than placebo. Since then, fenfluramine was removed from the market in 1997 when there were reports of pulmonary hypertension and damage to heart valves.

See Also

▶ Serotonin

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Feral Children

Lorna Wing
Centre for Social and Communication Disorders, Bromley, Kent, UK

Definition

The term “feral” children is used to refer to children who are known, or are believed, to have been deprived of contact with other human beings or to have been neglected or treated inhumanely by a human being since infancy. They have had little or no experience of human love or care and no experience of human communication through spoken language. Some are thought to have been reared by animals. Accounts of such children can be found in myths, legends, fiction, and in stories believed to be true, though some have turned out to be hoaxes. The relevance

for autism spectrum conditions lies in the inability to understand and speak and the odd behavior of many of the children and young adults, who are known or thought to have been deprived of human love and communication in their early years.

Historical Background

The accounts of such children in myths, legends, and fiction do not, however, have any link with autism. The legend of Romulus and Remus is one example. They were twin brothers, sons of the god Mars. When very young, they were abandoned by the banks of the river Tiber, but were found by a she-wolf who fed them with her milk and sustained them until they were able to feed themselves. Later, they were found and given a home and care by a shepherd, and they grew up to be strong and clever. They decided to build a city on the place where the shepherd had found them – the legendary origin of the city of Rome. After it was built, they quarreled over who should be in charge, and Romulus killed Remus. Romulus became King of Rome, which he named after himself.

The most famous "feral" characters in fiction are Mowgli and Tarzan. Mowgli was a character in stories written by Rudyard Kipling. Mowgli's parents were killed by a tiger in the Indian jungle when Mowgli was still a baby. He was found and reared by a mother and father wolf in a pack of wolves. He developed remarkable skills in hunting and tracking. He had close relationships with a black panther and a brown bear and was able to communicate with them. In the end, he returned to human society and had no problems communicating in human speech.

Tarzan was the fictional character invented by Edgar Rice Burroughs (although it has been suggested that he was influenced by the Mowgli stories). Tarzan's parents died when he was a year old, and he was adopted by a group of great apes, the mangani, which is a species unknown to science! Tarzan's jungle upbringing gave him remarkable abilities, beyond those acquired by most human beings. He was strong, agile, and well able to wrestle with all kinds of animals. He learned languages rapidly, including the language of the apes who reared him and other jungle animals and English, French, German, Latin, and ancient Greek, among others. He met and fell in love with a young American woman, Jane, whom he eventually married. They tried living together in England but found civilization hypocritical, so they returned to live in Africa.

The stories of Mowgli and Tarzan have been and are enormously popular and have led to many more books, as well as films, DVDs, television programs, and even Disney cartoons. There seems to be something about these stories that people all over the world find endlessly fascinating. However, the fictional pictures of highly intelligent, gifted individuals who have learned languages and can communicate with animals and with human beings stand in stark contrast to the known facts concerning real "feral" children. Those in fiction bear no relationship to autism, unlike many of those, who, in real life, in their early years, were deprived of care and love from other human beings (Frith, 2003a).

Current Knowledge

Another inappropriate use of the term "feral" children is to refer to those who live on the streets of cities. They are also known as "street" children. Such children have been deprived of family care because of neglect, or abuse, or parental death, or abandonment by poor parents who cannot afford to care for them. Large numbers of such children can be found in great cities throughout the world. Professor Martin Patt (2010) has created a website on which it is possible to find details of street children in different countries throughout the world. Patt emphasizes that such children can fall under the control of traffickers, who may disfigure the children in order to increase the money they can make by begging. The street children are also vulnerable to sexual abuse.

The suggestion of a link with autism is not appropriate especially for those street children

who do not fall under the control of criminal adults. Typically, such children form their own gangs, with their own social rules and hierarchies to support each other and survive through various forms of criminal behavior. (See note on Romanian street children, Patt (2010) below.) They appear in fiction, for example, as the young pickpockets organized by Fagin in Charles Dickens' book "Oliver Twist" or the street gangs, the "Baker Street Irregulars" who carried out various tasks for Sherlock Holmes in the stories by Sir Arthur Conan Doyle. They are quite different from the isolated child deprived of any human concern, care, and communication.

The children sometimes referred to as "feral," whose history and behaviors are most relevant for any discussion of autism spectrum conditions, are those who have been deprived of human companionship and loving care for all or most of their infancy and childhood. There are accounts of children who are known or believed to have been kept in solitary confinement from very early in life, perhaps from infancy. These children have been given minimal basic feeding and minimal care by a human being who does not show them any love and companionship and does not communicate beyond simple orders. One of these unfortunate children was Kaspar Hauser, who appeared in Nuremberg in 1828, at the age of 16–17 years. A detailed account of Kaspar and his behavior was written by Anselm von Feuerbach (1833), then President of the Bavarian Court of Appeal at Anspael near Nuremberg. It seems that, during his first 16 years, Kaspar had been kept in a dark cellar. He had been fed on bread and water and had never seen his keeper. His only companion was a toy horse. When found in Nuremberg, he was able to say only a few phrases and fragments of speech. He preferred darkness to daylight and liked to sit on the ground with his legs stretched out in front of him. He had problems with distance vision, which could be linked to the fact that his vision had been limited by the darkness and walls of the cellar he had lived in.

His story is one of those discussed by Uta Frith (2003b). She notes that Kaspar rapidly learned to spell. He also showed social interest and affection towards children and adults who became close to him. He loved the toy horse he was given to replace the one he used to have in the cellar, and offered it a share of his food. He continued to have an odd gait, and he showed a marked preference for cleanliness and order for his possessions. Despite these continuing oddities, Professor Frith concluded that Kaspar was certainly not autistic – his social attachments and rapid improvement being strong indicators that he was not affected by this developmental disorder. Kaspar survived one attempt by a stranger to kill him. However, he was assassinated 5 years after his arrival in Nuremberg. The mysteries of his origin, why he was kept in a cellar, why he was assassinated, and who killed him have never been solved, though there were unverified rumors that he was the unwanted child of a royal person.

A much more recent account of a child deprived of contact with loving human care and the outside world from early childhood is that of the girl known as Genie (not her real name). The psycholinguist who studied her, Susan Curtiss (1977), referred to her as a modern-day "wild child." Genie was born in the USA, the daughter of a violent, difficult father and a sad, frightened mother. She was the fourth and last child in the family, and was delivered by cesarean section. The first had died in infancy because his father objected to his crying and so kept him in the unheated garage where he developed pneumonia, which caused his death. The second child also died. The third, a boy, at 5 years, was taken into the care of his paternal grandmother, where his development, previously retarded, improved markedly, and he finally returned to his parents. At the age of 14 months, Genie developed pneumonitis and was seen by a pediatrician, who suggested that she showed signs of retardation, though the pediatrician could not be sure because Genie had a high fever.

When Genie was 20 months old, her paternal grandmother died in a road accident. Genie's father was intensely disturbed by this. He moved the family into his mother's house and isolated them from the outside world. Genie was kept in a small bedroom, tied by a harness

to a potty chair. At night, she was placed in a sleeping bag, which severely restricted her movements. Genie was kept secluded and had no toys to play with or radio to listen to. Her father hated any sort of noise, so conversation in the home was kept to a minimum and at a low volume. Genie's father did not speak to her but barked and growled at her, and then beat her if she made any noises. She was fed baby foods that were stuffed into her mouth. This pattern of life for Genie lasted until she was 13½ years old, when her parents had a violent argument and Genie's mother left her husband and home, taking Genie with her. After spending 3 weeks with the maternal grandmother, Genie was seen by a social worker, who sensed that there were serious problems. Genie was admitted to hospital with severe malnutrition. The police were involved and charges brought against the parents. On the day of the trial, the father killed himself.

For 5 years from November 1970, when Genie was released from virtual captivity and isolation, her skills and disabilities were studied in detail by Curtiss and other colleagues. At first, she was very underweight and undersized and had major problems of self-care, movement, and distance vision. She was silent, even when sobbing, apart from a high-pitched whimpering and a few words. To quote from Susan Curtiss (1977), ". . .she salivated copiously, spitting onto anything at hand. Genie was unsocialized, primitive, hardly human."

Susan Curtiss analyzed in detail aspects of Genie's ability with understanding and using language. Among other problems, Genie consistently confused *you* and *me*. She used entire phrases as labels, she failed to acknowledge questions, requests, etc., addressed to her. These are features also seen in children with autism spectrum disorders. Despite all this, Curtiss reports that Genie was alert and curious, avidly exploring her new surroundings. She was intensely eager for human contact and made good eye contact. These aspects of her behavior counteract any suggestion that she had an autism spectrum disorder. Susan Curtiss concluded that Genie appeared to be a "right hemisphere thinker." She suggested that this was due to the deprivation of language experienced in her childhood.

Many questions relevant for a possible diagnosis of autism remain unanswerable. Was the pediatrician right in thinking that Genie was retarded when he or she saw her at 14 months, before her isolation? Did Genie show any of the odd hand and arm movements associated with autism, as well as her odd gait and posture? What was the underlying cause of Genie's father's bizarre behavior to his family – did he have a developmental disorder? There is no way of knowing.

Very relevant to the subject of children deprived of loving personal care and human communication from infancy or early childhood are the stories of the Romanian orphans. Nicolae Ceausescu was president of Romania from 1974 to 1989. During his presidency, birth control was made illegal in order to boost the population. The result was that many families had more children than they could possibly afford to care for, so these children were placed in state-run institutions. The care provided was minimal and often appallingly bad, and the children were kept in cots, deprived of human love, social interaction, and communication as well as lacking adequate nutrition and other health needs. In addition, during the Ceausescu regime, children who did not pass the state-organized physical and psychological assessments carried out on preschool children were also placed in institutions.

After the fall of Ceausescu and his regime, the sad life of the children in such institutions was publicized outside Romania. Some children from Romanian institutions were adopted by non-Romanian families, including some in the UK. A sample of 144 children who had experienced institutional care in Romania and who were later adopted by UK parents were assessed at ages 4, 6, and 11 years by Professor Michael Rutter and colleagues (2007; O'Connor et al., 2000; Rutter et al., 1999) They were compared with 52 non-deprived UK children adopted in the UK and placed before 6 months of age.

The children from Romania, when they arrived in England, showed marked signs of physical and psychological deprivation. They

had been kept in their cots with no social or intellectual stimulation of any kind. The majority of the group showed remarkable catch-up by the time they were seen at 4 years of age. However, 16 children (11.1% of the sample of 144 children) showed "quasi-autism." Three of the 16 had IQs below 50, the rest having IQs above this level. These 13 children were studied in detail.

Their pattern of symptoms was called "quasi-autism" for specific reasons. While their scores on the ADI-R assessment at age 4 years were similar to a group of children with typical autism studied using the Autism Diagnostic Observation Schedule (Lord, Rutter, & Le Couteur, 1994), their scores at age 6 years were significantly lower (less autistic) than those of the typically autistic children. There was also a tendency for the IQ scores to rise. They showed more flexibility in communication. Some showed substantial social approaches, though of an abnormal kind, some being indiscriminately friendly. Those with "quasi-autism" had a much greater degree of cognitive impairment than the remainder of the adoptees from Romania. The sex ratio in the "quasi-autistic" children was approximately equal, compared with an excess of boys in the typically autistic children. Unlike the typically autistic children, the head circumference was not raised in those with "quasi-autism." The follow-up at 12 years showed that one quarter of the affected children had lost their autistic-like features by age 11 years.

Rutter and his team considered the possible causes of the "quasi-autistic" behavior, particularly in those (the majority) who did not have IQs below 50. They were significantly older than the rest when leaving institutional care to come to the UK. To quote from Rutter et al. (1999), "The quasi-autistic pattern seems to be associated with a prolonged experience of perceptual and experiential privation, with a lack of opportunity to develop attachment relationships and with cognitive impairment." The strongest predictor was the children's age of entry to the UK. Those adopted before 6 months showed almost complete physical and cognitive catch-up by 4 years. Those adopted between 6 and 24 months had lower scores on cognitive tests than those adopted before 6 months. Those adopted between 24 and 42 months had the lowest scores of all and a general developmental impairment. Rutter and his colleagues concluded that children adopted before 2 years of age were likely to show considerable resistance and to catch-up in cognitive and physical development.

The research team also found another 12 children who had some autistic-like features but not enough to qualify for "quasi-autism."

Future Directions

Dr Sue Sheppard, a chartered specialist educational psychologist who is working in the field of developmental disorders and is a consultant to the NAS Diagnostic Centre, went to Romania in 1992 as one of the group of professionals whose aim was to help the children in the state institutions. Among the children in the institutions, Sue Sheppard (personal communication, in conversation with the author in December 2010) found a small minority who quite clearly had autism spectrum disorders. This is not surprising, since parents who sent or abandoned children to institutions would be likely to select those whose behavior was challenging or whose development was clearly abnormal. Furthermore, state-implemented preschool screening tests would identify those with developmental disorders. In addition to those who were clearly in the autism spectrum, there was another minority who had some autistic traits, especially odd movements and other repetitive behaviors, and who were not socially responsive, though they did not present the full picture of an autism disorder. They closely resembled the children Rutter and colleagues described and called "quasi-autistic" (see above). Unlike those with clearly diagnosable autism, these children showed definite improvement over a period of time when given loving care and help by people who understood their problems, who came from relief agencies from other countries, sent in response to the problems children were having in Romania. These children's physical health and intelligence levels also improved markedly.

The majority of the institutionalized children did not show either of the above abnormal behavior patterns, seeming to be highly resistant despite the deprivation suffered. This was most likely to be the case if, as Rutter and his ream also found, the children were under 2 years old when their living conditions were improved.

Sue Sheppard also observed another group of children, often described as "wild" or as "bandits." These were children who lived in gangs on the streets and were sociable according to the values of the group they were with. They had become "street children" for various reasons, including having run away from abusive parents or institutions. They could smile sweetly and act in a charming way in order to get near enough to someone to steal from them. Although their behavior was antisocial from the point of view of society as a whole, as a group, they had their own social system, which certainly does not fit with a diagnosis of autism spectrum disorder.

The last group of "feral" children to be discussed here are those who are given the name "feral" because they are thought to have been reared by animals in their early childhood years. Stories of such individuals found wandering, captured, and reintroduced into human society can be traced back to the 1300s. Lucien Malson (1972) gives a list of 53 "feral" children recorded from 1344 up to 1961. Wikipedia adds another 10 in the years from 1961 to 1999 and 8 from 2000 to 2009. The animals alleged to have cared for the children include wolves, cows, pigs, sheep, leopards, bears, chimpanzees, monkeys, dogs, goats, and gazelles. A few children were actually found with animals (most such accounts are of children found with packs of dogs), but there is no direct evidence of them being suckled by the animals in their infancy. Several of the stories are known or thought to be fictitious, including the two that described children reared by gazelles. Where sufficient contemporary information is available, it is clear that those thought to have been reared by animals lacked basic social skills. The great majority of those reported did not learn to speak or else managed only a few words; they had problems learning to walk upright and to use a toilet and had no interest in other human beings or human activity.

The one "feral" child concerning whom we have a considerable amount of information is the "wild boy of Aveyron." This boy was found in 1798 wandering naked and wild in the woods in south-central France. He was captured and eventually given into the care of the physician Dr. Jean Marc Gaspard Itard and his house keeper, Madam Guerin. Itard wrote two detailed reports in 1801 and 1806 (see English translations, Itard, 1962) describing the boy's behavior and his progress in learning the skills that Itard tried to teach him.

When captured, Victor (the name given him by Itard) was thought to be about 12 years old, though small for his age. He had many scars on his body and limbs including one across his throat. He had been glimpsed in the forest on occasions for 2 years before his capture, so it seemed that he had lived in the wild at least from around age 10 up to 12 years.

In her book on autism, Uta Frith (2003c) includes a detailed discussion of the evidence concerning the possibility that Victor had autism. The relevant aspects of his behavior, as described by Itard, are as follows.

Victor showed no marked social attachment to anyone. He showed some attachment to his caretakers, Dr Itard and Madame Guerin, which seemed to be due to the fact that they provided his food. He showed no sympathy and no evidence that he had any concept of other people's ideas, wishes, or feelings. He had no ability to conform to any social rules.

Victor made various noises but did not learn to speak. He did learn that the word "lait" referred to milk, but never used it to request milk, only as a label when he was given milk. He used limited nonverbal ways of communicating the few messages he wished to communicate. For instance, he enjoyed having Dr. Itard touch and stroke his head and would take Itard's hand and place it on his head to indicate what he wanted. He never showed any evidence of pretend play or imaginative skills.

As is typical in autism, Victor did respond in unusual ways to various kinds of sensory input

(Leekam, Nieto, Libby, Wing, & Gould, 2007). He ignored most sounds, however sudden and loud, but turned at once to the sound of a nut cracking (nuts being a favorite food), however faint the sound. He seemed oblivious of heat, cold, or pain. This was one of the possible reasons why he could exist for at least 2 years in the wild.

He would wake early in the morning, wrap his head and body in his blanket and rock himself back and forth. He would lie down from time to time and then rock again until it was time for breakfast. He was fascinated by the moonlight, and would sit and watch the moon while rocking himself.

Despite lack of language skills, Victor was very efficient at shelling and preparing beans for cooking, including picking out any that were bad. He learned to carry out some other practical domestic tasks that did not require language to perform.

These behaviors indicate impairment of social interaction, communication, and imagination, and a preference for repetitive routines (rocking), which are diagnostic of an autism spectrum disorder. Uta Frith considers that it is highly likely that Victor had such a disorder. The present writer agrees with her, though, from such a distance in time, there can be no absolute certainty.

Harlan Lane (1979), professor of social psychology, does not accept that Victor could have had an autism spectrum disorder. However, the aspects of Victor's behavior he lists as evidence that would exclude a diagnosis of autism could be seen in many children with this diagnosis. Lane notes that Victor was not totally aloof and indifferent to others. For example, as already mentioned above, Victor did show some signs of attachment to Dr Itard and to Madame Guerin, who were his carers. There is an account of him weeping when reproached by Itard. But many children with typical autism spectrum disorders are not totally aloof – their social behavior is limited and can be inappropriate. Lane appears to be aware only of the most severe manifestations of autism and not the wide range of behavior across the whole autism spectrum and the changes that can occur with increasing age and the positive effects of education and of loving care.

While it is highly likely that Victor was in the autism spectrum and a few other "feral" children were clearly not autistic, for the great majority of cases of "feral" children, a diagnosis of autism or at least autistic traits appears to be a possibility, but there is rarely enough evidence to be certain.

A question that inevitably arises in any discussion of "feral" children is whether the autism spectrum disorder is due to biological causes and was present from birth (Feinstein, 2010; Frith, 2003d), leading to the child's loss or abandonment, or whether the lack of loving human care was the cause (Bettelheim, 1967). The available information on "feral" children cannot provide an answer to this question. The known cases of "feral" children are just a minute fraction of all those with autism spectrum disorders. The latest prevalence studies suggest that around 1% or more of children and adults have some form of this disorder (Gillberg & Wing, 1999; Wing, 2004; Wing & Potter, 2002). Research into the nature and origins of autism has shown that autism spectrum disorders are most likely to be due to genetic or other biological causes (Feinstein, 2010; Frith, 2003d). The amount of progress possible for those with autism has been shown to be linked to their innate level of ability, as measured by IQ tests, though the prognosis even for those with IQ over 70 is very variable (Beadle-Brown, Murphy, & Wing, 2006; Howlin, Goode, Hutton, & Rutter, 2004).

Another question that has not been answered concerns whether there is a "critical" or "sensitive" period for the acquisition of language. The critical period hypothesis holds that there is a limited time in early childhood when language can be acquired in response to exposure to facilitative speech presented by carertakers. If a child is deprived of this essential experience during the appropriate age period, he or she will not learn to understand and speak (Lenneberg, 1967). It has been suggested that the histories of "feral" children support this theory. The findings of Rutter et al. concerning the importance of removal to a supportive environment before 2 years, described above, do provide some support. The problem is that there is no way of knowing

whether or not the "feral" children who do not speak suffered from autism, or a developmental language disorder, or intellectual disability, as the reason why they did not speak and did not learn language when education was provided.

The sad truth is that the stories of "feral" children in all their variation, in most cases, present fascinating but insoluble mysteries. The questions they raise can only be solved (if at all) by appropriate modern research.

See Also

- ▶ Atypical Autism
- ▶ Child Abuse in Autism
- ▶ Factors Affecting Outcomes
- ▶ Romanian Adoptive Children

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Ferster, C.B.

A. Charles Catania
Department of Psychology, UMBC (University of Maryland, Baltimore County), Baltimore, MD, USA

Name and Degrees

Charles Fester

Ferster, Charles Bohris (Nov. 1, 1922–Feb. 3, 1981)

B.S. (Rutgers University, 1947), M.A. (Columbia University, 1948), Ph.D. (Columbia University, 1950)

Major Appointments (Institution, Location, Dates)

US Army Air Force, 1943–1946
Research Fellow under B. F. Skinner, Harvard University, 1950–1955
Postdoctoral Research, Yerkes Laboratory, Emory University, 1955–1957
Postdoctoral Research, Indiana University Medical Center, 1957–1962
Institute for Behavioral Research (Silver Spring, MD), 1962–1968, as Executive Director (1962–1963), Associate Director (1963–1965), Senior Research Associate (1965–1968)
Professor of Psychology, Georgetown University (Washington, DC), 1967–1968
Professor of Psychology, American University (Washington, DC), 1969–1981 (Department Chair, 1970–1973)

Major Honors and Awards

First Editor and Executive Editor of the *Journal of the Experimental Analysis of Behavior*, 1958–1961.

Landmark Clinical, Scientific, and Professional Contributions

With B. F. Skinner, developed the experimental methods of the experimental analysis of behavior, including the design of apparatus and procedures, especially with regard to schedules of reinforcement.

Participated in the founding of the first major journal in the field, the *Journal of the Experimental Analysis of Behavior*.

Extended the basic principles of behavior analysis to clinical problems and to education, in areas such as clinical depression, psychotherapy, and individual instruction.

Distinguished between the natural consequences of behavior and the arbitrary consequences that must be arranged when for some reason the natural consequences are not effective.

Opened the way to the treatment of autism by demonstrating that the behavior of the autistic child is sensitive to its consequences.

Short Biography

Charles B. Ferster was a pioneer in the experimental analysis of behavior and its extension to areas of application. His undergraduate career at Rutgers University was interrupted by service in the US Army Air Force during World War II. Upon completing his Rutgers B.S. in 1947, he entered the graduate program in Psychology at Columbia University. There Fred S. Keller, with

W. N. Schoenfeld, had established a curriculum based on B. F. Skinner's analysis of behavior (Keller & Schoenfeld, 1949, 1950). Keller referred Ferster to Skinner at Harvard University, and Ferster, completing his Ph.D. in 1950, moved to Harvard as Skinner's research assistant. Once there, he developed many innovations in methodology and in the automation of Skinner's pigeon laboratory, summarized in a journal article that became a guide for many other researchers (Ferster, 1953). By 1957, he and Skinner had published a seminal book, "*Schedules of Reinforcement*" (Ferster & Skinner, 1957), that detailed the properties and the results of these novel procedures in the analysis of behavior. Skinner was sufficiently impressed by Ferster's contributions to the research enterprise that he gave Ferster first authorship.

Behavior is maintained by its consequences, as when a pigeon's key peck or a rat's lever press occurs frequently because it produces access to food. This property of behavior is called reinforcement; reinforcement is not a theory but rather is simply a name for a ubiquitous phenomenon. What Skinner discovered about reinforcement in his earlier research (Skinner, 1938), and what he and Ferster explored in depth, was that the maintenance of behavior by reinforcement depends crucially on the contingencies arranged for responses, their schedules of reinforcement. In the real world as well as in the laboratory, not every response is reinforced. In what is called a ratio schedule, for example, only some fraction of responses is reinforced, as when some percentage of bets pays off; one must play to win, but one will not win every time. In what is called an interval schedule, however, whether a response is reinforced depends on when the response occurs rather than on how many responses have occurred, as when one finds mail in one's mailbox only if checking it after the mail has been delivered; checking more often will not affect when the mail comes. A full account of reinforcement schedules, which include both fixed and variable versions of ratio and interval schedules as well as other categories of contingencies, is beyond the scope of this article. For the present purposes, the crucial point is that different schedules produce and maintain very different patterns of behavior. For example, ratio schedules typically maintain substantially higher rates of behavior than do interval schedules, and details of the schedules determine the patterning of behavior over time (e.g., some schedules produce relatively constant responding, others produce gradual increases, and still others produce abrupt shifts from not responding to high rates of responding). Schedules of reinforcement soon became commonplace procedures for the study of a broad range of topics, such as the analysis of sensory processes, effects of psychopharmacological agents, and motivational operations.

One procedure studied by Ferster (1954) involved using a blackout, turning off the lights in the pigeon chamber and therefore eliminating the pigeon's opportunity to produce food. The name was later changed to time-out, and it was discovered that time-outs were sometimes useful because they could be used to reduce behavior, as when errors in some visual task produced time-outs from positive reinforcement. The use of time-out in some behavioral programs and eventually by some parents and teachers evolved from this early pigeon research.

In those early days of operant research, extending the basic results from the animal laboratory to significant human problems had just begun. Examples were Fuller's (1949) demonstration that reinforcers could be effective even on the behavior of a vegetative human, work by Lindsley (1956) showing the effects of schedules of reinforcement on the behavior of adult psychotic patients, and Bijou's (1957) extensions to the study of child development. After moving to a postdoctoral position at the Medical Center at Indiana University in 1957, Ferster undertook an extension of his work with schedules of reinforcement to the behavior of autistic children. In those days, diagnostic criteria were highly variable, and such children were often described in terms of early-stage schizophrenia. More important, the classification of autism implied individuals who were virtually confined within themselves, impervious to events in their environments. Ferster, in collaboration with DeMyers (Ferster & DeMyer, 1961, 1962), arranged laboratory settings functionally similar to those used in the

analyses of reinforcement schedules and found that when appropriately arranged food and candy and trinkets could become effective reinforcers of the behavior of these children. Other studies examined ways in which these reinforcers might eventually provide a foundation for building social reinforcers, which are typically ineffective with these children (DeMyer & Ferster, 1962). The research also showed that food could be used to make coins effective as conditioned reinforcers and that reinforcers could be arranged so as to enhance the attention of these children to some of the stimuli around them.

Beyond the fundamental observation that the behavior of these children could be reached through reinforcement schedules, albeit more slowly than with children without these deficits, this research provided the precedent for extensions that were soon to follow, including work by Lovaas and others (e.g., Lovaas et al., 1965, 1966). In his basic research, Ferster had shown that relatively simple and small changes in the contingencies arranged by reinforcement schedules could produce profound changes in ongoing behavior, and he brought these observations to bear in his subsequent work, which moved to analyses of other clinical deficits and pathologies, such as clinical depression (Ferster, 1965, 1972, 1973). Some additional contributions Ferster has made to the analysis of behavior have been described by Keller (1981) and by Skinner (1981).

See Also

- ▶ Applied Behavior Analysis
- ▶ Behavior Analysis
- ▶ Contingencies of Reinforcement
- ▶ Operant Behavior
- ▶ Schedule of Reinforcement

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Fetal Alcohol Effects

► Fetal Alcohol Spectrum Disorder

Fetal Alcohol Spectrum Disorder

John Thorne
Department of Speech and Hearing Sciences, University of Washington Fetal Alcohol Syndrome Diagnostic and Prevention Network, Seattle, WA, USA

Synonyms

Alcohol-related neurodevelopmental disorder; Fetal alcohol effects; Fetal alcohol syndrome

Short Description or Definition

Used to refer to the range of negative outcomes associated with prenatal ethyl-alcohol exposure, fetal alcohol spectrum disorders (FASD) is an umbrella term that describes a heterogeneous set of permanent birth defects. When an FASD includes growth deficiency, a unique cluster of three minor facial anomalies (i.e., small eyes, a thin upper lip, and a smooth philtrum), and evidence of central nervous system (CNS) abnormalities, it is referred to as *fetal alcohol syndrome* (FAS). Other FASD can include any combination of these impairments in the context of confirmed prenatal alcohol exposure. CNS abnormalities are the most common impairment found in individuals with an FASD. FASD may also include ophthalmologic, otologic, cardiac, renal, and orthopedic abnormalities.

Categorization

Diagnostic categories
FAS, alcohol exposed
FAS, alcohol exposure unconfirmed
Partial FAS, alcohol exposed
Sentinel physical finding(s) with static encephalopathy, alcohol exposed
Static encephalopathy, alcohol exposed
Sentinel physical finding(s) with neurobehavioral disorder, alcohol exposed
Neurobehavioral disorder, alcohol exposed
Sentinel physical finding(s), alcohol exposed

Epidemiology

Although the impact of maternal drinking on the pre- and postnatal development of children was examined as early as the late nineteenth century (Sullivan, 1899), teratogenic effects of prenatal ethyl-alcohol exposure were not characterized until the late 1960s (Lamache, 1967) and not widely recognized until the work by Smith and colleagues in 1973 (Jones, Smith, Ulleland, & Streissguth, 1973). The *fetal alcohol syndrome* (FAS) they described is now recognized internationally as a permanent birth defect syndrome resulting from prenatal alcohol exposure and is understood to include growth deficiency, a unique cluster of three minor facial anomalies (i.e., small eyes, a thin upper lip, and a smooth philtrum), and evidence of central nervous system (CNS) abnormalities. Subsequent work has shown that significant impairments are frequently found in children with prenatal alcohol exposure even in the absence of the full set of symptoms required for a diagnosis of FAS. Over time, the term fetal alcohol spectrum disorders (FASD) has come to be used to describe the full range of impairments associated with prenatal alcohol exposure. FAS is the most easily recognized FASD, largely because the distinctive FAS facial phenotype provides a specific diagnostic marker of prenatal alcohol exposure (Astley, 2004a). With prevalence of FAS estimated at 1–5 cases per 1,000 live births, it is the leading known preventable cause of developmental and

intellectual disabilities. Given lifetime costs for FAS estimated at two million dollars per case (Lupton, Burd, & Harwood, 2004), FAS is estimated to have an annual cost in the United States in excess of five billion dollars (Riley & McGee, 2005). FASD that lack the telltale facial features of FAS are more difficult to diagnose, but share a similar range and severity of central nervous system impairments and social costs. With numbers approaching 1% of all children, other FASD are many times more prevalent than FAS (Bailey & Sokol, 2008).

Natural History, Prognostic Factors, and Outcomes

The impairments associated with prenatal ethyl-alcohol exposure are permanent birth defects that effect on the developing system throughout the life course of individuals with fetal alcohol spectrum disorders (FASD). The degree and extent of damage caused by prenatal alcohol exposure is both dose and timing dependent, and the central nervous system is the most vulnerable body system. Individuals with FASD are at risk for learning, behavioral, and mental health impairments throughout their lives. Protective factors that reduced the chances of negative outcomes in individuals with prenatal alcohol exposure and/or FASD include early diagnosis, stable home placement with quality care, protection from violence, absence of substance abuse in the home, and the presence of appropriate social services. Early intervention in a coordinated system of care that maximizes these protective factors may reduce the chances that children with FASD will experience negative outcomes and should support their chances of success as they grow to adulthood (Streissguth et al., 2004).

Clinical Expression and Pathophysiology

The degree and extent of damage caused by prenatal ethyl-alcohol exposure is both dose and timing dependent. The central nervous system (CNS) is the most vulnerable body system, and various regions, developmental processes, and cell types in the CNS are differentially vulnerable to prenatal alcohol exposure at different stages of development. Prenatal alcohol exposure changes many ontogenetic processes in the developing CNS-disrupting or distorting neurulation and neural tube development, neuronal proliferation and migration, synaptogenesis, neuronal differentiation, and apoptosis. Prenatal alcohol exposure alters the hormone balance in the system as well as astroglial development and gene expression. Alcohol interferes with gliogenesis and development, delays myelination, and causes changes in the production of important neurotrophic factors (e.g., cell adhesion molecules such as the integrins and laminins) and neurotransmitters such as glutamate, NDMA, and serotonin.

Neurological Abnormalities

As expected, given its impact on developmental processes, prenatal alcohol exposure is associated with structural abnormalities throughout the CNS. These can include microcephaly, microgyria, gray and white matter defects, heterotopias, and regional hypoplasia. Imaging studies of the population with FASD are in their early stages, but some trends are emerging in the findings. Studies using functional magnetic resonance imaging and electroencephalography consistently find atypical results suggesting abnormal processing and development in individuals with prenatal alcohol exposure (e.g., D'Angiulli, Grunau, Maggi, & Herdman, 2006). The few magnetic resonance spectroscopy studies conducted have all found differences in neurochemical profiles associated with prenatal alcohol exposure (e.g., reduced choline; Astley et al., 2009). Prenatal alcohol exposure can result in reduced frontal lobe volume relative to overall brain volume particularly in the presence of FAS facial features (Astley et al.). There is also emerging evidence that temporal and parietal lobes may also be disproportionately vulnerable to prenatal alcohol exposure with exposure resulting in reduced volume of these structures. The caudate nucleus of the basal ganglia seems particularly

sensitive to prenatal alcohol exposure (Astley et al.), while the putamen, amygdala, and hippocampus seem relatively resistant to damage from prenatal alcohol exposure (Spadoni, McGee, Fryer, & Riley, 2007).

Findings related to the volume of the cerebellar vermis (CV) are equivocal (e.g., Astley et al., 2009), making it unclear whether findings of reduced CV in children with FASD result from smaller overall brain size or not. However, when found in children with FASD, reduced volume of the CV is associated with deficits in verbal learning tasks. The effects of prenatal alcohol exposure on the corpus callosum (CC) are also not clear, but there is a consistent finding across studies of shorter CC, with some regions of the CC apparently more sensitive to prenatal alcohol exposure than others (Astley et al.). When they are found, CC abnormalities are associated with functional deficits.

Neuropsychological Findings

Neuropsychological impairments have been documented across a variety of domains in children with FASD. Deficits may include impairments of attention, motor and choice reaction time, visual spatial learning, fine/gross motor control and balance, response conditioning, executive function, working memory, mathematical reasoning, nonverbal inductive reasoning, and information processing (Kodituwakku, 2007). Communication deficits are among the most frequently reported in FASD literature, and peripheral and central hearing impairments are common in FAS. Differences in IQ between children with prenatal alcohol exposure and their unexposed peers are well documented. However, in the FASD population, IQ scores cover the range from profoundly handicapped to above average intelligence. Although lower IQ scores have been shown to be associated with the presence of the face of FAS and increased prenatal alcohol exposure levels, even among children with FAS, approximately 75% have IQ scores above 70 (Riley & McGee, 2005).

Executive Functioning, Attention, and Behavioral Regulation: The caudate nucleus of the basal ganglia and the frontal lobes, structures which support executive functioning, seem particularly vulnerable to PAE, and impairments of executive function appear widespread in FASD. The most commonly reported concern is inattention. Deficits in planning, set shifting, problem solving, verbal and nonverbal fluency, and working memory are also commonly described. Deficits in social skills and adaptive functioning are widely reported, as are clinically significant problems maintaining appropriate behaviors (Kodituwakku, 2007; Riley & McGee, 2005).

Communication: Communication deficits have been documented for children with FASD in all areas of language and communication. These include impairments in auditory processing, phonology, morphosyntax, semantics, and integrative language/pragmatics. Later-developing discourse-level communication skills such as narrative storytelling may be particularly vulnerable to prenatal alcohol exposure (e.g., Coggins, Timler, & Olswang, 2007).

Sensory-Motor Development: Children with FASD frequently have deficits in fine motor, gross motor, and visual-motor abilities (Riley & McGee, 2005). These can include delayed achievement of early motor milestones, tone abnormalities, tremulousness, and oral-motor (e.g., suck-swallow) difficulties. Older children with FASD may experience impaired balance, reduced fine motor control, visual spatial problems, and motor immaturities. The poor sensory modulation commonly described among clinic-referred children with FASD may play a role in motor and postural deficits and poor behavioral regulation (Jirikowic, Carmichael-Olson, & Kartin, 2008).

Mental Health: Mental health and psychiatric problems are found at high rates among clinical samples of individuals with prenatal alcohol exposure. Indeed, even low levels of prenatal alcohol exposure have been shown to increase risk of clinically significant mental health issues in young girls. Other adverse pre-/postnatal risk factors commonly associated with prenatal alcohol exposure (e.g., polydrug exposure, abuse/neglect) may exacerbate early biologic vulnerability and increase the risk for secondary disabilities later in life (Streissguth et al., 2004).

Evaluation and Differential Diagnosis

Fetal alcohol spectrum disorder (FASD) diagnoses are based on the degree of presentation of (1) prenatal and/or postnatal growth deficiency, (2) the fetal alcohol syndrome (FAS) facial phenotype, and (3) central nervous system (CNS) abnormality, in the context of (4) prenatal alcohol exposure. There are several competing diagnostic systems used in the diagnosis of FASD. This discussion of FASD diagnosis will be structured around the most established system, the FASD *4-Digit Diagnostic Code* (3rd edition, Astley, 2004a: diagnostic guidelines, Lip-Philtrum Guides, and facial measurement software available at www.fasdpn.org). The other major diagnostic systems, the *Canadian Guidelines for FASD Diagnosis* (Chudley, Conry, Cook, Loock, Rosales, & LeBlanc, 2005) and the *Guidelines for Identifying FAS* from the Centers for Disease Control (CDC; Bertrand, Floyd, Weber, O'Connor, Riley, Johnson, Cohen, 2005), incorporate methods/tools from the *4-Digit Diagnostic Code* but use different diagnostic criteria.

In individuals being assessed for an FASD, as the presentation of key features of FAS move from not characteristic to highly characteristic of FAS, the *4-Digit Diagnostic Code* quantifies the expression of these features using a set of four 4-point Likert ranks that move from 1 to 4. The 4-digit code is created by combining these four Likert ranks from left to right in the order GROWTH, FACE, CNS, and PRENATAL ALCOHOL EXPOSURE (PAE). This numeric code is then used to assign an individual the appropriate FASD diagnosis. A brief summary of how each Likert rank is determined is presented below.

Growth

In the *4-Digit Diagnostic Code*, a GROWTH rank of 1 is given when both prenatal and postnatal height and weight are above the 10th percentile; a GROWTH rank of 2 indicates that either prenatal or postnatal height, weight, or both are above the 3rd percentile, but less than or equal to the 10th percentile; a GROWTH rank of 3 indicates that either prenatal or postnatal height or weight is less than or equal to the 3rd percentile; and a GROWTH rank of 4, severe growth deficiency, is given when both height and weight are less than or equal to the 3rd percentile during either the prenatal or postnatal period.

The FAS Facial Phenotype

Three key facial features are considered diagnostic of FAS: (1) small eyes with palpebral fissure length (PFL) more than 2 standard deviations (SD) below the mean for age; (2) smooth philtrum consistent with a rank 4 or 5 on the University of Washington Lip-Philtrum Guide; and (3) thin upper lip consistent with a rank 4 or 5 on the University of Washington Lip-Philtrum Guide. *At these values*, this constellation of features has high specificity for prenatal ethyl-alcohol exposure, and their presence can serve as a proxy indicator of prenatal alcohol exposure when exposure is unknown (Astley, 2006). In the *4-Digit Diagnostic Code*, a FACE rank of 4 indicates that all three facial features are present; a FACE rank of 3 indicates that deficits have been measured in all three features, but that one is only moderately impacted (e.g., PFL between -1 and -2 SD or a lip/philtrum Likert of 3); a FACE rank of 2 indicates that at least one feature is present; and a FACE rank of 1 indicates that there are no features present. Increased FACE rank is associated with increased risk/severity of CNS abnormality (Astley et al., 2009).

Central Nervous System (CNS) Integrity

The *4-Digit Diagnostic Code* includes a nested ranking of CNS abnormalities. This ranking quantifies both evidence of structural abnormality and severity of functional impairment. A CNS rank of 4 indicates that there is direct structural/neurological evidence (e.g., microcephaly, seizures) of a "definite" abnormality in CNS structure; a CNS rank of 3 indicates that there is a "significant" functional impairment in three or more domains of brain function (i.e., a deficit >2 SD on a standardized test); a CNS rank of 2 indicates mild to severe impairment in some domain of functioning; and a CNS rank of 1 indicates that the individual does not meet criteria for CNS ranks of 2 through 4.

Documenting Prenatal Ethyl-Alcohol Exposure (PAE)

The *4-Digit Diagnostic Code* includes 4 exposure ranks. A PAE rank of 4 indicates a "confirmed exposure to high levels of alcohol." Blood alcohol concentration greater than 100 mg/dL present on a weekly basis is given as a general guideline for defining "high levels" of exposure (Astley, 2004a, p. 43). A PAE rank of 3 indicates "confirmed exposure" of either unknown quantity or of an amount that falls short of a PAE rank of 4. A PAE rank of 2, "unknown exposure," indicates that exposure cannot be confirmed present nor absent. A PAE rank of 1 indicates a "confirmed absence of exposure from conception to birth."

Diagnostic Categories

When combined, ranks for GROWTH, FACE, CNS, and PAE provide a *4-Digit Diagnostic Code*. This code can then be converted into 1 of 22 possible diagnoses. Eight of these fall under the umbrella of FASD. Importantly, only a diagnosis of FAS implies a causal role for prenatal alcohol exposure in producing the impairments identified during assessment. This causal inference is grounded in the specificity of the FAS facial phenotype to prenatal ethyl-alcohol exposure. For this reason, both FAS diagnoses in the *4-Digit Diagnostic Code* require a FACE rank of 4 (FAS = GROWTH 2–4, FACE 4, CNS 3–4, PAE 2–4). The other 6 FASD diagnostic categories simply describe the degree and type of impairment and document prenatal alcohol exposure. The eight FASD categories from the *4-Digit Diagnostic Code* are:

Diagnostic category	4-Digit Code Criteria
FAS, alcohol exposed	GROWTH 2–4, FACE 4, CNS 3–4, PAE 3–4
FAS, alcohol exposure unconfirmed	GROWTH 2–4, FACE 4, CNS 3–4, PAE 2
Partial FAS, alcohol exposed	GROWTH 1–4, FACE 3, CNS 3–4, PAE 3–4
Sentinel physical finding(s) with static encephalopathy, alcohol exposed	GROWTH 3–4, FACE 1–2, CNS 3–4, PAE 3–4
Static encephalopathy, alcohol exposed	GROWTH 1–2, FACE 1–2, CNS 3–4, PAE 3–4
Sentinel physical finding(s) with neurobehavioral disorder, alcohol exposed	GROWTH and/or FACE 3–4, CNS 2, PAE 3–4
Neurobehavioral disorder, alcohol exposed	GROWTH 1–2, FACE 1–2, CNS 2, PAE 3–4
Sentinel physical finding(s), alcohol exposed	GROWTH and/or FACE 3–4, CNS 1, PAE 3–4

In the *Canadian Guidelines for FASD Diagnosis*, there are four FASD diagnoses. These diagnoses are based on the *4-Digit Diagnostic Code* and include two FAS categories: FAS, alcohol exposed; and FAS, alcohol exposure unconfirmed. The *Canadian Guidelines* use loosened facial criteria for FAS compared to the *4-Digit Diagnostic Code*. In the Canadian system, FAS can be diagnosed with the following ranks: GROWTH 2–4, FACE 3–4, CNS 3–4, and PAE 2–4. The *Canadian Guidelines* diagnosis of Partial FAS is also a broader category than in the *4-Digit Diagnostic Code* and includes a FACE rank of 2. The final diagnostic category in the Canadian system is alcohol-related neurodevelopmental disorder (i.e., ARND), which corresponds to the *4-Digit Diagnostic Code* diagnoses of static encephalopathy with or without sentinel physical findings.

The Centers for Disease Control's *Guidelines for Identifying FAS* has the most liberal FAS diagnostic criteria. CDC guidelines relax requirements for eye size to include PFL as large as 10th percentile. The CDC guidelines also relax criteria for CNS impairment and only require mild functional impairments (i.e., one standard deviation from the mean below the 16th percentile) in three domains of functioning for a diagnosis of FAS.

Treatment

Evidence-based interventions for fetal alcohol spectrum disorders (FASD) are emerging in the literature (Premji, Benzies, Serrett, & Hayden, 2007). Currently, primary prevention and direct individualized instruction to remediate specific learning and/or behavior problems are the primary tools available for treating individuals with FASD. Because children with FASD

commonly face disruptive challenges from their environment as well as complex neurophysiologic deficits (Coggins, Timler, & Olswang, 2007), successful treatment for this population demands multimodal services. These services should incorporate the knowledge and expertise of multiple disciplines (e.g., medicine, psychology, social work, special education, speech-language pathology, audiology, occupational therapy) in order to remediate both environmental barriers to success and central nervous system (CNS) dysfunction.

Primary Prevention: FASD are entirely preventable, and simple, low-cost informative counseling directed at women who use alcohol and are pregnant or thinking about pregnancy can decrease FASD risk (Bailey & Sokol, 2008). More intensive interventions for women at high risk who have already given birth to one child with prenatal alcohol exposure are also important. For example, the *Parent–Child Assistance Program* (Grant, Ernst, Streissguth, & Stark, 2005) provides participants with a paraprofessional advocate throughout their pregnancy and for 3 years after their child is born. The program helps mothers to decrease or eliminate their use of alcohol and/or to use family planning to prevent pregnancies when using alcohol. The program also promotes addiction recovery, supports economic and personal self-sufficiency, and helps participants connect to community resources.

Screening: Screening in high-risk populations has also been demonstrated to be effective in reducing FASD (e.g., Astley, 2004b). The state of Washington, for example, developed an effective program for screening children in the foster care system by using a review of medical records and facial photograph. In the program, those children with a positive screen for the FAS facial phenotype or for CNS damage (e.g., microcephaly) are invited for a diagnostic evaluation. Benefits from this kind of program include early diagnosis, earlier access to community supports and services, and increased caregiver knowledge regarding signs and symptoms of FASD.

Medical Interventions: Medical providers play an important role in FASD prevention, screening, diagnosis, and treatment. Early referrals to appropriate service providers are of particular importance and should always include formal vision and audiological screenings. Because efficacy and safety data on psychotropic medications are not generally available for the FASD population (Premji et al., 2007), there is little guidance for providers on how to target specific psychiatric or behavioral symptoms associated with FASD. Children with FASD may be at risk for paradoxical reactions and can be more sensitive to dosing. While research using animal models has revealed a number of promising medical approaches for protecting against or even reversing the long-term impacts of alcohol exposure, a more rigorous evidence base needs to be established before these approaches can play a major role in FASD treatment. Avenues being pursued include perinatal choline supplementation, prenatal folic acid, exercise training, and postnatal aniracetam treatment.

Educational and Behavioral Interventions: Because the impairments associated with prenatal ethyl-alcohol exposure are timing and dose dependent, the FASD population is extremely heterogeneous. As a result, no single behavioral or educational intervention approach will meet the needs of all those in the FASD population. Interventions need to be designed to target the specific needs of a particular individual with FASD in order to be most effective. Indeed, targeted and multimodal interventions are reported to make important changes across behavioral and learning domains for children with FASD while also improving caregiver outcomes (Kalberg & Buckley, 2007). Effectiveness may be maximized when interventions include a functional behavioral analysis to identify problem behaviors and antecedent events leading to those behaviors. This analysis can then be paired with caregiver and teacher education to "reframe" problem behaviors from a neurodevelopmental perspective. Accommodations and environmental modifications to compensate for specific neurological deficits are also important pieces of any effective intervention program (c.f., Olson et al., 2005). Social skills training can be another important element in any comprehensive intervention plan

and has been shown to be effective with school-age children with FASD. Targeted interventions for specific academic deficits are effective for children with FASD, and approaches that work with typically developing populations and children with other similar developmental disabilities should be effective interventions for individuals with FASD.

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Fetal Alcohol Syndrome

▶ Fetal Alcohol Spectrum Disorder

Fetal Anticonvulsant Syndrome

Usha Kini
Consultant Clinical Geneticist, Oxford Radcliffe Hospitals NHS Trust University of Oxford, Oxford, UK

Synonyms

Fetal antiepileptic drug syndrome (fetal AED syndrome); Fetal carbamazepine syndrome; Fetal phenytoin syndrome; Fetal valproate syndrome (FVS)

Short Description or Definition

"Fetal anticonvulsant syndrome" or "FACS" is the term used to describe the adverse effects of in utero exposure to anticonvulsant medication (e.g., phenobarbitone, phenytoin, carbamazepine, valproate, lamotrigine, etc.) and may manifest as dysmorphic facial features, major and minor structural anomalies, learning difficulties, and/or behavioral problems. Specific health and developmental problems are noted following exposure to specific antiepileptic drugs, e.g., phenytoin, valproate; the terms fetal phenytoin syndrome and fetal valproate syndrome are then used.

Epidemiology

Epilepsy is a common neurological condition affecting 0.6–1% of the population, and about a third of these are women of reproductive age. The majority of these women will be treated with antiepileptic drugs. Hence, approximately 1 in 160–250 pregnancies are exposed to antiepileptic drugs.

Phenobarbital was introduced for use as an antiepileptic drug (AED) in 1912, phenytoin in 1938, carbamazepine in 1961, and sodium valproate in 1964. In the early 1960s, there were reports from Germany (Janz & Fuchs, 1964), followed shortly later by reports from England, describing a pattern of major and minor anomalies affecting orofacial, cardiac, and skeletal development in children with prenatal exposure to AEDs. Gradually, reports of cognitive deficits and behavioral problems emerged.

The exact incidence of fetal anticonvulsant syndrome (FACS) is not clear, as not all individuals with in utero exposure to AEDs show the adverse effects. Also, variability in the manifestation of the adverse effects is noted even between individuals who have been exposed to the same dose of the same AED. Some AEDs like valproate may be used in the treatment of neuropsychiatric disorders such as bipolar disorders. Offspring of these women are also at a risk of developing the adverse effects, if exposed in utero.

Natural History, Prognostic Factors, and Outcomes

Natural history: The diagnosis of FACS is first suspected when ultrasound scan abnormalities are identified in a pregnant woman taking AEDs. However, the absence of scan abnormalities is not reassuring, as minor structural abnormalities may not always be picked up on scan and also because the scans do not usually provide any clues to the neurodevelopmental problems that may develop later on in life.

The rate of obstetric complications in women with epilepsy is thought to be increased, but no single complication has been identified as a common finding, and in most studies, the increased rate of obstetric complications has not been found to be statistically significant. An increased incidence of stillbirths has been reported but not proven to be significant statistically. The birth weight is usually average. There is an increased rate of admission to special care baby units due to withdrawal symptoms such as

Fetal Anticonvulsant Syndrome, Table 1 Congenital malformations associated with FACS

Systems involved	Abnormalities	Additional information
Neural tube defects	Spina bifida, anencephaly	1–2% risk of spina bifida with valproate, 0.5–1% with carbamazepine exposure
Congenital heart defects	Ventricular septal defects, atrial septal defects, patent ductus arteriosus	Seen with exposure to any AED
Orofacial clefts	Cleft lip and palate, cleft palate	Cleft palate has been reported with valproate exposure, but not cleft lip
Limb defects	Radial ray abnormalities, split hand malformation, polydactyly, tibial hypoplasia, camptodactyly	Seen with exposure to valproate; camptodactyly may be seen with other AED exposure as well
Genitourinary defects	Hypospadias, renal hypoplasia, hydronephrosis	More common with valproate exposure
Brain abnormalities	Agenesis of corpus callosum, porencephaly, hydranencephaly	Not frequently seen but have been reported
Eye abnormalities	Cataracts, optic nerve hypoplasia, iris defects	Iris defects have been reported with exposure to valproate only
Abdominal wall defects	Omphalocele	
Respiratory tract abnormalities	Laryngeal hypoplasia, abnormal lobation of lung, lung hypoplasia, tracheomalacia	More common with valproate exposure
Craniosynotosis	Trigonocephaly	Seen with valproate exposure only and is caused by premature fusion of the metopic suture; it may need surgical intervention

lethargy, irritability, poor feeding, jitteriness, and due to the presence of major malformations such as neural tube defects, heart defects, kidney abnormalities, etc.

Congenital malformations: There is a 6–7% risk of structural abnormalities at birth compared to 2–3% in the general population. This risk may be as high as 9% in children exposed to valproate (Table 1).

Minor anomalies: Inguinal hernia, joint laxity, club feet, capillary hemangiomas (birth marks), strabismus (squint), nail hypoplasia, digital hypoplasia, clinodactyly, and overlapping toes are more frequently seen compared to the general population.

Facial dysmorphism: There are subtle facial features that are described with exposure to different AEDs. There is an overlap in the facial features described with exposure to different drugs. Of these, the facial gestalt of valproate exposure is the most characteristic and is formed by the following features: thin arched eyebrows with medial deficiency, epicanthic folds, infraorbital grooves, broad nasal bridge, short anteverted nose, smooth long philtrum, and a thin upper lip (Kini et al., 2006).

Childhood medical problems: Visual and hearing problems are more frequently seen. Myopia (short-sightedness) and squints are more common; hearing problems include recurrent otitis media with effusion.

Developmental delay and learning difficulties: Global developmental delay may be seen, but speech delay alone is more common. Joint laxity and poor muscle tone contribute to poor coordination which may affect daily activities such as writing, dressing and undressing, riding a bike, swimming, etc. A large proportion of children have additional educational needs, and some of them may need one-to-one help much of the time (Adab et al., 2001). Many studies have shown that children exposed to AEDs such as carbamazepine, phenytoin, and even the newer drug, lamotrigine, have an IQ in the normal range

(Gaily et al., 2004; Mawer, Clayton-Smith, Coyle, & Kini, 2002; Dean, Hailey, Moore, Lloyd, & Turnpenny, 2002; Meador et al., 2011). However, the verbal IQ of individuals with valproate exposure appears to be significantly lower (Adab et al., 2004).

Behavioral problems: Autistic spectrum disorders have been reported more frequently with valproate exposure (Rasalam et al., 2005). Difficulty in developing peer relationships, sharing interests and enjoyment, using language in social communication, symbolic or imaginative play, changes to routine, and lack of emotional or social reciprocity have been reported commonly. Poor concentration and hyperactivity are other behavioral issues reported.

Prognostic factors: Several factors influence the severity of FACS: (1) Type of AED – Valproate exposure causes the most severe adverse effects. (2) Number of AEDs – Polytherapy (exposure to more than one AED) has a worse outcome than exposure to monotherapy (one AED). (3) Dose of the drug – Although the adverse effects have not been found to be dose related with exposure to other AEDs, exposure to /= 1,000 mg/day of valproate has been associated with more severe problems. Specific dose-related features of valproate are neural tube defects, radial ray abnormalities, obviously dysmorphic face, and a reduction in verbal IQ (Kini, 2006). (4) Time of exposure – First trimester exposure is associated with structural abnormalities. It is possible however that AED exposure through the second and third trimesters may have an effect on neurodevelopment as the brain continues to grow throughout the pregnancy. (5) Parental and environmental factors – The child's IQ is known to strongly correlate with the parental IQ and also with the socioeconomic status. Women with epilepsy may themselves have learning difficulties, contributing further to the phenotype. (6) Genetic factors – The teratogenic effects of AEDs differ between fetuses that have been exposed to the same dose of the same drug suggesting that maternal genetic factors may contribute. Studies have shown the contribution of genetic factors such as MTHFR (Kini et al., 2007), epoxide hydrolase genes, etc.

Clinical Expression and Pathophysiology

The clinical expression of FACS is very variable. The full spectrum of problems, i.e., dysmorphic facial features, major and minor malformations, developmental delay, childhood medical problems, learning difficulties, and behavioral problems, is rarely seen in a single individual with prenatal AED exposure. The presence of specific dysmorphic facial features may be a clue to early diagnosis. However, the facial gestalt is usually obvious in the most severely affected patients only. In fact, the facial features with exposure to some AEDs such as carbamazepine are very mild, adding to the difficulty of making a conclusive diagnosis. In addition, some individuals may present with subtle dysmorphic features and neurodevelopmental problems only. In these individuals, a diagnosis of "fetal anticonvulsant effects" should be considered.

Mechanisms of Teratogenicity

The exact mechanism by which AEDs cause their teratogenic effects is not known. Several plausible theories have been proposed:

1. Folic acid deficiency: AEDs such as phenytoin, phenobarbitone, and carbamazepine interfere with the intestinal absorption of folic acid, while valproate acts on its metabolism. Embryonic folic acid deficiency may disrupt gene expression, increase oxidative stress, and cause changes in protein synthesis (Wegner & Nau, 1992). In animal studies, the rate of neural tube defects was reduced in mice exposed to valproate, when high doses of folic acid and folinic acid were given. Alteration in expression of folate pathway genes has been demonstrated in mice following valproate exposure (Finnell, Wlodarczyk, Craig, Piedrahita & Bennett, 1997). Certain genetic polymorphisms in the folate pathway (*C677T* in the MTHFR gene) have been shown to be associated with a higher rate of malformations in AED-exposed children (Kini et al., 2007).
2. Oxidative stress: Intermediate metabolites of AEDs may increase oxidative stress in the

developing embryo, whose antioxidant defense mechanism is immature. This may damage the developing organs of the embryo. Embryonic oxidative stress due to free radical formation following hypoxia from phenytoin-induced embryonic bradycardia has been demonstrated in animal studies.

3. Arene oxide intermediates: Reactive intermediate metabolites of AEDs such as phenytoin and carbamazepine in the form of arene oxides may bind cell macromolecules affecting cell function and causing cell death. Arene oxides are detoxified by epoxide hydrolases; genetic variability in the activity of these enzymes may influence the susceptibility of the fetus to the adverse effects.
4. Histone deacetylase inhibition: Valproate is a potent histone deacetylase inhibitor. Histone deacetylases are enzymes that induce chromatin changes to enable transcription of genes. Histone deacetylase inhibition therefore leads to interruption in the cell cycle, growth arrest, and apoptosis (cell death). Valproate also causes demethylation of DNA which results in changes in gene expression and hence in congenital malformations.

Evaluation and Differential Diagnosis

FACS is a difficult diagnosis to make as it is a diagnosis of exclusion. There are no specific tests by which the diagnosis of FACS can be confirmed. Hence, basic tests such as karyotyping, fragile X syndrome, and urine metabolic tests should be offered to the patients to rule out other causes. Other fetal insults such as exposure to alcohol, toluene, and maternal diabetes may cause similar features as FACS and should be kept in mind while taking the medical history. Malformations related to specific drug exposure such as radial ray defects and trigonocephaly with valproate exposure may give a more definitive diagnosis, but other dysmorphic syndromes such as Baller-Gerold syndrome which have similar features need to be ruled out. This differentiation is important as it will impact on the recurrence risk given to these families. Referral to a clinical geneticist is therefore recommended. In those with a history of AED exposure, who present mainly with behavioral problems such as autistic spectrum disorder, evaluation for other syndromes such as Rett syndrome, tuberous sclerosis, and fragile X syndrome should be considered.

Treatment

Management

The management of FACS is symptomatic. Specific structural abnormalities, such as heart defects, kidney abnormalities, etc., may need monitoring, medication, or surgical intervention. Children with developmental delay may need help in the form of physiotherapy, speech and language therapy, and occupational therapy. Surveillance for childhood medical problems such as myopia and recurrent otitis media is recommended. Assessment for special educational needs should be carried out early and appropriate help instituted early to allow the child to develop to his or her full potential. Behavioral therapy and sometimes medication may be helpful in dealing with behavioral issues such as autistic spectrum disorder and attention deficit/hyperactivity disorder.

Prevention

For women with epilepsy, prepregnancy counseling should be provided so as to reduce the risk of FACS. Every attempt should be made to reduce the dose (e.g., valproate should be reduced to $<$1,000 mg/day) and number of AEDs to the minimal level required to control the epilepsy, and this should be done in liaison with the neurologist. Monotherapy is preferable to polytherapy. Valproate should be avoided wherever possible as this has been deemed to be the most teratogenic AED (Mawer et al., 2002). The weaning of the AED should be done well in advance of the pregnancy as seizures during pregnancy may have a detrimental effect on the health of the mother and baby.

High-dose folic acid (4–5 mg/day) is recommended during pregnancy, starting from

at least 6 weeks prior to conception. While intake of folic acid in the first trimester may help prevent congenital malformations, intake in the last two trimesters may help prevent neurodevelopmental problems due to continued development of the fetal brain throughout pregnancy. Antenatal scans should be offered at about 12 weeks (gross structural abnormalities such as anencephaly can be identified) and a detailed anomaly scan should be offered at 18–20 weeks. Malformation such as isolated cleft palate is likely to be missed on antenatal scans.

See Also

► Intellectual Disability

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Fetal Antiepileptic Drug Syndrome (Fetal AED Syndrome)

► Fetal Anticonvulsant Syndrome

Fetal Carbamazepine Syndrome

► Fetal Anticonvulsant Syndrome

Fetal Phenytoin Syndrome

► Fetal Anticonvulsant Syndrome

Fetal Valproate Syndrome (FVS)

► Fetal Anticonvulsant Syndrome

Fidelity of Implementation

- ▶ Procedural Fidelity
- ▶ Treatment Fidelity

Figurative Language

Maura Moyle and Claire Plowgian
Speech Pathology & Audiology, Marquette University, Milwaukee, WI, USA

Synonyms

Figures of speech; Metaphoric language; Nonliteral language

Definition

Figurative language is language that is used in nonliteral ways in order to achieve a special effect or meaning. Similes, metaphors, idioms, proverbs, and slang are examples of figurative language. The ability to comprehend and produce nonliteral language is an important development in school-age children. Figurative language is commonly used in a variety of communicative contexts, including casual conversation, teacher talk, and written texts.

Individuals with autism spectrum disorders (ASD) often have difficulty comprehending figurative language and may interpret figurative statements literally. Upon hearing the expression "It's raining cats and dogs," an individual with ASD may think that the speaker means that cats and dogs are literally falling from the sky. The meaning and appropriate use of figurative language may need to be explicitly taught to individuals with ASD.

See Also

- ▶ Metaphoric Language

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Figure-Ground Discrimination

Francesca Happé
MRC Social, Genetic and Developmental Psychiatry Centre at the Institute of Psychiatry, King's College London, London, UK

Definition

Figure-ground discrimination or perception refers to the ability to separate the elements of a visual image on the basis of contrast (e.g., light, dark), to perceive an object (figure) against a background (ground). A classic illustration of figure-ground perception is the Rubin vase, a simple black and white image which can be seen as two dark faces against a white background, or a white vase against a dark background. As well as simple properties, such as light and dark, more complex gestalt properties help separate the important objects in a scene from the background. The ability to discriminate figure from ground can also refer to the more general capacity to tell foreground from background information, or identify what is important versus what is less salient.

In ASD, superior ability is seen in some tasks requiring identification of parts within wholes, such as the Embedded Figures Task (see entry). This has been interpreted within theories postulating a detail-focused cognitive style in ASD, such as the "weak central coherence" account. Ambiguous figures (e.g., duck/rabbit), of which the Rubin vase may be considered an example, have been used in autism research to investigate flexibility (see ▶ Behavior Observation Scale) and also self-awareness. Findings are mixed; whether people with ASD show neurotypical patterns of

processing ambiguous figures depends in part on whether this is tapped through explicit or implicit tasks (e.g., Allen & Chambers, 2011). Links have also been found between ASD-like *traits* and the perception of multiple interpretations of ambiguous figures, perhaps reflecting figure-ground discrimination processes (Best et al. 2008).

See Also

► Global Versus Local Processing

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Figures of Speech

► Figurative Language

Financial Aid

► FAFSA

Fine Motor Development

Jane Case-Smith
Division of Occupational Therapy, School of Health and Rehabilitation Sciences, Columbus, OH, USA

Definition

Fine motor skills are also termed hand skills, fine motor coordination, object manipulation, or dexterity. Components of fine motor development include reach, grasp, release, in-hand manipulation, and bimanual coordination (Exner, 2010). Early development of these skills requires integration of the visual motor and somatosensory systems. Fine motor skills initially develop through the infant's sensory motor exploration, e.g., the infant's reach and grasp of objects. They are refined as the young child develops functional and pretend play skills that include manipulation, eye-hand coordination, and bimanual skill. In older children and adults, fine motor development involves learning the precise, complex sequences of movement required to perform writing, keyboarding, activities of daily living, leisure pursuits, and vocational tasks. Delays or disorders in fine motor development can relate to low muscle tone or poor strength; impairment in visual motor skills, bimanual coordination, or precision of manipulation (dexterity); and motor learning deficits or dyspraxia (motor planning problems).

Historical Background

In the past, researchers and practitioners did not broadly recognize that children with ASD have impaired fine motor development. The few studies that included motor development noted that motor skills were a strength in some children, particularly when compared to cognitive and communication skills (Gillberg et al., 1990; Mayes & Calhoun, 2003). Using parent report of motor development, Mayes and Calhoun found that 67% of their sample with ASD had normal motor milestones. Early research highlighted the exceptional manipulation skills of certain individuals with ASD, e.g., savants (Hermelin, Pring, & Heavey, 1994).

Contributing to the confusion about whether or not children with ASD have fine motor impairment, a number of studies found that the motor development of children with ASD was similar to that of children with general developmental delay (Provost, Lopez, & Heimerl, 2007), learning disabilities (Miyahara et al., 1997), or language delays (Landa & Garrett-Mayer, 2006). One

interpretation of these findings is that fine motor delays may reflect general developmental disability rather than define a core feature of ASD. More recent literature confirms that fine motor impairments are not only prevalent in ASD but are long lasting and persistent, affecting function throughout the lifespan (Vanvuchelen, Roeyers, & DeWeerdt, 2011).

Current Knowledge

Prevalence

Recent studies and systematic reviews have estimated the extent and prevalence of fine motor delay and motor coordination impairment in children with ASD. Ming, Brimacombe, and Wagner (2007) found high prevalence of motor impairment in a cohort of 154 children with ASD. In young children 2–6 years, 63% exhibited hypotonia, and in children 7–18 years, 38% exhibited hypotonia. Motor planning problems, i.e., Dyspraxia were common (34%) in this cohort.

When prevalence was examined using clinical data from standardized motor development scales, 63–68% of a sample of children with ASD had significant motor delays of more than 25% (Provost et al., 2007). Eighty-four percent of the children demonstrated at moderate fine motor delays. Although the fine motor skills of the young children in this sample were significantly lower than the typical children and the normative sample, their performance was similar to a comparison sample of children with developmental disabilities. This finding that motor skill development of children with ASD is similar to children with other developmental disabilities is consistent across studies (e.g., Bhat, Landa, & Galloway, 2011; Landa & Garrett-Mayer, 2006).

Using rigorous meta-analysis techniques with 83 studies of motor coordination in ASD, Fournier, Hass, Naik, Lodha, and Cauraugh (2010) examined 51 comparisons of children with ASD to typically developing peers. The standardized mean difference in motor coordination was substantial with an average effect size of 1.2. Given these robust findings, Fournier et al. (2010) concluded that motor coordination deficits are a cardinal feature of ASD.

Infants and Young Children

Beginning in the late 1990s, researchers who analyzed videotapes of infants later diagnosed with ASD reported that the infants showed fine motor differences and stereotypic arm movements (Adrien et al., 1993; Baranek, 1999). These retrospective studies revealed that at young ages, children exhibit deficits in specific movement patterns, hypotonia, hypoactivity, and aberrant motor patterns. These retrospective studies motivated researchers to complete prospective studies to examine fine motor skills in young children at risk for ASD (Bryson et al., 2007; Gernsbacher, Sauer, Geye, Schweigert, & Goldsmith, 2008; Landa & Garrett-Mayer, 2006).

Prospective studies of infants and toddlers at risk for ASD suggest that motor characteristics may provide a reliable early sign of ASD. When compared to the manual skills of a typically developing sample, those of children later diagnosed with ASD were significantly delayed (Gernsbacher et al., 2008). At 6 months, the infants were less skilled in reaching and grasping of objects, and at 12 months, they were significantly less skilled in stacking, scribbling, and banging. At 18 and 24 months, the toddlers were deficit in the fine motor skills important to communication (pointing) and play (using puzzles and blocks). Although this study identified motor delays in the youngest age groups (6 and 12 months), other studies have reported that significant differences in fine motor skills among children with ASD do not emerge until 14 months (Landa & Garrett-Mayer, 2006). As an early sign of ASD, motor delays or atypical movement patterns can contribute to its early identification.

In preschool age children, 3–4 years, Jasmin et al. (2009) found that 53% had fine motor delays. The mean fine motor composite score and mean scaled scores for the children with ASD were below the normal range, suggesting moderate fine motor impairment. These studies

conclude that young children with ASD exhibit mild to moderate delays in fine motor skill and estimate that between 53% and 84% of all children with ASD have fine motor delays or problems.

Based on the assumption that movements (e.g., gestures, oral motor skills, motor imitation) are fundamental to social interaction, researchers (Bhat et al., 2011) suggest that motor impairments may negatively influence children's acquisition of social and communication skills. The correlation of fine motor skills to development of social interaction and communication proficiency needs to be further researched to confirm or refute this hypothesis.

Older Children and Adolescents

Older children and adults with ASD, 7–32 years, have shown impairment in visual motor and manual dexterity tasks (Bhat et al., 2011; Green et al., 2002). When fine motor skills of older children with ASD were compared to children with learning disabilities (LD), they were lower in manual dexterity such as manipulation, drawing, and cutting (Miyahara et al., 1997). Manjiviona and Prior (1995) found that children, 7–17 years with Asperger's syndrome (AS) and high-functioning autism (HFA), demonstrated motor impairment in manual dexterity, including speed and accuracy of hand movement, eye-hand coordination, and bimanual coordination. They also demonstrated more movement errors. Hilton et al. (2007) found that children with Asperger's syndrome scored below the mean on a standardized assessment of motor skill. These findings are consistent with others who found a slower reaction time in skilled motor tasks (Mostofsky, Goldberg, Landa, & Denckla, 2000). They suggest that children with AS and HFA have difficulty with motor learning and motor planning.

Children with ASD often demonstrate poor handwriting legibility. When children with ASD were compared to typically developing children on a standardized handwriting assessment, the children with ASD had significantly lower legibility. Specifically, they showed impairments in forming letters, leading to lower overall quality and legibility (Fuentes, Mostofsky, & Bastian, 2009). One contributing factor to learning legible handwriting is using a consistent preferred hand to write. Frequently, children with ASD are delayed in establishing hand dominance and show mixed laterality or left-hand dominance. Hauck and Dewey (2001) found that children with ASD often do not establish hand dominance and that this lack of preference was not related to cognitive level or lack of motor skills. Studies from the 1980s found that 22–36% of children with ASD do not show a hand preference at ages that hand dominance should be established.

These studies suggest that precision of manipulation, quality of movement, and movement speed are lower in older children with ASD, and these differences can occur in children across the spectrum. The research literature suggests that the majority of children with ASD have significant motor delays or deficits and that these deficits can limit daily living skills, play, communication, and peer interaction.

Motor Imitation

Motor imitation is fundamental to learning and is important to both social and cognitive development (McDuffie et al., 2007). Motor skills are also learned, in part, through a child's imitation of others, i.e., attending to another person's movement and then repeating that movement. The child's goal may be social interaction or may be self-motivated imitation and mastery. Children with ASD have impairments in imitation that are believed to be core to their social impairment (Rogers, Hepburn, Stackhouse, & Wehner, 2003; Stone, Ousley, & Littleford, 1997). Because these children imitate less, they may have less practice of age-appropriate motor skills. On the other hand, fine motor delays may be a reason that children exhibit limited motor imitation. When a sample of children with Asperger's syndrome (AS) were compared to a matched sample of children with developmental motor delays, the children with AS had difficulty imitating gestures, particularly when the gesture required symbolic representation of an object (Green et al., 2002). Motor performance

using a standardized test accounted for 45% of the variance in imitation performance.

In further investigation of motor imitation, Vanvuchelen, Roeyers, and deWeerdt (2011) reported that boys with ASD had problems in motor imitation, whether or not they had cognitive impairment. They concluded that the motor imitation problems were more related to general motor competence than executive function. McDuffie et al. (2007) examined the correlates of motor imitation in children with ASD. They investigated the relationship of attention-following and nonimitative fine motor ability to motor imitation. Both fine motor ability and attention-following accounted for unique variance in motor imitation performance. Whether impaired imitation is a cause or result of lower fine motor skill, it likely relates to and influences fine motor development. Mostofsky et al. (2006) further clarified that motor imitation appears to be delayed but not disordered in children with ASD. Therefore, the delay in acquisition can catch up during late childhood. Previously reported cross-sectional studies of motor development noted that motor delays are common in young children with ASD and less prevalent in older children.

Motor Planning

Motor planning or praxis is the ability to plan and sequence novel or unpracticed movements to reach a goal. A number of studies have identified apraxia or motor planning problems in children with ASD. Using kinematic analysis for reaching, Forti et al. (2011) found that children with ASD were slower in planning their movements and needed to reorient their hand as they reached toward a target. They required more self corrections to accurately reach the target, indicating less automaticity and slower processing. These findings concur with Rinehart et al. (2001) who found that children with ASD are slow to prepare movement and have impaired anticipatory guidance.

When children with ASD were tested specifically on praxis, e.g., using gestures and tools, they made significantly more errors than children who were typically developing (Mostofsky et al., 2006). This study confirmed earlier findings that high-functioning children with ASD have impaired performance of gestures and problems in motor planning of precise movements (Rogers et al., 1986). Motor planning deficits appear to relate to perception of kinesthetic/spatial aspects of movement (i.e., sensory feedback) and planning of goal-directed actions (Mostofsky et al., 2006). These authors concluded that children with ASD can have deficits in conceptual planning or learning of movement.

Praxis problems or dyspraxia implies that a child has a distinct impairment in planning movement that cannot be accounted for by impaired motor execution or a general movement disorder. To analyze if children with ASD have basic motor skill deficits or dyspraxia, Dziuk et al. (2007) measured performance on a praxis test and on a basic motor skill assessment in a sample of children with ASD, aged 8–14 years. They found that basic motor skill had a significant effect on praxis performance. Although basic motor skills and dyspraxia in ASD were related, the dyspraxia could not be entirely accounted for by basic motor skill deficits. These authors concur with Mostofsky et al. (2006) that praxis deficits in ASD relate to children's perception of sensory feedback from movement, spatial representation of movement, and ability to learn movement.

Stereotypic Movements

In addition to motor delays and motor planning problems, children with ASD may exhibit abnormal movement or motor stereotypies. These include arm flapping or finger flicking and are characterized as repetitive, seemingly nonpurposeful movements (Bhat et al., 2011). Retrospective studies of children with ASD have found that toddlers exhibit atypical hand and finger movements and more stereotypical object play, such as spinning objects. These repetitive movements may interfere with motor development or may create a propensity for stereotypic movements through adulthood (Matson, Dempsey, & Fodstad, 2009). These motor stereotypies are believed to relate to sensory processing and may serve to lower or raise arousal.

Repetitive movements are also associated with lower levels of overall functioning. Cuccaro et al. (2003) found that children use stereotypic movements for sensory stimulation or as an adaptive function to cope with transitions or changes in the environment. In general, stereotypic movements appear to decrease with age. When useful to support a person's arousal level or ability to cope, stereotypic or repetitive movements may serve an adaptive, functional purpose.

Summary

Fine motor skills are fundamental to social skills and communication and are an important consideration when remediating the core deficits of ASD. A majority of children with ASD have delays in fine motor development, and motor impairments tend to continue through adulthood (Fournier, Hass, Naik, Lodha, & Cauraugh, 2010). Fine motor delays are more apparent in young children than in older children, suggesting that motor learning is a factor. Although the severity and prevalence of fine motor deficits appear to improve as children with ASD reach adulthood, motor coordination and motor planning impairments persist.

Fine motor development is among the variables that significantly influence function in children with ASD. Underlying motor delays can impair a child's ability to gesture, imitate, manipulate toys, and participate in social play. In older children, motor planning and motor coordination problems can relate to poor handwriting, school difficulties, and limited participation in sports and recreation. In adults, the fine motor deficits can restrict vocation or leisure pursuits. Researchers recommend that comprehensive intervention programs for persons with ASD include specific strategies to improve fine motor skills (Bhat et al., 2011; Fournier, Hass, Naik, Lodha, & Cauraugh, 2010).

Future Directions

Recent studies of children and adolescents with ASD suggest that they present with motor learning and motor planning impairments. It is not clear how the fine motor and motor coordination impairments relate to the cognitive features of ASD. Research is needed to better understand motor skill development and to further analyze how imitation, praxis, and motor learning influence fine motor performance.

With increased understanding of the basis for fine motor deficits, specific interventions can be designed. Efficacy research on motor skill interventions for ASD is scant (see Baranek, 2002), and rigorous trials are needed.

See Also

- ▶ Motor Planning
- ▶ Sensory Processing

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Finger Oscillation Test

► Finger-Tapping Test

Finger-Tapping Test

Lauren Schmitt
Psychiatry, UT Southwestern Medical Center, Dallas, TX, USA

Synonyms

Finger oscillation test; Finger-tapping test; FOT; FTT

Definition

The finger-tapping test (FTT) is a neuropsychological test that examines motor functioning, specifically, motor speed and lateralized coordination. During administration, the subject's palm should be immobile and flat on the board, with fingers extended, and the index finder placed on the counting device. One hand at a time, subjects tap their index finger on the lever as quickly as possible within a 10-s time interval, in order to increase the number on the counting device with each tap. The original procedure calls for five consecutive trials within a 5-point range for each hand, but variations include a total of six trials, in two sets of three. Results from FFT can be compared to age and gender normative data and may indicate motor impairment or lateralized brain dysfunction. The FTT is included in the Halstead-Reitan neuropscyhological test battery.

The FTT may be included as part of a comprehensive neuropsychological assessment for children with Autism Spectrum Disorder. Additionally, the FTT has been used in multiple research protocols as a standardized method for assessing motor speed as it relates to brain functioning and anatomy.

See Also

► Halstead-Reitan Neuropsychological Test Battery

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Finite Mixture Models

► Latent Variable Modeling

First "Period"

► Menarche

First Words Project

Moira Lewis
Speech-Language Pathologist, Marcus Autism Center Children's Healthcare of Atlanta, Atlanta, GA, USA

Definition

The First Words Project is a clinical and research program, staffed by speech pathologists, researchers, and interdisciplinary consultants, aiming to investigate developmental communication disorders while providing services for young children with communication delays and their families.

An overarching goal of the First Words Project is to emphasize the importance of early screening and diagnostic methods to detect social-communication delays and disabilities in young children and to provide assessment and intervention to children and families of toddlers with delayed development who otherwise may have not had access to knowledge of the need for services.

Historical Background

Led by Amy Wetherby, Ph.D., The First Words Project conducts longitudinal research in the Autism Institute in the College of Medicine at Florida State University. The goals of the project are to identify early red flags of autism spectrum disorders and other developmental disorders in children under 2 years of age, to develop and improve early screening tools and early detection of communication disorders, and to provide training and support to families of children with ASD and other developmental disorders affecting communication.

A number of early intervention screening and assessment recommendations have been developed based on the research and work with children and families since the beginning of the First Words Project, including the use of the Communication and Symbolic Behavior Scales (Wetherby & Prizant, 2001) to identify children 6–24 months of age at risk for developmental disabilities, including autism.

The First Words evaluation model follows two-step process designed to involve participating families, thereby reducing the need for clinicians and in-home providers. The goal here is twofold, providing parents and caregivers with training and resources for developing communication and social skills in the home and reducing costs surrounding early intervention home services.

Current Knowledge

First Words Project receives national and private funding to evaluate the communication development of children from 6 to 24 months of age. For children who are delayed in communication, intervention services are offered at no cost to families or service providers, based on availability of grant funds.

Future Directions

First Words is also developing educational materials and assessment tools for training of healthcare and childcare providers to improve early detection of communication problems in young children.

The First Words Project continues efforts to disseminate research, clinical tools, and information to all those involved in serving children with developmental disabilities and autism, including parents and caregivers. Online educational tools such as the *ASD Video Glossary*, an innovative web-based video application, are available to help parents and professionals learn more about the early red flags of autism spectrum disorders.

This ASD Video Glossary is available free of charge through the First Words Project website as well as the Autism Speaks website. The ASD Video Glossary contains over 100 video clips. Each video highlights diagnostic areas important to assess and consider when diagnosing ASD and/or differentiating ASD from other common childhood developmental delays. Each section of clips contains both child examples and short tutorials to help viewers detect subtle differences between typical and delayed development in young children and to spot the early red flags for ASD. As per the First Words Project website, all of the children featured in the *ASD Video Glossary* as having red flags for ASD are, in fact, diagnosed with ASD.

See Also

- ▶ Communication and Symbolic Behavior Scale
- ▶ Early Diagnosis
- ▶ Early Intervention

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Project Homepage

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Resource for Clinicians, Parents & Students: ASD Video Glossary (Free of Charge)

ASD video glossary also available at the Autism Speaks website: http://www.autismspeaks.org/video/glossary.php

ASD video glossary. http://firstwords.fsu.edu/ASDglossary/ASDabout.html

Fixed Interval

- ▶ Fixed Ratio

Fixed Ratio

Mary Jane Weiss
Institute for Behavioral Studies, Endicott College, Beverly, MA, USA

Synonyms

Fixed interval

Definition

Fixed ratio is a schedule of reinforcement. In this schedule, reinforcement is delivered after the completion of a number of responses. The required number of responses remains constant. The schedule is denoted as FR-#, with the number specifying the number of responses that must be produced to attain reinforcement. In an FR-3 schedule, 3 responses must be produced in order to obtain reinforcement. In an FR-15 schedule, 15 responses must be emitted before reinforcement is delivered. This ratio requirement (number of responses to produce reinforcement) is conceptualized as a response unit. In other words, it is the response unit (not the last response) that leads to the reinforcer (Cooper, Heron, & Heward, 2007; Skinner, 1938).

Applications of FR schedules can be found in business and in education. Some tasks are paid on an FR schedule (e.g., piecework). Students might receive a token after the completion of ten spelling words.

FR schedules are associated with a particular pattern of responding. After the first response, there is generally little hesitation between responses and the required responses are completed. After the ratio requirement is met, reinforcement is delivered. This is followed by a postreinforcement pause, in which the participant does not respond for a period of time following reinforcement. The size of the ratio influences the length of the pause. Duration of the postreinforcement pause is greater for large ratio requirements. Shorter pauses occur with small ratio

requirements. The FR schedule is visually depicted as a stepwise pattern.

FR schedules produce high rates of responding. It is associated with rapid response speed, since completion of the ratio requirement leads to reinforcement. In general, higher ratio requirements produce higher rates of responding (to gain more reinforcement). If the ratio requirements are too stringent, however, the rate of response can decrease. The maximum ratio must be individually determined and continually monitored. The determination of the maximal value is influenced by motivating operations, the individual's reinforcement history, and the quality of the reinforcer.

See Also

► Schedule of Reinforcement

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Flat Prosody

► Monotone

Flight of Ideas

Jan Van der Rutger Gaag
University Medical Centre St. Radboud, Karakter Child & Adolescent Psychiatry University Centre, Nijmegen, Utrecht, Netherlands

Synonyms

Derailment; Loose associations; Tangentially

Definition

Flight of ideas is a formal thought disorder. It refers to the expression of rapidly shifting thoughts in an individual. Thoughts are expressed through language. In individuals with "flight of ideas," thoughts are expressed in a highly associative manner. These associations may be linked to cue in the surrounding environment, elicited by associations stemming from the topic or merely by words. The individual's speech becomes incomprehensible, because he does not tune into the listener's needs by providing the listener with information that prepares him for changes of topic of conversation, thus making these better understandable.

Flight of ideas may occur in the course of a manic episode, during a psychosis, but is not uncommon in individuals with an autistic disorder of multiple complex developmental disorder. In the latter individuals, the question is raised whether this is really a thought disorder or a communicative deviance more related to expressive language disorder in these individuals. Others (Raymond Lake, 2008) consider thought disorder as an expression of mood disorder.

See Also

► Attention
► Linguistic Idiosyncrasies and Neologisms
► Mania

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Floor Effect

Hillary Hurst
Department of Psychology, University of Massachusett Boston, Boston, MA, USA

Definition

The floor effect is a statistical phenomenon in which most data points fall in the very low range of possible values ("bottom out" on the "floor" of the measure). The floor effect is often seen in assessment when a test is too challenging for a given target population. In turn, many subjects obtain scores that are clustered together at the bottom of a measure, with very few extending across the possible range of scores. This results in a skewed distribution with very limited variability. Floor effects greatly limit the clinical utility of measures. For example, if there are floor effects on a measure prior to and following an intervention designed for individuals with ASD, it will not be possible to observe any actual benefits that might have occurred for participants.

See Also

► Ceiling Effect

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Floor Time/Circle of Communication

Laura Bonazinga
Vermont Speech Language Pathology Private Practice Services, South Burlington, VT, USA

Definition

Floortime is one component of the Developmental, Individual-Difference, Relationship-based (DIR®) approach for engaging children with autism spectrum and related socioemotional disorders. The DIR® model provides an interdisciplinary approach for practitioners, educators, and families to comprehensively assess and provide intervention according to the unique developmental profile of the child. Floortime focuses on creating opportunities that foster learning through emotionally engaging and meaningful interactions while encouraging mastery of six foundational stages of social, emotional, and intellectual development (Greenspan & Wieder, 2006). It is typically implemented within the early toddler years. The initial goal is to discover the individual differences in the child's view of the world by identifying the child's interests, sensory modulation and processing, motor planning, and symbol formulation. The practitioner enters into the child's world by imitating or following his/her lead, attempting to draw the child into a shared interaction. Once an initial affective interaction is established, the clinician encourages a response, creating a *circle of communication* marked by an increase in intentional or purposeful two-way communication.

When the foundational skills of opening (initiating) and closing (responding to) circles of communication have been established, more complex areas of social, emotional, and intellectual capacities are fostered by creating small obstacles or variations during Floortime (Greenspan & Wieder, 1997). The DIR® approach, of which Floortime is a part, emphasizes individualized early intervention to treat the developmental differences often observed in

autism (Landa, Holman, & Garrett-Mayer, 2007) in consideration of the child's unique presentation of the social, cognitive, and processing deficits.

Historical Background

Stanley I. Greenspan first developed and presented the DIR®/Floortime approach for treating children with developmental and emotional disorders in 1992. Greenspan and colleagues, including, Serena Wieder, drew from interventions across disciplines to hone and perfect a comprehensive approach for intervening with nonprogressive developmental disorders, such as autism spectrum disorders (ASD), from a functional developmental framework instead of treating individual symptoms (Greenspan & Wieder, 1999).

The development of the DIR®/Floortime model has as its foundation the most influential research in the field of developmental psychology (ICDL, 2000) dating back to the early work of Jean Piaget (1896–1980) and Lev Vygotsky (1896–1934). Piaget hypothesized that there was a typical progression through human development whereby babies construct or build on innate "schemas" about the world as they progress through four stages of cognitive development. This theory, which became known as the constructivist view of learning, suggested that children have an innate or intrinsic motivation to interact with others and play, gathering and constructing knowledge about the world, through which they form the foundation for understanding abstract and symbolic representations of the world (Huitt & Hummel, 2003; Prelock, 2006). The significance of play in learning and development was also an idea shared by Vygotsky; however, he suggested a more sociocultural view of cognitive development (Prelock, 2006), as he posited that it was through the continued interaction with others that developmental changes occurred in individuals (Cole & Scribner, 1978).

In 2004, Greenspan and Shanker suggested that while play was important for general development, emotions and, more specifically, affective signaling between communicative partners during play directly allowed for individuals to develop their "first idea," a mental construct or representation of something intangible. They hypothesized that through the development of emotional understanding and reciprocal signaling, play and social interaction lead to symbolic reasoning and higher-order cognition. Drawing from the literature and research on the core deficits defining ASD, they developed a model of functional developmental capacities that when absent or reduced led to decreased formation of the foundations for relating, communicating, and thinking.

Historically, treatment approaches for ASD follow two models for intervention: behavioral approaches for reducing maladaptive or inefficient behaviors and cognitive approaches for training individual skills according to the absence or presence of expected abilities at a given age (Greenspan & Wieder, 2006). In the development of the DIR®/Floortime model, the authors defined the core goal areas based on relevant areas of developmental functioning that may or may not have been impaired in a given disorder. They reasoned that the very foundation of a comprehensive treatment program was to build upon a core set of skills that are requisite for the development of more broad and higher-cognitive skills. Using the authors' experience in emotional and social development, and their focus on treating a multifaceted neurodevelopmental disorder with interventions across several disciplines, they suggested the DIR® model, which included all relevant and critical areas of development, including systems responsible for emotional, social, language, motor planning, and sensory processing and modulation.

Rationale or Underlying Theory

The development and expansion of play skills has gained recognition as serving an integral role in the acquisition of cognition, language, emotional intelligence, and social skills in children (National Research Council [NRC], 2001; Prelock, 2006). Further, it has been suggested

that a connection with an adult, during play and social routines, scaffolds and informs the child's ongoing development of meaning, beliefs, and values for the social context, thereby influencing the child's ability to expand his/her knowledge through experiences with objects, actions, and events (Greenspan & Shanker, 2004; Greenspan & Wieder, 2006; NRC, 2001; Prelock, 2006). As intervention techniques begin to broaden and include more intensive and early intervention programs, some suggest a developmental and relationship-based approach may be more effective for treating children with ASD.

Limited ability to relate and emotionally connect within the social context is inherent in the core deficits often observed in ASD. This limitation, coupled with a reduced or lack of initiation and/or attention toward others, restricts the child's ability to form ideas and expand their knowledge of reciprocal and symbolic play (Greenspan & Wieder, 2006; Mundy, Sigman, & Kasari, 1990; Prelock, 2006). Greenspan and Shanker (2004) refer to the early development of a "first idea" as a primitive construct that leads to the integration of symbolic, linguistic, and intellectual knowledge. This, in turn, allows for the child to appreciate the ever-changing social context and navigate the dynamics of play leading to increased interpersonal communication.

In 1997, Greenspan and Wieder presented a paradigm of Functional Developmental Levels (FDLs) that reflected expected capacities in relating, emotional understanding, and symbolic representation throughout six stages of typical development. They posited that typical development progresses through successive mastery of each stage and is essential for normal development of emotional reasoning, a matured sense of self as well as higher-level cognitive development. Later these stages were expanded to include some higher-level aspects of emotional understanding, affective reasoning, and cognition and are now referred to as Functional Emotional Developmental Capacities (FEDCs) (Greenspan & Wieder, 2006). Consequently, for the purpose of clarity, they will be referred to as FEDCs and are described in more detail below.

Stage one of the FEDCs is the capacity for *mutual or shared attention* or the ability to regulate attention and behavior while monitoring and processing sensory information from the environment and one's own body. Self-regulation is one component of this stage whereby a child learns the association between a physical state and an emotion. Another component is the emergence of joint attention, typically occurring between 6 and 9 months of age, although children begin processing sensory information shortly after birth. The capacity for joint attention is also viewed as a prerequisite skill for later language and communicative development (Mundy, Sigman, & Kasari, 1990). Successful joint attention between caregiver and child begins to foster the child's knowledge for discriminating pleasurable from unpleasurable experiences (Greenspan & Wieder, 2006). It is through initial sensory processing paired with increased attention to others during mutually shared experiences that a child's emotional understanding begins to deepen.

Stage two typically emerges between 2 and 5 months of age and is marked by *engagement and relating with others*. As a child's ability to engage in emotional interactions increases, so does the ability to discriminate between an interaction with another social being and an inanimate object. Through this discrimination, the child recognizes patterns about his/her world and demonstrates an emerging ability to assign meaning to various acts of communication and symbolic representation. During this stage, a child's genuine desire for engaging and relating begins to emerge, without signs of distress or withdrawal, affording more opportunities for increased reciprocal communication.

The third stage, *intentionality and two-way communication*, is marked by the child beginning to interpret emotions as "signals" of communication. By 6 months of age, caregivers and babies begin to engage in intentional or purposeful communication where caregivers read and respond to children's emotions and, in turn, require them to read and respond to theirs. The result is a back-and-forth flow of emotional signaling, which is referred to as a "circle of communication." As the

child approaches 8 months of age, circles of communication are mastered and become longer and more frequent, expanding the child's understanding of interactions as following a causal and logical pattern, mimicking the turn-taking pattern of more complex and later developing skills such as in maintaining a conversation.

Between 9 and 18 months of age, children begin to master the fourth stage of the FEDCs, *social problem solving, mood regulation, and formation of a sense of self*. In this stage, two-way reciprocal communication is used to solve problems and achieve a desired goal (e.g., pointing to direct others' attention or to obtain a desired object). Developing emotional maturity at this stage allows for an increase in *shared social problem solving*, where a child considers not only his/her own thoughts and beliefs but also the thoughts and desires of another individual and engages in negotiations during play and communication. Further, the emergence of skills in *regulating mood and behavior* allows a child to rapidly recognize and adjust his/her emotional signals. It is during this stage that children begin to develop an understanding of cause and effect and become more aware of complex patterns of communication leading to an early *sense of self*. They begin to refer to themselves as "I" or "me" and others as representing "you." This sense of self and others leads to early empathetic reasoning or a theory of mind.

By one and half years of age, the child's development progresses into the fifth stage of FEDCs, *creating symbols and using words and ideas*. Through joint attention and complex emotional signaling the child learns to separate perceptions from actions and begins to pair mental constructs, or images, with a linguistic or gestural symbol. Communication becomes meaningful and pretend play becomes more imaginative (e.g., a block as a car or a banana as a telephone). A better understanding of the function of words and symbols emerges, and a child learns that words and actions communicate ideas, convey mental and physical states, and signal intent.

As a child progresses through the second year of life, at approximately two and a half years of age, he/she enters into the sixth stage of FEDCs, *emotional thinking, logic, and a sense of reality*. The child begins to connect symbols and ideas in a logical manner. The ability to link an internal to an external idea emerges as well as a child's sense of events across time. As the child's logical thinking expands, higher-cognitive skills emerge such as inventing characters or games and following the rules of another's game.

Assessment of the child's functional capacity at each of the aforementioned stages forms the core of the DIR® model and further informs treatment at each stage through child-centered, interactive, and guided Floortime sessions. At the heart of the intervention is the premise that through maturing emotional development, a child learns to construct his/her knowledge of the world, learning to construe multiple representations or symbols for events, eventually leading to reciprocal social competence through an understanding of the self and others (Greenspan & Wieder, 2006).

Goals and Objectives

Two common goals are at the heart of the Floortime approach: (1) following the child's lead, identifying that child's unique processing style and/or interests in the environment, and (2) bringing the child into a shared world, using two-way intentional and reciprocal interactions (Greenspan & Wieder, 2006). In addition, three cornerstones have been identified as essential to participation in the program. First, relationships are formed through emotional and meaningful interactions and are the context for learning that leads to the development of language, cognition, and social skills. Second, motor and sensory processing capacities are varied in children, and these variations affect the emotional processing that goes along with interpreting and modulating sensory information. In essence, children vary in the way they process information, thereby making their availability for learning and relating individually unique. Finally, progress across all developmental areas should be viewed as interrelated.

Considering these three cornerstones the main goal of the Floortime approach is to increase a child's capacity for thinking of themselves as intentional and interactive individuals as they progress through mastery of the six FEDCs described above (Greenspan & Wieder, 2006). The initial step is to administer a comprehensive interdisciplinary assessment of the child's developmental capacities within the context of his/her unique sensory processing profile, the dynamic of his/her family relationships, as well as his/her interactive patterns (Greenspan & Wieder, 1999). A functional developmental profile is derived from clinical observations of interactions with both clinicians and parents, chart reviews, interviews with other care providers, assessments across disciplines (speech-language pathology, occupational therapy, physical therapy, etc.), a developmental history, a review of current programs, as well as a biomedical evaluation.

Following the generation of a functional developmental profile, a comprehensive DIR® intervention program is developed with goals that are individualized to the unique profile of the child while considering functioning across the six areas of functional emotional development. The foundational goal is meeting the child's need for a connection with others by providing stable and secure relationships with providers. Upon the foundation of ongoing emotional interactions with consistent care providers, individualized therapeutic goals can be designed to specifically address the child's functional developmental level, his/her individual differences in sensory processing, motor planning, and sequencing, as well as specific skills in the areas of speech and language and general education. Continuation of team and family consultation and ongoing assessment of the presentation of core deficits as well as the functional developmental capacities further informs the progression and predicted outcome of a DIR® program.

Treatment Participants

The DIR®/Floortime approach is designed for children with socioemotional deficits as often observed in ASD. It is appropriate for children who are verbal or nonverbal and with a diagnosis of ASD (Ingersoll, Dvortcsak, Whalen, & Sikora, 2005). While there are no prerequisite skills necessary to begin a DIR® program, researchers have found that children who benefit most from this treatment tend to present as having a specific profile, quite possibly allowing them to be more capable of learning to relate and interact with intentionality and symbolically. The children who have a more positive prognosis with the DIR® approach are those that have demonstrated some capacity for joint attention and emerging ability for using gestures in an affective interaction. In addition, they typically evidence some form of functional language use or some degree of pretend play (Greenspan & Wieder, 1997). Lastly, while this approach has typically been implemented with toddlers, recognizing early intervention leads to better outcomes for children who are identified early as having developmental delay (Landa et al., 2007), it can also be used with older adolescents with autism who have some functional capacity at each of the six FEDCs, but continue to struggle with higher-level thinking. For older children, this model is typically implemented within a school program or in community environments that purport to foster and facilitate social interaction (Greenspan & Wieder, 2006).

Treatment Procedures

A comprehensive DIR® program typically involves a home-based program, an educational setting-based program, as well as the addition of specialized therapies such as speech and language, occupational, physical, and/or certain biomedical therapies, depending on the needs of the child (Greenspan & Wieder, 2006). The mean age for entry into a DIR® program is typically 36 months, ideally beginning as early as 22 months (NRC, 2001) or immediately following identification of key strengths and challenges in affective reasoning, sensory modulation and processing, symbol formulation, and motor planning through a comprehensive, interdisciplinary assessment.

Floortime training and mentoring can be implemented in the home or clinical setting by a trained professional in the discipline of speech-language pathology, psychology, or a person with training and expertise in the application of the Floortime approach. Eventually parents are trained to implement these techniques as well. Duration of intervention ranges from 10 to 25 h per week (NRC, 2001). One pilot study has suggested that implementation of the approach by parents for as little as 15 h a week can potentially produce both positive outcomes and remain cost effective for families (Solomon, Necheles, Ferch, & Bruckman, 2007). However, one limitation noted that, for children whose parents spent less than the prescribed time implementing the program, there was less progress in the functional developmental level observed, supporting the importance of duration and frequency of treatment, as suggested by the NRC (2001).

The most common technique applied in the DIR® model is Floortime interactions, which occur both at home between clinician and child as well as between parent and child. These sessions are designed to be approximately 20 min per session and are ideally spontaneously interspersed throughout the day for a frequency of at least eight sessions. Once the child has mastered the initial functional developmental capacities required for interaction (e.g., joint or shared attention and can complete multiple circles of communication), peer play sessions can be introduced to facilitate shared problem solving, play negotiation, and the exchange of symbols, gestures, or words. Additional structured and semistructured sessions are implemented to address specific skills in the areas of motor, sensory and spatial skills, balance and coordination, and visuospatial functioning.

There is a clear interdisciplinary and collaborative approach inherent in the DIR® approach, allowing a plan to be individualized to each child's unique functional developmental profile. Specialized therapeutic services, such as speech-language pathology, occupational, and/or physical therapy, are implemented, as needed, in addition to Floortime sessions. Several components of the DIR® model, however, are implemented or facilitated by parents. The affect-based curriculum is one such component, which is specifically designed to include highly motivating semistructured, structured, and spontaneous activities that are based on a common goal of sharing affect and attention while simultaneously focusing on the foundations of language, such as phonology, syntax, grammar, and semantics (Greenspan & Wieder, 2006).

Efficacy Information

Efficacy of the DIR® model has been measured using the *Functional Emotional Assessment Scale* (FEAS) for the child and the caregiver, by clinical measurement of the Functional Emotional Development Level (FEDL), or the child's functioning according to the six developmental levels suggested by Greenspan and Wieder (1997) as well as in overall presentation and severity of symptoms in autism. In a retrospective chart review of 200 cases conducted by Greenspan and Wieder (1997), it was reported that children's progress could be organized into three outcome areas of the intervention. Fifty-eight percent of the cases were interpreted as having *good to outstanding* outcomes, 25% of the cases had *medium outcomes*, and 17% demonstrated *ongoing difficulties*. The conclusions of this review suggest that a small subgroup made progress in learning to relate to others, communicate, think creatively and abstractly, and showed an increase in emotional understanding and empathy, while other cases, measured as having more severe symptoms, continued to have difficulties despite intervention. Limitations noted in this chart review included variability in the assessment procedures used for the 200 children, the limitations associated with a case study design, and a small sample size, therefore suggesting that these results could not be definitively generalized to the greater population with ASD.

Later, a 10–15-year follow-up of a subgroup of children with ASD who received the DIR® intervention program was conducted by the same authors. This study revisited 16 in the

subgroup of children who were reported to have *good to outstanding* outcomes from the earlier chart review of 200 cases (Greenspan & Wieder, 1997). Conclusions from the follow-up study indicated that most individuals from that subgroup maintained the progress in the areas of social relatedness, creative thinking, and empathy, suggesting that early implementation of the DIR® approach may have effects later into adolescence. Limitations included questions about the generalizability to the larger population of children with ASD, the sample size, and extraneous factors that could not be controlled for, such as other interventions implemented across the years (Wieder & Greenspan, 2005).

In 2005, Ingersoll and colleagues examined the effects of the Developmental Social-Pragmatic model (DSP) on the use of spontaneous functional communication of three children with ASD between the ages of 2 and 3 years of age. Through a single subject-multiple baseline design, trained therapists implemented a DSP model of treatment, which included the DIR®/Floortime approach, Hanen program, SCERTS model, and responsive teaching approaches. The children received treatment twice weekly for 10 weeks, followed by a 1-month follow-up to assess generalization. The authors found that two of the three children demonstrated gains in spontaneous language use after treatment began that was maintained and generalized across new treatment settings. The third child exhibited gains in spontaneous communication; however, he also demonstrated an increase in his baseline, thereby confounding the interpretation that his gains could be solely attributed to the DSP intervention. The conclusions of this study speak to the strength in the application of this research model and support evidence for DSP models implemented by specialists in the field of speech and language as well as the use of spontaneous functional communication as a preferred outcome measure. In addition, progress in spontaneous communication following the use of the DSP model for both verbal and nonverbal children with ASD was found. Limitations included the absence of a follow-up session for one child, a failure to explore generalization to more naturalistic settings, as well as an inability to control for the therapists being the sole providers of the DSP approach throughout the duration of treatment.

Further support for the DIR®/Floortime approach was found by Solomon, Necheles, Ferch and Bruckman (2007) who conducted a pilot study that focused on training and consultation for parents implementing the Floortime approach across an 8- to 12-month program. Their study found significant gains for 45.5% of the children enrolled as indicated by an increase in their overall FEAS score. In addition, no significant changes in the caregivers FEAS scores were observed, suggesting that parent's participation was appropriate for implementing the relationship-focused intervention. Mahoney and Perales (2005) also reported that the parent implementation of the relationship-based intervention model for children with pervasive developmental disorder (PDD) was effective, encouraging the caregivers to provide more responsive interactions with their child, which in turn led to more than two thirds of the children demonstrating an increase in their targeted developmental behaviors. The research published on the DIR® model suggests that parent education, training, and provider roles are factors that positively influence the outcome of changing pivotal behaviors in children treated with this approach (Mahoney & Perales, 2005; Solomon et al., 2007). Another factor affecting the outcome of treatment is the amount of time spent with the child implementing Floortime (NRC, 2001; Solomon et al., 2007).

Finally, although there is some research supporting positive outcomes for a subgroup of children who participate in an early DIR® intervention, most are case studies and chart reviews indicating the need for further and more stringent research models. Additionally, the National Standards Report (2009) indicated that developmental and relationship-based approaches are considered to have emerging evidence, further highlighting the need for more research examining the outcomes of such programs for addressing the individuals with ASD.

Outcome Measurement

The DIR® model uses two outcome measures, the *Functional Emotional Assessment Scale (*FEAS) and the *Greenspan Social Emotional Growth Chart* (SEGC), to assess baseline levels at the initial evaluation, measure progress at follow-up sessions, and establish outcomes of the therapy (NRC, 2001). The initial evaluation of a child should include developing a profile of that child's individual functioning when compared to the six stages of emotional development. Visual-spatial and motor planning should also be assessed to determine the child's ability to process information (Greenspan & Wieder, 2006).

The FEAS is a criterion-referenced tool for measuring functional emotional capacities in children ages 7 months through 4 years of age. More specifically, the FEAS systematically assesses the child's ability to negotiate play activities in relation to people or objects, to self-regulate mood and attention, to form attachments with the caregiver, to use emotional and communicative reciprocity, and to represent emotions, ideas, and emotional thinking within the context of play interactions from videotaped samples. A caregivers' capacity to support development in the aforementioned areas of emotional development is also assessed (Greenspan & DeGangi, 2001). In addition to the two measures noted above, continued assessment of the child's functioning in each of the stages of FEDCs informs the direction of treatment as well as progress across developmental areas (Greenspan & Wieder, 2006).

Qualifications of Treatment Providers

The DIR®/Floortime model is considered to be a comprehensive and individualized approach that can be developed and adjusted according to the child's individual differences, family dynamics, and level of development. The development of the program requires participation of several disciplines: speech-language pathologists, occupational or physical therapists, early educators and/or special educators, and a therapist trained in intensive Floortime work (Greenspan, 1992a as cited in Greenspan & Wieder, 1997).

Although multiple care providers with expertise in several disciplines are required for the development and implementation of a comprehensive DIR® program, parents play an essential role as providers of spontaneous Floortime sessions throughout the day (Greenspan & Wieder, 2006). Additionally, while several specialists with relevant training in his/her area of expertise may cooperate in implementing the Floortime approach, the application of a comprehensive DIR® approach should be managed and overseen by a specialist from a relevant discipline, who has training in applying DIR® theory. The Interdisciplinary Council on Developmental and Learning Disorders (ICDL) provides a few training opportunities for licensed professionals from several disciplines, such as speech-language pathology, occupational and physical therapy, as well as social work, to name a few. Among the training opportunities offered, there is a DIR® introductory level course, a DIR® beginning course, as well as an intensive DIR® certification program. Information regarding further information and training opportunities on the DIR® model can be found at www.icdl.com.

See Also

- ▶ Affective Development
- ▶ Emotion Regulation
- ▶ Relationship Development Intervention (RDI) Model
- ▶ Sensory processing

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Fluency and Fluency Disorders

Diane R. Paul
Clinical Issues in Speech-Language Pathology, American Speech-Language-Hearing Association, Rockville, MD, USA

Synonyms

Cluttering; Disfluency; Dysfluency; Nonfluency; Stammering; Stuttering

Definition

Fluency is the aspect of speech production that refers to the continuity, smoothness, rate, and effort with which language is spoken.

Stuttering is the most common fluency disorder. Stuttering is an interruption in the forward flow of speech and is characterized by atypical rate, rhythm, and disruptions of speech. More specifically, stuttering refers to speech that contains whole-word repetitions, part-word repetitions, sound prolongations, or silent blocks.

These may be accompanied by reactive secondary behaviors/mannerisms (i.e., behaviors used to escape and/or avoid stuttering) and physiological reactions such as blushing and heart pounding.

Approximately 1% of the general adult population stutters. Among children, reports vary between 2.5% and 5%. Stuttering is more common among males than females. Among elementary school-age children, it is estimated that boys are three to four times more likely to stutter than girls.

All speakers have disfluencies, which may include hesitations for language formulation including silent pauses as well as insertion of word fillers (e.g., "The color is *like* red") and nonword fillers (sometimes called *interjections*, e.g., "The color is *uh* red"); other examples also may include whole-word repetitions (e.g., "*But*-but I don't want to go") and phrase repetitions or revisions (e.g., "*This is a-* this is a problem"). These may be considered "more typical" disfluencies (produced by normal speakers) in acceptable quantities.

Less typical, more stuttering-like dysfluencies include *part-word* or *sound/syllable repetitions* (e.g., "Look at the *buh-buh*-baby"), *prolongations* (e.g., "*Sssssss*ometimes we stay home"), and *blocks* (i.e., silent fixations or inability to initiate sounds). In addition, stuttering-like dysfluencies are usually accompanied by decidedly greater than average duration, effort, tension, or struggle than are typical disfluencies.

Stuttering most often begins during childhood. A large majority of early stuttering remits without clinical intervention (between 65% and 89%).

Similarly, some young children may go through a period of excessive disfluency. However, they go on to display typical fluency skills as their language abilities develop.

Stuttering can greatly interfere with school, work, or social interactions. Children and adults who stutter may report fear or anxiety about speaking and frustration or embarrassment with the time and effort required to speak. Despite popular beliefs, emotional problems or emotional trauma do not cause stuttering. However, dealing with stuttering can result in significant emotional reactions and avoidance behavior.

The cause of stuttering is presently unknown but may reflect subtle underlying differences in brain structure and function, particularly when an individual is processing and producing language. Stuttering may have a genetic component and the risk of stuttering is elevated in families having other members with persistent stuttering. Stuttering can also occur following neurological trauma (e.g., head injury) and following psychological trauma (e.g., posttraumatic stress disorder).

Stuttering can co-occur with other disorders. For example, although there is little systematic evidence describing disfluency in autism spectrum disorder (ASD), increasing numbers of case reports indicate atypical, stuttering-like behaviors that are additionally distinguished by unusual features, such as repetition of final segments of words (Klin, Volkmar, & Sparrow, 2000; Paul, Shriberg, McSweeny, Cicchetti, Klin & Volkmar, 2005; Shriberg, Paul, McSweeny, Klin, Cohen & Volkmar, 2001).

The term *disfluency or dysfluency* often is used synonymously with *stuttering*. However, the term *disfluency* refers to both normal and abnormal breaks in the forward flow of speech, while *dysfluency* refers only to disordered fluency. *Stammering* is synonymous with *stuttering* and is the common term for the disorder in Great Britain. In North America, the term *stammering* is rarely used. *Secondary behaviors* are also known as *accessory behaviors, secondary mannerisms, secondaries, concomitant behaviors*, or *extraneous behaviors*. For example, a person may tap his foot or nod her head while talking in a misplaced effort to speak fluently. Secondaries usually start coincidentally and then become incorporated when the person notices an improvement in fluency. If a behavior appears to help, it may persist, even though it loses its effectiveness.

Cluttering is another fluency disorder. Cluttering is characterized by a rapid and/or irregular speech rate, and excessive disfluencies, which are usually of the more typical type

(revisions and overuse of fuller words, such as "*um*"). The speech of a person who clutters often contains pauses in unusual places and unusual prosody. Other symptoms may include language or phonological errors.

Speech-language pathologists (SLPs) are the professionals who perform fluency assessments and intervention individually or in groups. SLPs also serve as members of collaborative teams that may include the individual, family/caregivers, educators, and other relevant persons.

See Also

- ▶ Cluttering
- ▶ Disfluency
- ▶ Dysfluency
- ▶ Nonfluency
- ▶ Speech-Language Pathologist (SLP)
- ▶ Stammering
- ▶ Stuttering

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Fluent Aphasia

▶ Wernicke's Aphasia

Fluent Speech

▶ Verbal Communication

Fluid Intelligence

Francesca Happé
MRC Social, Genetic and Developmental Psychiatry Centre at the Institute of Psychiatry, King's College London, London, UK

Synonyms

Fluid reasoning

Definition

Fluid intelligence (abbreviated Gf) is the ability to reason quickly, think abstractly, and problem-solve, independent of acquired knowledge. Cattell (1971) proposed two factors underlying general intelligence: fluid and ▶ crystallized intelligence. Tests of fluid intelligence tap current reasoning ability and are considered to be more "culture-fair," being less affected by differences in learning experience or test familiarity. Raven's Progressive Matrices, and other tasks with novel stimuli, are thought to tap fluid intelligence.

People with ASD often do well on certain tests thought to measure fluid intelligence, such as Raven's Matrices (Hayashi et al., 2008) and Block Design (Shah & Frith, 1993). Interpretation of these peaks in an uneven intelligence test profile has varied: many fluid intelligence tests tap visuospatial problem solving, which

may be superior to verbal reasoning in many people with autism. Alternatively, Dawson et al. (2007) have suggested that good Raven's Matrices scores reveal the true level of intelligence in autism, underestimated by standard IQ tests. Scheuffgen et al. (2000) suggested that people with autism may have good potential processing efficiency (as seen in fluid intelligence tests), not reflected in standard IQ test performance because of failure to acquire skills and knowledge through socially mediated learning. Fluid intelligence is thought to include several processes, only some of which may be superior (relative to own profile or others') in autism; visualization and pattern recognition, for example, may be areas of skill, while ideational fluency or generativity (see ▶ Behavior Observation Scale) may not.

See Also

▶ Behavior Observation Scale
▶ Crystallized Intelligence

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Fluid Reasoning

▶ Fluid Intelligence

Fluoxetine

Carolyn A. Doyle[1] and Christopher J. McDougle[2]
[1]Indiana University School of Medicine, Indianapolis, IN, USA
[2]Lurie Center for Autism/Harvard Medical School, Lexington, MA, USA

Synonyms

Brand names: Prozac; Prozac Weekly (once-weekly dosing); Rapiflux; RECONCILE; Sarafem; Selfemra

Indications

Fluoxetine is FDA-approved to treat major depressive disorder (MDD) in both adults and in children aged 8–18 years old, obsessive-compulsive disorder (OCD) in both adults and children aged 7–17 years old, and bulimia nervosa in adult patients. In these disorders, fluoxetine can be used both acutely and as maintenance therapy. It is approved for adults only in the acute treatment of panic disorder with and without agoraphobia. In combination with olanzapine, marketed as Symbyax, it is approved for the acute treatment of depressive episodes associated with bipolar I disorder and treatment-resistant depression in adults only (Lilly 2009).

Fluoxetine is not approved for the treatment of autism spectrum disorders (ASDs), which include the DSM-IV-TR diagnoses of autistic disorder, Asperger's disorder, and pervasive developmental disorder, not otherwise specified (PDD-NOS). The decision to use fluoxetine in the treatment of established clinical indications that may co-occur with ASDs, as mentioned above, or for commonly observed symptoms of ASDs, such as hyperactivity, inattention, irritability, aggression, repetitive behaviors, and social impairment, can be made on an individual basis by the treating practitioner. This will be discussed in more detail in the "Clinical Uses" section.

Mechanisms of Action

Fluoxetine is a selective serotonin reuptake inhibitor (SSRI). Normally, serotonin is released from the presynaptic axon terminal to travel across the synaptic cleft and attach to the postsynaptic axon terminal. Any excess serotonin remaining in the cleft is picked up by the serotonin reuptake transporter in the presynaptic axon to be degraded and recycled. It is hypothesized that a depletion of serotonin available to act on the postsynaptic axon results in symptoms experienced in mood and anxiety disorders, like sadness, nervousness, guilt, loneliness, apathy, and avolition. Fluoxetine attaches itself to the serotonin reuptake transporter and prevents whatever serotonin remains in the cleft from being degraded. The excess serotonin is free to attach to postsynaptic receptors and exert its intended effects (Stahl, 2008).

Although fluoxetine begins acting immediately to inhibit serotonin reuptake, its antidepressant and anxiolytic effects are often not experienced for up to 6–8 weeks. This is thought to be due to gradual changes fluoxetine makes on serotonin receptor sensitivity. It is believed that the decreased amounts of serotonin found in mood and anxiety disorders cause the postsynaptic axon to upregulate the number of postsynaptic axonal receptors expressed. In other words, a decreased amount of serotonin in the synaptic cleft results in an increased amount of receptors at the ready. When fluoxetine enters the picture, the level of serotonin in the cleft rises. Postsynaptic receptors recognize the serotonin increase and send messages to the axon nucleus about the changes in the synaptic cleft. The nucleus begins downregulating the amount of serotonin receptors ready to receive serotonin. These changes are gradual and are believed to take 6–8 weeks before fully completed, reflecting the amount of time before many patients begin feeling relief from their symptoms (Stahl, 2008).

Fluoxetine's antagonism of the postsynaptic 5-HT_{2C} receptor results in some of its unique therapeutic effects. When serotonin attaches to the 5-HT_{2C} receptor, it blocks the release of neurotransmitters norepinephrine and dopamine in the brain. When fluoxetine blocks the postsynaptic 5-HT_{2C} receptor, norepinephrine and dopamine are instead released and exert their effect in the prefrontal cortex. The effect is activating, often leading patients to feel more energized, less fatigued, and to have improved concentration and attention. Fluoxetine is therefore considered for patients whose constellation of symptoms includes fatigue, apathy, and avolition as opposed to nervousness, insomnia, and agitation. Other therapeutic effects from postsynaptic 5-HT_{2C} antagonism include anorexia and antibulimia, particularly at higher doses of fluoxetine, and relief from bipolar depression when combined with olanzapine, a second-generation antipsychotic. Of note, neither olanzapine nor fluoxetine alone is approved for the treatment of bipolar depression. However, they both antagonize the 5-HT_{2C} receptor. This combined antagonism likely boosts norepinephrine and dopamine release in the cortex and may be what drives the antidepressant effect seen in the treatment of bipolar depression. The combination of fluoxetine and olanzapine is marketed as Symbyax with a fixed-dose ratio of fluoxetine to olanzapine, although both can be prescribed separately.

Fluoxetine also weakly attaches to the norepinephrine transporter and prevents norepinephrine's reuptake from the synaptic cleft. This may contribute to its clinical effects at higher doses.

Fluoxetine inhibits the cytochrome P450 CYP 2D6 and 3A4 enzymes via the parent compound and the active metabolite. The half-life of the parent compound is 2–3 days and that of the active metabolite is 2 weeks (Stahl, 2008).

Specific Compounds and Properties

N-Methyl-γ-[4-(trifluoromethyl)phenoxy] benzenepropanamine hydrochloride

Clinical Use (Including Side Effects)

FDA-Approved Clinical Uses

Fluoxetine is FDA-approved for the treatment of some mood and anxiety disorders. Mood disorders include major depressive disorder (MDD),

premenstrual dysphoric disorder (PMDD), and bipolar depression. Anxiety disorders include OCD and panic disorder, with or without agoraphobia (Lilly, 2009).

In the treatment of mood and anxiety disorders, fluoxetine is usually started between 10 and 20 mg once daily and gradually increased to 20–80 mg daily in adults (Lilly, 2009). The goal of treatment is complete remission of symptoms and prevention of future relapses, so fluoxetine is taken both throughout and in between clinical relapses (Stahl, 2009). Fluoxetine does not cure mood or anxiety disorders, and symptoms can reoccur after the medicine has been stopped.

In adults with MDD, fluoxetine is used to treat acute depressive episodes and as maintenance therapy between episodes (Lilly, 2009). After the first episode of depression, fluoxetine should be taken for 1 year (Stahl, 2009). After the second or any subsequent episodes of depression, treatment with fluoxetine may be indefinite to avoid relapse of symptoms. Fluoxetine has also been helpful in the short-term treatment of acute depressive episodes associated with bipolar I disorder when used in combination with olanzapine (Lilly, 2009). Combination therapy with set doses of fluoxetine and olanzapine is manufactured as Symbyax by Eli Lilly, although these two medications can be prescribed separate from each other. Separate dosing may allow for more flexibility on the part of the prescribing clinician. Fluoxetine should not be used as monotherapy for treatment of depression associated with bipolar I disorder. The combination of fluoxetine and olanzapine is also indicated for treatment-resistant depression, referring to depression that does not respond to two separate trials of different antidepressants of adequate doses and duration within the current episode. A once-weekly dosing of fluoxetine is marketed as Prozac Weekly for the treatment of MDD.

In adults with OCD, fluoxetine is given to treat acute episodes and as maintenance therapy between episodes. Fluoxetine is also used in the acute treatment of adults with panic disorder, with or without agoraphobia. Fluoxetine can be helpful in the treatment of bulimia nervosa, an eating disorder. Moderate to severe bulimia nervosa, which is defined as three bulimic episodes per week within 6 months, has been shown to benefit from treatment with fluoxetine. For bulimia, fluoxetine is dosed between 60 and 80 mg daily and patients may require indefinite treatment (Stahl, 2009).

Regarding children and adolescents, fluoxetine is FDA-approved for the treatment of MDD in pediatric populations aged 8–18 years and OCD in those aged 7–17 years (Lilly, 2009). In MDD, fluoxetine is given for acute depressive episodes and as maintenance therapy between episodes. The starting dose is typically between 10 mg and 20 mg/day. Due to higher plasma levels in lower weight children, the target dose may not exceed the initial dosage or much beyond. In OCD, fluoxetine is given for acute episodes and as maintenance therapy between episodes. The starting dose is typically 10 mg/day, increased to as high as 60 mg/day in adolescents. Lower weight children may only tolerate a lower target dose, such as 20 mg/day.

Clinical Uses of Fluoxetine in Autism Spectrum Disorders

Regarding pediatric populations, there is limited evidence to suggest the effectiveness of fluoxetine in children and adolescents with autism. Interestingly, antidepressants have been the most commonly prescribed psychotropic medications in treating symptoms associated with ASDs (Aman, Lam et al., 2005). Given the frequent use of SSRIs in autism, it is imperative that prescribing clinicians balance the benefit of such medications in lieu of their side effects. In 2004, the FDA released safety warnings about the increased risk of suicide-related behaviors in children and adolescents taking SSRIs, which subsequently curtailed the prescribing of SSRIs to this population (Nemeroff, Kalali et al., 2007). The decision to use fluoxetine in the treatment of established clinical indications that may co-occur with autism should therefore be made on an individual basis by the treating practitioner after careful consideration of the available data.

In the only published double-blind, placebo-controlled trial using fluoxetine to treat symptoms associated with ASDs in children and adolescents, fluoxetine was found to be

significantly better than placebo in reducing repetitive behaviors (Hollander, Phillips et al., 2005). There was no improvement on measures of speech or social interaction, and side effects were not significantly different between fluoxetine and placebo. This study used low doses of liquid fluoxetine in a population of 45 children between the ages of 5 and 16 years (mean age, 8.2 years) who met criteria for autism, Asperger's disorder, or PDD-NOS. The starting dose began at 2.5 mg/day and was increased weekly to the target dose of 0.8 mg/kg/day, although the highest dose given was 0.4 mg/kg/day. Unfortunately, the remaining research in this area has been open label or retrospective, limiting further conclusions about the effectiveness of fluoxetine in treating children and adolescents with ASDs. There is no data regarding the effect of fluoxetine on anxiety, depression, hyperactivity, inattention, irritability, or aggression in children with ASDs.

In adult populations, there is minimal data supporting the use of fluoxetine in the treatment of ASDs as evidenced by only one small, placebo-controlled pilot study. This trial focused on six adult patients, five of whom had autism and one with Asperger's disorder (Buchsbaum, Hollander et al., 2001). The patients showed significant improvement of anxiety and obsessive-compulsive symptoms while on fluoxetine. There was no significant improvement in depression. The starting dose was 10 mg daily, which was gradually titrated up to 40 mg daily in most of the patients. One should be cautious in applying these findings to the greater population of adults with autism as the small number of patients included in the study limits its interpretation. Unfortunately, there is no data regarding the effect of fluoxetine on hyperactivity, inattention, irritability, aggression, or social impairment in adults with ASDs.

It should be noted that the repetitive thoughts and behaviors observed in individuals with ASDs are qualitatively different from the symptoms of OCD (McDougle, Fleischmann et al., 1995). For example, individuals with ASDs are more likely to experience compulsions of ordering, hoarding, telling or asking, and touching, tapping, or rubbing. They are less likely to experience obsessions of contamination, sexual, religious, symmetry, or somatic content, unlike individuals with OCD. In the study of fluoxetine cited above, participants were rated on symptoms of OCD but no distinction was made regarding which repetitive behaviors improved.

Side Effects

Fluoxetine's side effect profile results from the stimulation of serotonin receptor subtypes as well as lesser actions on other neurotransmitters and enzymes. Stimulation of serotonin receptor subtypes (5-HT_{2A}, 5-HT_{2C}, 5-HT_3, and 5-HT_4) in various parts of the brain likely causes many of SSRIs' observed side effects (Stahl, 2008). A small amount of increased synaptic serotonin shortly after initiating therapy is often enough to mediate side effects, even if the clinical benefit is not yet apparent to the patient. Therefore, it is possible that side effects may be experienced earlier than symptom relief when first starting treatment with fluoxetine (Stahl, 2008). Side effects experienced may also be dose dependent (i.e., they increase as the dose increases) or time dependent (i.e., they start right after taking the medication but diminish with time) (Stahl, 2009).

Patients who are treated for MDD are at increased risk for experiencing suicidal thinking and behavior. Antidepressants as a class have been shown to increase the risk of suicidal thinking and behavior in children, adolescents, and young adults (ages 18–24) with MDD and other psychiatric disorders. Such risk should be carefully considered when prescribing fluoxetine in the pediatric and young adult population. According to Stahl (2009), gastrointestinal side effects are common and can include decreased appetite, nausea, diarrhea, constipation, or dry mouth. Sexual dysfunction is a common side effect in both men and women and is due to the effect of increased serotonin in both the brain and the region of the spinal cord regulating sexual response. In men, this includes delayed ejaculation, erectile dysfunction, and decreased sexual desire. In women, this includes decreased sexual desire and anorgasmia. Central nervous system side effects include insomnia or sedation, agitation, tremors, headache, and dizziness or lightheadedness. Patients may experience vasodilatation, dry

mouth, diaphoresis, or abnormal vision. Increased serotonin can lead to diminished dopamine release, which may lead to emotional flattening, apathy, and cognitive slowing in some patients. Anorexia and weight loss have been reported when using fluoxetine, so it should be used with caution in underweight patients or those who are prone to eating disorders (Lilly 2009).

Of note, SSRIs have been found to yield side effects in people with ASDs, particularly agitation, hyperactivity, aggression, and insomnia (Posey, Erickson et al., 2006). Extrapyramidal symptoms (Sokolski, Chicz-Demet et al., 2004) and hypomania (Damore, Stine et al., 1998) have also been observed in case reports. These results are limited by a lack of placebo-controlled studies, although there is some evidence to suggest that children with ASDs may be more likely to develop such side effects relative to adults with ASDs (Posey, Erickson et al., 2006).

See Also

- ▶ Antidepressant Medications
- ▶ Depressive Disorder
- ▶ Dopamine
- ▶ Norepinephrine
- ▶ Obsessive-Compulsive Disorder (OCD)
- ▶ Repetitive Behavior
- ▶ Serotonin
- ▶ Serotonin Reuptake Inhibitors (SRIs)
- ▶ Stereotypic Behavior

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Fluvoxamine

Kate S. Perri[1], Marcel Moran[2], Kimberly Stigler[3] and Christopher J. McDougle[4]

[1]Christian Sarkine Autism Treatment Center, Riley Hospital for Children, Indianapolis, IN, USA

[2]Indiana University School of Medicine, Indianapolis, IN, USA

[3]Christian Sarkine Autism Treatment Center, James Whitcomb Riley Hospital for Children, Indianapolis, IN, USA

[4]Lurie Center for Autism/Harvard Medical School, Lexington, MA, USA

Synonyms

Fluvoxamine maleate; Luvox; Luvox CR

Indications

Fluvoxamine is FDA-approved for the treatment of obsessive-compulsive disorder (OCD).

Mechanisms of Action

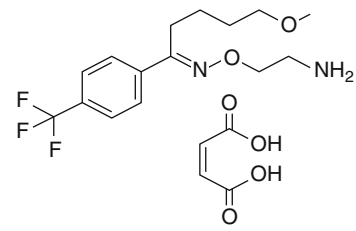
Fluvoxamine is a selective serotonin (5-HT) reuptake inhibitor (SSRI). Its efficacy in the treatment of OCD is thought to result from inhibition of the serotonin reuptake process and increased serotonergic neurotransmission (Stahl, 2008).

Fluvoxamine primarily inhibits the oxidative drug-metabolizing enzyme CYP1A2 and also significantly inhibits CYP3A4, CYP2C9, and CYP2C19. It is a weak inhibitor of CYP2D6 (Irons, 2005; Kuzma & Black, 2008). Additionally, it is a sigma-1 receptor agonist (Ordacgi, Mendlowicz, & Fontenelle, 2009). Sigma-1 receptors regulate the release of glutamate, dopamine, serotonin, norepinephrine, and acetylcholine. Fluvoxamine's high affinity for these receptors may account for some of its effects on depression and anxiety.

Specific Compounds and Properties

Fluvoxamine maleate has the International Union of Pure and Applied Chemistry (IUPAC) name (E)-5-methoxy-1-[4-(trifluoromethyl)phenyl] pentan-1-one *O*-2-aminoethyl oxime maleate and the empirical formula $C_{15}H_{21}O_2N_2F_3 \bullet C_4H_4O_4$ (Fig. 1).

Fluvoxamine is metabolized by the liver into at least 11 products, all of which are pharmacologically inactive. It is excreted primarily as metabolites, with less than 4% of the original compound remaining. The precise CYP isoenzymes involved in its metabolism are not known. There is evidence that fluvoxamine has nonlinear steady-state pharmacokinetics so that plasma concentrations are disproportionally higher when the dosage is increased. It has a mean plasma half-life of 12–22 h, which is increased by 30–50% at steady state. It has been demonstrated to be eliminated from the brain at a slower rate than from the plasma (2.4:1 ratio; Ordacgi, 2009).



Fluvoxamine, Fig. 1 Chemical structure of fluvoxamine maleate

In vitro studies have found it to be a stronger inhibitor of serotonin reuptake than fluoxetine, but weaker than paroxetine, sertraline, citalopram, and escitalopram. It is also a weak inhibitor of norepinephrine and dopamine reuptake when compared to other serotonin reuptake inhibitors (Ordacgi, 2009).

Clinical Use (Including Side Effects)

Luvox is the brand name of fluvoxamine maleate in the United States. It is available in both immediate-release (IR) and extended-release (controlled-release; CR) tablets and is FDA-approved for the treatment of obsessions and compulsions in individuals with OCD. The IR form is approved for use in patients 8 years and older, while the lowest dosage of the CR form may not be appropriate for pediatric patients (U.S. Food and Drug Administration, 2011a, 2011b). Its efficacy in the treatment of OCD has been demonstrated in numerous randomized, double-blind, controlled studies. Fluvoxamine has repeatedly been shown to be more efficacious than placebo when measured by the Yale-Brown Obsessive Compulsive Scale (Y-BOCS), National Institute of Mental Health-Obsessive Compulsive (NIMH-OC) Scale, and Clinical Global Impression (CGI) scales (Ordacgi et al., 2009). Studies have also compared fluvoxamine to the tricyclic antidepressants clomipramine and

desipramine. Fluvoxamine was found to be equally efficacious as clomipramine and more efficacious than desipramine in the treatment of OCD (Irons, 2005). It has additionally been shown to be efficacious in the treatment of OCD in children and adolescents aged 8–17 years (Irons).

Luvox CR was initially approved for the treatment of social anxiety disorder, but this indication was removed from its label in 2011. However, a number of double-blind, placebo-controlled studies have shown promise for the treatment of social anxiety disorder with fluvoxamine. The results of several studies, two involving over 250 patients each, have demonstrated significantly greater improvements with fluvoxamine than with placebo, using measures such as the Liebowitz Social Anxiety Scale, CGI scales, and the Hamilton Anxiety Scale. Furthermore, fluvoxamine has been demonstrated to be significantly more effective than placebo in a study of 128 children and adolescents aged 6–17 years with social anxiety disorder, separation anxiety disorder, or generalized anxiety disorder (GAD) (Irons, 2005).

Fluvoxamine may also be an effective treatment for some of the behaviors associated with autism. In a randomized, double-blind study with 30 adults, 53% of the patients treated with fluvoxamine were classified as responders. There was a significant reduction in repetitive thoughts and behaviors, maladaptive behavior, and repetitive language usage in the fluvoxamine group compared to the placebo group (McDougle et al., 1996).

There have been mixed results in studies reporting the use of fluvoxamine for the treatment of autism in children. In one double-blind crossover study of fluvoxamine and placebo, 10 out of 18 children were classified as responders, while in another, improvements were shown in behaviors such as eye contact and language use (Fukuda, Sugie, Ito, & Sugie, 2001; Sugie et al., 2005). However, an open-label trial and a double-blind, parallel groups comparison with placebo, which both administered fluvoxamine to 18 subjects, classified only 3 and 1 subjects, respectively, as responders (Martin, Koenig, Anderson, & Scahill, 2003; McDougle, Kresch, & Posey, 2000).

Numerous studies of adults have shown benefits for the treatment of depression, panic disorder, GAD, and posttraumatic stress disorder (PTSD), for which fluvoxamine is also commonly prescribed (Irons, 2005; Stahl, 2008). Additionally, several randomized, double-blind studies have demonstrated fluvoxamine's efficacy in the reduction of binge eating, relapse of bulimia nervosa, compulsive buying, and pathological gambling (Irons).

The recommended starting dose of fluvoxamine is 25 mg in pediatric patients and 50 mg in adults, taken before bed. The dose should be increased every 4–7 days in 25-mg increments in children and 50-mg increments in adults until the maximum benefit is reached. The dose should not exceed 200 mg/day in children up to age 11 years and 300 mg/day in adolescents and adults. If the dose is more than 50 mg for the pediatric population or 100 mg for adults, it is recommended for it to be divided into two doses. With uneven doses, the larger one should be taken before bed (U.S. Food and Drug Administration, 2011b).

Side Effects

The most common adverse reactions in trials with adults with OCD and depression include nausea, somnolence, insomnia, asthenia, nervousness, dyspepsia, abnormal ejaculation, sweating, anorexia, tremor, and vomiting. Less frequent side effects include anorgasmia, decreased libido, dry mouth, rhinitis, taste perversion, and urinary frequency in individuals with OCD and agitation, depression, dysmenorrhea, flatulence, hyperkinesia, and rash in pediatric patients with OCD (U.S. Food and Drug Administration, 2011b).

As with other antidepressants, fluvoxamine may result in the worsening of depressive symptoms and an increased suicide risk. It should not be used in combination with monoamine oxidase inhibitors or within 14 days of ending treatment with one. Additionally, tizanidine, thioridazine, alosetron, and pimozide should not be coadministered with fluvoxamine. Serotonin syndrome and neuroleptic malignant syndrome have also been reported with

SSRIs, including fluvoxamine. There is evidence that the CR form has a lower maximum plasma concentration, reducing the risk of adverse effects (Ordacgi, 2009).

See Also

- ▶ Antidepressants
- ▶ Selective Serotonin Reuptake Inhibitors (SSRIs)

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Fluvoxamine Maleate

- ▶ Fluvoxamine

fMRI

- ▶ Event-Related Functional Magnetic Resonance Imaging (MRI)

Folk Psychology

- ▶ Intentional Stance

Food Intolerance

Susan Hyman
Division of Neurodevelopmental and Behavioral Pediatrics, University of Rochester Golisano Children's Hospital, Rochester, NY, USA

Definition

Food intolerance refers to the occurrence of physical symptoms relative to ingestion of foods that are not the result of conventional IgE-mediated food allergy. IgE is the subclass of immunoglobulins that cause an individual to make antibodies to food or other compounds that result in allergic symptoms. Food intolerance may be more common than true food allergy. The physical symptoms of food intolerance may impact the skin and the gastrointestinal and respiratory tracts. Central

nervous system symptoms like migraines may occur. Food intolerance may manifest in a delayed fashion, unlike IgE-mediated allergy. Food intolerance is attributed to the following mechanisms:

1. Absence of an enzyme necessary for metabolism of an ingested food: An example of this is lactose intolerance. Approximately 10% of Americans are intolerant of the milk sugar, lactose, because of an absence of the enzyme necessary for digestion with resultant symptoms of diarrhea, bloating, and intestinal gas.
2. Symptomatic response to a food or food ingredient based on genetic susceptibility: An example of this is intolerance of natural salicylates. Salicylic acid is the active ingredient in aspirin. Approximately 2.5% of Europeans are intolerant of natural salicylates found in various fruits and vegetables. Other chemicals in food that are associated with intolerance include food additives like monosodium glutamate, nitrites, and artificial food colors.
3. Symptomatic response to a chemical compound in food based on a co-occurring condition: An example of this is development of physical symptoms relative to histamine ingestion when the enzymatic degradation of this vasoactive amine is impaired by an imbalance of intestinal flora or a medication prescribed for another condition.
4. Non-IgE-mediated immune response: The utility of measuring IgG to foods is controversial and at present does not guide therapy.

Food intolerance needs to be clinically distinguished from IgE-mediated food allergy. This is done by history and physical examination, allergy testing, elimination diets, and double-blind food challenges. Unlike food allergy, many people with food intolerance do not have symptoms with small and infrequent exposures. Dietary avoidance is the treatment.

See Also

- ▶ Food Intolerance
- ▶ Gastrointestinal Disorders and Autism

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Formal Complaint

Margaret St. John
Quinnipiac University School of Law, Hamden, CT, USA

Definition

Formal Complaint

In General

A formal complaint is an initial filing that starts a civil lawsuit and usually states the basis for a court's jurisdiction, the basis for a plaintiff's claim, and a demand for relief.

Individuals with Disabilities Education Act

Individuals (typically parents) may submit a formal complaint to a child's school district and/or state education agency alleging violation of the Individuals with Disabilities Education Act.

Any party has an opportunity to present a formal complaint regarding the identification, evaluation or educational placement (IEP) of a child, or the provision of a free appropriate public education (FAPE) to such child. The complaint must be filed within 2 years of the date when the parent or public agency knew or should have known about the alleged action that forms the basis of the complaint.

When filing a complaint alleging that a local educational agency has violated a requirement of the Individuals with Disabilities Education Act, the complaint must be written, signed, and must cite the facts upon which the allegation is made and the specific statutory requirement that was violated. The state educational agency must resolve the issues of the complaint within 60 days after it is filed.

Americans with Disabilities Act

If an individual believes that he or any other person has suffered discrimination, he may file a complaint with the Department of Justice. Such a complaint should include contact information, the name of the business, organization, institution, or person that perpetrated the alleged discrimination and a description of the discrimination.

See Also

- ▶ Eligibility (for Services Under IDEA/ADA, etc.)
- ▶ Individuals with Disabilities Education Act (IDEA)

References and Readings

Americans with Disabilities Act (ADA), U.S.C. § 12101

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FOT

- ▶ Finger-Tapping Test

Fragile X Syndrome

- ▶ Fragile X Syndrome (FXS)

Fragile X Syndrome (FXS)

David Hessl[1] and Andrea Schneider[2]
[1]Department of Psychaitry and Behavioral Sciences, University of California, Davis Medical Center, M.I.N.D. Institute, Sacramento, CA, USA
[2]Department of Pediatrics, University of California, Davis Medical Center, M.I.N.D. Institute, Sacramento, CA, USA

Synonyms

Escalante's syndrome; Fragile X syndrome; Gender-linked mental retardation with macroorchidism; Marker X syndrome; Martin-Bell syndrome

Short Description or Definition

Fragile X-associated disorders like fragile X syndrome (FXS), fragile X-associated tremor/ataxia syndrome (FXTAS), and fragile X-related premature ovarian insufficiency (FXPOI) are a group of different developmental and neurodegenerative conditions linked to a mutation on the X chromosome (Hagerman & Hagerman, 2002).

Fragile X syndrome (FXS) is the most common inherited cause of intellectual disability and the most common known single-gene mutation leading to autism.

Categorization

The first report of a family pedigree with intellectual disability and characteristic physical features was published by Martin and Bell in 1943. The inheritance pattern was clearly X-linked – from mothers to sons and fathers to daughters (Martin & Bell, 1943). A study by Herbert Lubs found a cytogenetic irregularity he described as "marker X chromosome" linked to intellectual disability in three generations (Lubs, 1969). Since the discovery of the *FMR1* gene in 1991, the diagnosis of FXS can be made with *FMR1* DNA testing.

In general, male individuals with FXS are more severely affected than females who possess a second X chromosome without the mutation. There is a wide spectrum of symptoms that are associated with FXS: soft skin, elongated face, large or protruding ears, flat feet, larger testes (macroorchidism), high-arched palate, hyperextensible joints, epilepsy, and low muscle tone. In addition to the intellectual disability, individuals with FXS show language delays, behavioral problems, autism spectrum symptoms (poor eye contact, hand flapping, odd speech), hyperactivity, high levels of anxiety, delayed motor development, and/or sensory problems.

Most carriers of a fragile X premutation have normal cognitive abilities, although there is evidence that some carriers are affected by neurodevelopmental disorders like autism spectrum disorders (ASD) or attention deficit hyperactivity disorder (ADHD), and working memory problems. Carriers with a CGG repeat size of >150 may have features of the full mutation FXS, especially when FMRP is reduced.

Epidemiology

The fragile X full mutation is found in 1 out of 2,500 individuals in the general population (Hagerman, 2008). Premutation alleles are more common than full mutation alleles, with approximately 1 in 130–250 females and 1 per 250–813 males in the general population. Approximately 16–20% of female premutation carriers develop primary ovarian insufficiency (POI) before age 40. The prevalence of FXTAS was found to be 17% in male premutation carriers in their 50s, 38% in their 60s, 47% in their 70s, and 75% in their 80s among families with known FXS. Only 8% female carriers are affected by FXTAS later in life.

Natural History, Prognostic Factors, and Outcomes

Usually, the diagnosis of FXS is not considered until an individual demonstrates developmental delays. In male infants with FXS, those delays can be seen as early as 9–12 months of age with motor problems, hyperactivity and irritability, low muscle tone, and failure to reach subsequent developmental milestones, like language or walking. Female individuals with FXS are generally higher functioning, and a diagnosis can be more challenging and often determined later in development. From a medical perspective, individuals with FXS have a high rate of recurrent ear infections and vision problems (strabismus, ptosis) during the first 3 years of life. Common behavioral problems in preschoolers with FXS are temper tantrums, hyperactivity, aggression, attention deficits, and mood instability. There is significant overlap with ASD symptoms, especially in males, such as hand flapping, hand biting, perseverations, social anxiety, repetitive speech, and poor eye contact. Autistic disorder occurs in approximately one-third of males with FXS. Individuals with lower cognitive abilities tend to have a higher rate of ASD symptoms.

At school age, the intellectual disability is prominent in over 90% males and 50% females with FXS, with a slow cognitive growth rate. The individuals have difficulties in executive control, visual-motor coordination, processing sequences, short-term memory problems, arithmetic, attention, inhibition, motor development, and coordination.

During adolescence and early adulthood, the rate of disruptive behavior decreases, but social interactions remain difficult because of the high social anxiety rate. Aggression and irritability are common problem behaviors in male individuals with FXS during adolescence. Older adults with FXS show increased rates of heart problems, musculoskeletal disorders, epilepsy, and visual impairments.

Although many carriers with the premutation are unaffected, several studies demonstrate significant clinical symptoms of hyperactivity, social deficits, autism spectrum disorders, and executive function problems during childhood. Boys with the premutation have been reported as having raised rates of delayed development of adaptive behavior, autism spectrum and attention deficit disorders, and speech and language

difficulties, mostly related to social language use, speech intelligibility, and expressive language.

Several studies show a higher rate of mood and anxiety disorders, especially social phobias and major depression in female premutation carriers.

Clinical Expression and Pathophysiology

From a neurobiological standpoint, the lack of fragile X mental retardation protein (FMRP), the gene product of *FMR1*, is responsible for the majority of symptoms in FXS. FMRP is essential for cellular processes to regulate synaptic plasticity and structure. It inhibits the production of proteins. In FXS, there is an abundance in the production of several proteins, especially those that are normally regulated by a specific excitatory pathway (metabotrobic glutamate receptors, mGluR1 and mGluR5). Animal models provide invaluable insight into the molecular functions of FMRP. The *FMR1*-deficient fruit fly (Drosophila) and the *FMR1* knockout mouse model show comparable behavior to human FXS: defects in circadian rhythms, synaptic branching, cognition, hyperactivity, audiogenic seizures, macroorchidism, and dendritic spine abnormalities, leading to weak synaptic connections. Many of these abnormalities are reversed by mGluR antagonists in mouse and fruit fly models, and several trials with mGluR blockers and other targeted treatments in humans are now underway (Bear, Huber, & Warren, 2004).

Individuals with FXS show a wide variability of behavioral and cognitive symptoms from mild emotional or learning problems to severe intellectual disabilities with autism. Other symptoms include hyperarousal and hyperactivity, especially impulsivity and short attention span, avoiding eye contact, hand flapping and hand biting, sensory defensiveness, and poor adaption to changes in routine, shyness and social anxiety, stereotypies and tics, aggressive outbursts, enuresis, and encopresis. A common finding in FXS is a comorbidity with psychiatric symptoms that is often overlooked because of diagnostic overshadowing. Increased rates of bipolar affective disorders, psychosis, and mood instability with aggression and depression have been reported.

The social impairments in individuals with FXS include social anxiety, gaze aversion with other visual, auditory, tactile, olfactory, and gustatory sensitivities, and self-injury. The overlap between FXS and ASD is complex. Approximately 2–6% of people with autism have FXS, and approximately 30% of boys with FXS have autism. Interestingly, the majority of FXS individuals with ASD show the paradoxical behavior of being friendly and sociable (even though shy and socially anxious) in the presence of multiple autistic-like communicatory and ritualistic qualitative impairments.

Another common symptom for FXS is executive function deficits and attention and hyperactivity problems that can be aggravated by anxiety. Sleep disorders are also frequent.

The connection between neuropsychological impairments and molecular findings in both full mutation and premutation carriers have been described as "continuum," influenced by environmental factors, mediating memory problems, impaired inhibitory control and cognitive switching abilities, impaired sequential information processing, and impaired arousal modulation. For example, the cognitive and emotional difficulties in female premutation carriers may be influenced by challenges associated with raising one or more children with the full mutation FXS.

Evaluation and Differential Diagnosis

So far, there are no practice guidelines available for the initial diagnosis of FXS. The American Academy of Pediatrics recommends fragile X testing in males and females with intellectual disability, autism spectrum disorders, or hyperactivity combined with behavioral problems of unknown etiology. Especially in males, a phenotypic checklist can be helpful to decide if molecular testing would be appropriate.

Today, a diagnosis of the full mutation allele of the *FMR1* gene is based on molecular assessment of the CGG repeat size for individuals with physical, cognitive, or behavioral features of a probable FXS. However, about 1% of *FMR1* mutations are not repeat expansions, but point mutations or deletions. The molecular assessment is essential because children with FXS may have very few if any evident physical features and may also be high functioning intellectually without obvious cognitive deficits, particularly among females with a full mutation allele. Molecular assessment is carried out with both a polymerase chain reaction (PCR) and a Southern blot. These methods provide the CGG repeat number, the X activation ratio, and the degree of methylation present in the full mutation.

If an individual has been identified with a *FMR1* mutation, genetic counseling and cascade testing of additional potentially affected family members are typically recommended.

Besides the molecular testing, individuals with FXS require a comprehensive medical, developmental, and psychological/psychiatric assessment. In addition to the standard medical care for children, specific recommendations for FXS include further health supervision in the following areas. In newborns, health-care providers should look out for orthopedic abnormalities and feeding difficulties. In infancy, hypotonia could result in mild motor delay, furthermore irritability, tactile hypersensibility, and gastroesophageal reflux. In early childhood, an ophthalmologic evaluation is recommended, also the assessment of orthopedic problems related to connective tissue dysplasia. In preschool years, structural changes of the face may start to develop, including an elongated face, high forehead, arched palate, and prominent ears. Furthermore, a history of seizures, and recurrent otitis media should be assessed. In late childhood, macroorchidism in boys with the full mutation can develop at around 9 years of age, whereas girls with the full mutation can be affected by precocious puberty. Because of the connective tissue dysplasia, the FXS individuals should be monitored for scoliosis. In adolescence and adulthood, seizures should be assessed. Because of the high rate of mitral valve prolapse, a cardiology evaluation is essential.

On the cognitive-behavioral level of development, the following categories are part of the assessment: cognitive functioning (intelligence, learning), physical-medical background, EEG, social and emotional abilities, speech and language, and sensory difficulties. In early childhood, the expressive and receptive language development should be monitored. Behaviorally, tantrums and hyperactivity are common, especially at transitioning and sensory overstimulation. First symptoms of anxiety, depression, aggressive outbursts, and obsessive-compulsive behaviors can manifest during that time. Before 3 years of age, symptoms of autism can develop (hand flapping, self-injury, poor eye contact) and require further assessment. In late childhood, intellectual disability, hyperactivity, and anxiety, especially social anxiety, are very common in both boys and girls with the full mutation FXS.

Treatment

So far, there is no cure for FXS. A multidisciplinary approach has been identified to be most helpful for the improvement of educational and behavioral problems in FXS.

Clinical reports show that individuals with FXS benefit from medication therapy, speech and language intervention, behavioral approaches, and special education services. Especially in the early years, the interventions appear to be most efficient for FXS children, including physical, speech and sensory training, and the promotion of a routine for the child, which helps to reduce anxiety. Supporting parents and siblings with family education and genetic counseling is important to facilitate the acceptance and understanding of individuals with FXS. In older individuals, a cognitive-behavioral approach can be used for treatment of anxiety and depression. Any treatment should be modified to

suit the individual's level of functioning, using nonverbal materials, visual aids such as drawings, symbols, videos, photographs, and dolls, and role play.

A variety of currently available medications (e.g., serotonin agents, stimulants, antipsychotics) can be given to treat behavioral problems, particularly mood instability, hyperactivity, and anxiety in FXS, but none of these medications are thought to change the neurobiological base defect of the mutation. However, several novel treatment studies, such as those targeting the glutamate and GABA systems, as well as minocycline, and other agents based on extensive animal studies, are currently underway. These treatments have the potential to reverse the neurobiological abnormalities and perhaps significantly improve cognitive, emotional, and behavioral problems characteristic of FXS. Individuals with *FMR1*-related disorders are affected with a variety of problems throughout their whole life span. Treatment approaches should maximize the success with a coordination of community resources, medical, behavioral, developmental, and emotional support.

See Also

- ▶ Autism
- ▶ Genetics
- ▶ Intellectual Disability

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National Fragile X Foundation. (www.fragileX.org)

Fraternal Twins

- ▶ Dizygotic (DZ) Twins

Free Appropriate Public Education

Juli Katon
Department of Special Education, University of Maryland, College Park, MD, USA

Definition

Free appropriate public education (FAPE) is a term that is used to describe the federal mandate that all school-aged special education students be provided with special education and related services at public expense. The word "free" means that the individual is entitled to daily schooling at no extra cost to the family. The word "appropriate" means that the student's education must be individualized to the unique learner's needs as decided upon during the development of the student's individualized education program (IEP).

The word "public" refers to who will be paying for, supervising, and directing the learner's education. The word "education" means that children who qualify for special education are entitled to schooling like their same-aged, typical peers. The services provided under FAPE must meet the standards of the local state education agency in addition to being provided at public expense to school-aged children from preschool to high school in accordance with the IEP.

For children with a diagnosis of autism, this means that the majority of their weekdays from the time they are 3 years old will be spent in a public school classroom. An individualized family service plan or an IEP is created for a student when they enter the school system. The IFSP or IEP team consists of several members whose input is critical to the development of an IFSP/IEP: parents, school personnel, and anyone else that the family feels should be a part of the team. Together, the team will decide on the least restrictive environment and the most appropriate setting for the individual with autism. For individual students, this means different settings. An appropriate education for one student may be to be included with same-aged peers in his or her home school. An appropriate education for another student may be a self-contained classroom for children with autism within a comprehensive school. When the IEP team has come to an agreement on what is appropriate for a student, the student's FAPE can begin.

See Also

▶ Individuals with Disabilities Education Act (IDEA)

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Free Recall

Hillary Hurst
Department of Psychology, University of Massachusett Boston, Boston, MA, USA

Definition

Free recall is a paradigm used in memory testing. It involves the presentation of information, such as a list of words or images, to the examinee, followed by a period of either immediate or delayed recall, during which the examinee is asked to produce as much of the presented information as possible. This recall can be oral or written. The primacy effect and recency effect, presented in a classic study by Murdock (1962), both affect free recall: "primacy effect" is the ability to recall information presented first more easily, and "recency effect" is the ability to recall information presented last more easily. Free recall of verbal stimuli tends to be an area of relative weakness for individuals with autism spectrum disorders because they may be less able than individuals without ASD to make subjective connections across words that, in turn, assist with their recall (Gaigg, Gardiner, & Bowler, 2008). Children with ASD have been found to perform more compared to children without ASD when recall is cued or assisted, not free (Tsatsanis, 2005).

See Also

▶ Memory
▶ Wechsler Memory Scale (All Versions)

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Friendships

Nirit Bauminger-Zviely
School of Education, Bar-Illan University, Ramat-Gan, Israel

Definition

Friendship is a type of social relationship appearing throughout the life span, from early childhood to old age. It is conceptualized under the *social relationships approach* (Hinde, 1976), according to which relationships are developed through continuous dyadic interactions over long periods of time with a specific partner (a minimum of 6 months to denote friendship; Howes, 1996), to extract a relationship model that goes beyond the influence of each member's characteristics (Dunn, 1993). Through friendship formation, children are "meshed" with each other to create a whole that is greater than the sum of its parts, and they share the properties and histories of their mutual interaction (Dunn, 1993). The essence of friendship as a relationship is a mutual liking, whereby children reciprocate an affectionate bond of emotional closeness. Having friends is cardinal to children's well-being and protects them from depression and loneliness, since friendship provides the child with a sense of belonging and self-worth (e.g., Asher, Parker, & Walker, 1996; Vitaro, Boivin & Bukowski, 2009).

The reciprocal-dyadic and stable nature of friendship is a challenge of child development and a necessary component in the formation of fundamental pro-socio-emotional capabilities (e.g., mutual caring, companionship, empathy, mutual regulation) as well as socio-cognitive capacities (e.g., awareness and responsiveness to another's emotions, desires, intentions, and thoughts; conflict resolution; complex information processing processes; Asher et al., 1996). Affective closeness, which is a marker of friendship, underscores the essence of friendship as an affective bond reflecting the strength of the child's attachment to the friend. In addition to mutual liking, closeness includes the sense of "specialness" to one another, manifested in verbal and nonverbal expressions of affect and caring (Bukowski, Boivin, & Hoza, 1994; Howes, 1996). Intimacy (i.e., making one's innermost thoughts and feelings known to a significant other) is another important dimension of friendship (Cassidy, 2001). Intimacy includes the belief that friends can be relied upon to provide support and help in times of need. Intimacy is also related to the sense of stability and reciprocity in friendship and to the belief that the friendship is strong enough to overcome negative events such quarrels (e.g., Bukowski, Boivin & Hoza, 1994). Markers of intimacy include sharing capabilities and pro-social resources such as providing help (e.g., Bukowski et al., 1994). Friendship should also serve the need for enjoyable companionship. Companionship is usually perceived as a child's ability to spend time with another child and to have fun (e.g., "playing together," "hanging out," "doing things together"; Howes, 1996). Markers of companionship include children's cooperative skills during shared work or play, as well as their social conversational skills and positive affect (e.g., shared fun) (Asher et al., 1996).

In sum, friendship is characterized by stable, frequent, and interconnected affective interactions, manifested by certain classes of behavioral markers (e.g., sharing, play, and conversational skills) that facilitate the functions of companionship, intimacy, and closeness. A satisfying friendship is an interpersonal achievement that both develops and builds upon a fundamental capacity for affective relationships and social cognition.

Historical Background

In his first description of the syndrome, Kanner (1943) historically conceptualized autism as a disorder of affective contact, "these children

have come into the world with an innate inability to form the usual, biologically provided affective contact with people" (p. 250). Kanner's description of Elaine (a 7-year-old girl with ASD) supports this conceptualization:

> She soon learned the names of all the children (in her school), knew the color of their eyes, the bed in which each slept, and many other details about them, but never entered into any relationship with them. . ..She has no relation to children, has never talked to them, to be friendly with them, or to play with them. She moves among them like a strange being, as one moves between the pieces of furniture of a room. (Kanner, 1943, p 240–241)

To this day, abnormalities in interpersonal relations are considered a defining characteristic of ASD (DSM-IV-TR, 2000). Friendship is a direct reflection of the child's capacity for interpersonal relationships, and for this reason, both the formation and the understanding of friendship in children with ASD are considered a major theoretical challenge. Contemporary perceptions of the disorder identify difficulties in friendship formation based on these children's inability to experience relationship-based emotions and on their deficit in intersubjective sharing. The results of such impairments are more impersonal and superficial rather than interpersonal friendship relationships; thus, intimacy and affective closeness, according to this view, are unattainable for ASD children (e.g., Hobson, 2005). Another view is provided by theorists that highlight ASD children's difficulty in understanding that other people have different thoughts, desires, and feelings than their own (i.e., the theory of mind hypothesis). Theory of mind also predicts crucial difficulties in reciprocity and empathic pro-social behaviors (e.g., comforting, caring, complimenting, listening), which are key defining characteristics of friendship (Tager-Flusberg, 2001). These two views, affective and social-cognitive, have led to a general consensus that friendship constitutes a major area of difficulty for children with ASD. In light of these theoretical assumptions, it is not surprising that friendship in ASD has recently been receiving more attention, but it is still an overlooked area of empirical investigation, despite its significance to the understanding of the social deficit in ASD.

Current Knowledge

Friendship Formation

Studies screening for the prevalence of friendship among individuals on the ASD spectrum have presented a fairly pessimistic picture, with significant percentages found to be outside of this meaningful social experience (e.g., Howlin, Good, Hutton & Rutter, 2004; Koning & Magill-Evans, 2001; Orsmond, Krauss, & Seltzer, 2004). For example, half of 69 adults with ASD examined were said to have no friends with whom they share activities (Howlin et al., 2004). In another study, Orsmond et al. (2004) found that of 235 adults and adolescents, 29% had at least one friendship that involved some activities outside prearranged settings and one-quarter (24%) had peer relationships only in prearranged settings. Orsmond et al. also reported a significantly higher number of friends for younger participants (adolescents rather than adults) and for individuals with less impaired social interaction skills. Similarly, in Koning and Magill-Evans' (2001) study, 44% of their adolescent participants with Asperger syndrome had one close friend. Findings based on a slightly different paradigm, examining children and adolescents' social network and social involvement, presented a similar picture, with children on the ASD spectrum more often peripheral in their classroom and forming less reciprocal friendships compared with children of typical development (e.g., Chamberlain, Kasari, & Rotheram-Fuller, 2007; Kasari, Locke, Gulsrud, & Rotheram-Fuller, 2010; Locke, Ishijima, Kasari, & London, 2010; Rotheram-Fuller, Kasari, Chamberlain & Locke, 2010). The difference is more dramatic in later elementary school and during adolescence (e.g., Locke, et al., 2010; Rotheram-Fuller et al., 2010). Studies on friendship in young children with ASD are rare. Church, Alinsanski, and Amanullah (2000) found that none of their participants with ASD between the ages of 3 and 5 had a friend. However, in a recent study based on mother's and teacher's reports and verified by an observation in the child's preschool, Bauminger and Agam-Ben Artzi (2012) were able to identify 30 high-functioning preschoolers with ASD who enjoyed

at least one reciprocal friendship. Other studies focusing specifically on friendship in HFASD (high-functioning ASD) have demonstrated the presence of at least one reciprocal friendship in these children (e.g., Bauminger & Kasari, 2000; Bauminger & Shulman, 2003; Bauminger et al., 2008b; Daniel & Billingsley, 2010). Based on these different findings, we may conclude that friendship, though not frequent, is an attainable experience at least for certain subgroups of ASD children, probably for those with higher cognitive and language skills. The two periods with somewhat higher percentages of friendship are middle childhood and early adolescence, with adults showing less frequent experiences of friendship and with as-yet unclear numbers regarding young children with ASD.

Friendship characteristics. Variables of friendship such as the friend's attributes (age, gender, type of disability), the duration of the friendship, and the frequency of play dates have rarely been explored in children with ASD. Based on the few findings presented in the literature (e.g., Bauminger & Agam-Ben Artzi., 2012; Bauminger & Shulman;, 2003; Bauminger et al., 2008b; Howlin, et al., 2004; Kasari, Locke, Gulsrud & Rotheram-Fuller, 2011; Orsmond, Krauss & Seltzer, 2004; Locke, Ishijima, Kasari & London, 2010), we may conclude with caution that both mixed and non-mixed friendships (i.e., friendship with a child of typical development and friendship with a child with a disability, most likely ASD, respectively) have been found in children with ASD, although the percentages of children who form each type of friendship are not yet clear. The majority of friendships consist of same-age and same-gender couples. Surprisingly, the duration of the friendship was found to be relatively long, ranging from about 1 to 4 years, but the frequency of play dates outside the school was reported to be about once a week, which is lower than for typically developing children. As mentioned, these somewhat optimistic findings should be further examined, especially with regard to the quality of the relationship. In typical development, friendship functions as an important source of emotional support and as a marker for social adjustment, but the consequences of friendship for children's psychosocial development is highly dependent on its quality (e.g., Vitaro, Boivin, & Bukowski, 2009).

Friendship quality and associated behaviors. The quality of the friendship relationship is probably the most debated topic in ASD. Even if researchers agree that some children with ASD do have friends, they will still question the quality of this relationship. More specifically, difficulties in experiencing intimacy and closeness are expected in these children. One source of information on the quality of friendship is children and adolescents' self-reports. Several studies have used Bukowski et al.'s (1994) Friendship Qualities Scale (FQS), which assesses children's reports about their relationship with a close friend in the following dimensions: companionship, intimacy-security, closeness, help, and conflict (e.g., Bauminger & Kasari, 2000; Bauminger et al., 2004; Bauminger et al., 2008b; Chamberlain, et al., 2007; Kasari, et al., 2011; Lock et al., 2010). In these studies, the friendship quality in ASD children was found to be poorer compared with friendship in children of typical development, especially for intimacy, help, and companionship (e.g., Bauminger & Kasari, 2000; Bauminger et al., 2004; 2008b; Chamberlain et al., 2007). Differences on the dimension of closeness between the two groups were reported by Bauminger, et al. (2008b) (preadolescence, age range 8.6–12.6) and Kasari et al. (2011) (middle childhood, age range 6–11). Interestingly, the FQS did not show friendships of children with ASD to be more conflictual than those of typically developing children.

In another study, this time examining adults, Baron-Cohen and Wheelwright (2003) provided support for lower friendship quality, revealing less close, empathic, and supportive friendships, in adults with ASD compared with typical controls. Jobe and Williams-White (2007) obtained similar results for adults with severer autism symptoms. Altogether, self-reported friendship quality of HFASD children and adults portrayed difficulties in the formation of quality friendship with peers. This finding has clinical significance: it is interesting that individual with such limited

interpersonal awareness and knowledge (e.g., Carrington, Templeton, & Papinczak, 2003; Hobson, 2005) can reveal a realistic picture of the quality of their friendship, implying that they are aware of their difficulties in interpersonal engagement. In Daniel and Billingsley's (2010) study, 10- to14-year-old boys described the establishment of friendship as the most difficult aspect in their social life. Higher ratings of loneliness were found in children with HFASD compared with typically developing children (e.g., Bauminger & Kasari, 2000; Locke et al., 2010), a finding which may also support the finding of children's awareness of their social difficulties in forming relationships. Such awareness has significant clinical implications, especially in light of accumulating evidence showing high rates of affective disorders (65%; Attwood, 2004) such as anxiety, depression, and social isolation, in high-functioning ASD adolescents and adults compared with typical controls (e.g., Attwood, 2004). Lack of friends may be one of the reasons for the increase in affective disorders during adolescence and adulthood.

Further support for the notion of lower friendship quality in children with ASD has been provided by observations on children's social interactions with a friend (e.g., Bauminger et al., 2008b) and comparisons between interactions with friends and non-friends (Bauminger & Agam Ben Artzi, 2012). In Bauminger et al.'s study, HFASD and typical children's social interactions and their dyadic qualities were coded during an encounter with a friend in two different, noncompetitive play activities: building with blocks and drawing. Results demonstrated significant differences between the two groups in cooperative skills and positive affect on both the building and the drawing activities; differences in sharing were found only in building, and differences in nonverbal behavior were found only while drawing. The play scale, coded only for the building activity, showed a lower frequency of coordinated play and higher frequency of parallel play in the HFASD group than in the typical group. Pairs in which at least one child had HFASD also differed in conversational flow and social conversation. The HFASD group revealed a more rigid conversational style and engaged in less social conversation compared with the typical children. Group differences emerged in all dimensions of the dyadic relationship Q-set that evaluate dyadic quality of interaction between friends (Park & Waters, 1989) (positive social orientation, cohesiveness, harmony, responsiveness, coordinated play, and control), except control, with HFASD pairs demonstrating poorer dyadic quality of friendship than typical pairs. A global evaluation of affective closeness and shared fun found that friendships between children with HFASD were lower on these two dimensions compared with typical friendships. It is important to note that children with higher receptive language abilities enjoyed better friendship quality.

Thus, it is safe to conclude that the quality of friendships among children with HFASD is poorer than among typical children. The picture is not complete, however, until a comparison is made between HFASD children's interactions with friends and with non-friends, for only such a comparison can demonstrate the differential role of a friend to the enhancement of these children's social capabilities. In a recent study (Bauminger & Agam-Ben Artzi, 2012), the author and colleagues explored friendship in preschoolers with HFASD. Each child was videotaped interacting with a friend and with a non-friend while building with blocks, drawing, and during snack time. Results indicated that HFASD children showed more positive affect and higher degrees of closeness, shared fun, and reciprocity when interacting with a friend compared with a non-friend. Thus, friendship may serve an important function in the enhancement of social skills in HFASD.

To complete the discussion on friendship quality, it is important to discuss the partner's contribution to the friendship, namely, whether differences can be found between mixed and non-mixed friendships. In Bauminger et al. (2008a), the author and colleagues examined differences between mixed (HFASD and typical child), non-mixed (HFASD and child with disability, mainly with HFASD), and typical friendships (both children typical). Findings showed that mixed dyads

were similar to typical friendship dyads in many of the observational measures collected. More specifically, mixed typical and HFASD dyads differed significantly from non-mixed dyads but not from typical dyads in the amount of engagement in goal-directed activity, sharing, positive affect, and parallel play. Mixed dyads were found to be more responsive and cohesive and to be higher in positive social orientation and to show more complex levels of play (coordinated play), compared with non-mixed dyads. This finding points to the possible benefits to the HFASD child of having a typical child as a friend. Further, according to this study, HFASD children in mixed friendships differed from HFASD children in non-mixed friendships only in receptive language, demonstrating higher language skills. Other differences were not significant: HFASD children in both friendship types (mixed and non-mixed) showed high social-emotional characteristics including theory of mind (ToM), affective recognition, and attachment to parents.

On the other hand, an important advantage of non-mixed friendship (i.e., friendship between two disabled children) was the element of equality in the exchange: children in such dyads maintained a balance in the degree to which each partner assumed dominant or subordinate roles such as leader and follower, whereas children in mixed friendships had fewer leadership opportunities. Thus, it appears that interactions with a similarly disabled friend (most likely another child with HFASD) are also very important. Based on the studies described, a complete understanding of friendship quality in HFASD requires consideration of the characteristics of the friend, with seeming benefits from participation in both mixed and non-mixed friendships. Further studies should investigate the different benefits of participation in each of the friendship types for the child with HFASD.

Understanding of Friendship

Although interpersonal awareness and understanding are considered to be severely deficient in ASD (Hobson, 2005), few studies have focused on ASD children's ability to understand the concept of friendship. One example is Carrington, Templeton, and Papinczak (2003) study, which examined descriptions obtained through semi-structured interviews with five adolescents with Asperger syndrome aged 14–18, of what a friend is, as well as what a friend is not. The adolescents demonstrated difficulties explaining what a friend is: explanations were usually superficial and focused on sharing activities (e.g., computer games such as Dungeons and Dragons) and other areas of interest, without mentioning more emotional aspects of friendship, such as liking each other. Bauminger et al. (2004) provided additional support for these children's major difficulties in capturing the more affective dimensions of friendship. In their study, children (8–17 years) were shown a picture depicting two children in an intimate exchange, looking at each other, smiling, and appearing to be telling each other a secret. Participants were asked to suggest a title and make up a story to go with the picture and to say whether the children in the picture were friends. Only 50% of the HFASD compared to 81.3% of the typical control group named the picture in a way that referred to friendship. Among those who did, only one child with autism referred to the intersubjective sharing and closeness aspect of the friendship (the child suggested the title "soul mates"), compared with many of the typical children (e.g., "best friends," "secret friends," "brotherhood"). In Bauminger and Kasari (2000), HFASD children showed difficulty in all three defining aspects of friendship, namely, closeness, companionship, and intimacy. In sum, it is clear that the pragmatic understanding of friendship is lacking in these children, as well as the use of language to describe it. However, it is important to emphasize that lack of understanding may not necessarily preclude the experience of friendship. These children may have specific difficulties in reflecting about their interpersonal experience.

Future Directions

There is little doubt that ASD is a disorder involving severe deficits both in experiencing and understanding friendship. However, friendship is not an all-or-nothing concept, and for these

children, some of whom do have friends and whose friendships are of great importance to them, even more limited forms of friendship can be fairly durable. Although consistent difficulties have been demonstrated across studies examining the understanding and experience of friendship in ASD, a subgroup of ASD children has nevertheless emerged, who show higher interpersonal awareness and a better quality of friendship. The phenomenon of friendship in children with ASD would probably best be described as a continuum of interpersonal resources, with some children able to develop relationships or even meaningful and enduring friendships, others capable of more superficial attachments in which the friend is merely a playmate rather than an intimate "soul mate," and still others being unable to develop any type of stable relationship with a friend or even having not yet developed the desire to have a peer as a friend. This continuum should be further explored in order to identify what characterizes each of these subgroups, and such exploration is indeed under way in the field of typical friendship research, which is beginning to examine various types and functions of friendships (see, e.g., Kerns, 2000, for preschool friendship, and Shulman, 1993, for adolescents).

Just as the child with ASD clearly plays a significant role in determining the quality of the friendship, so does the friend, with typically developing friends probably helping to promote more complex interactions, and friends with a disability contributing more to aspects of equality in the friendship. Language skills have also been found important, with children of higher language skill appearing to develop higher quality friendships. In this respect, it is important to note that children with ASD in both mixed and non-mixed friendships reveal high social-emotional abilities such as ToM, security of attachment, and affective recognition. Perhaps, within the spectrum of ASD, these children with higher social, cognitive, and emotional resources do develop friendship. What helps children develop a friendship with a typical peer, beyond better language skills, has yet to be explored.

Another reason why friendship is an uncommon experience in ASD may be because social interventions tend to neglect focusing on friendship. Theoretical assumptions of friendship as a non-tangible experience for children within the spectrum may limit professionals' attempts to discover ways to support, encourage, and build up ASD children's ability to form friendships when planning social interventions. Only rarely is friendship a target of such interventions for this population, and very few social intervention programs (specifically at younger ages) are aimed at developing and maintaining friendships (e.g., Frankel & Myat, 2003). In adolescence, for example, children with ASD consistently perceive themselves as lonelier than typically developing children (e.g., Bauminger & Kasari, 2000). For these reasons, friendship should be considered an aim of ASD social intervention across the lifespan, and future studies need to further explore the necessary skills leading to the formation of friendship in ASD. Friendship in young children particularly merits thorough investigation due to its preventive value. Another important direction for research is the identification of early markers of higher interpersonal resources and capacities for richer friendships in children within the ASD spectrum. Also, since friendship has not been carefully explored in children with lower cognitive and language skills and little is known about these children's ability to form friendship, further research is warranted. Companionship skills seem to be easier to teach, but the real struggle is helping these children share their innermost feelings and thoughts and develop intimacy and affective closeness with a friend. To conclude, we are presently at the most initial stages of untangling the intricacies of interpersonal engagement with friends in ASD. Longitudinal studies exploring the phenomenon are needed, alongside the planning of interventional goals to enhance friendship in children with ASD.

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Froidir

► Clozapine

Frontal Lobe Findings in Autism

Nouchine Hadjikhani
Harvard Medical School, Charlestown, MA, USA

Synonyms

Emotion and decision making; Executive functions; Frontal lobe functions; Mirror mechanisms; Social cognition

Structure

Frontal Lobes: Anatomy and Function

The frontal lobes constitute the largest part of the human brain. Anatomically, they represent those areas of the cortex anterior to the central sulcus. They are the last to mature during development, and only reach full maturity by the age of 20.

Function

The frontal cortex can roughly be divided into the following parts with corresponding functions:
Frontal operculum: language, mirror mechanisms.
Precentral: motor.
Dorsolateral prefrontal: executive functions.
Orbitofrontal/ventromedial: emotion, decision making, response inhibition.
Anterior cingulate: choice selection, reward, social emotion, empathy, response monitoring.

Lesions of the frontal lobes are characterized by profound personality changes, described in the famous case of Phineas Gage (Damasio, 1994). Other elements of frontal dysfunction may include perseveration, aphasia, lack of spontaneous activity, indifference, or disinhibition. Some of the effects resulting from frontal lesions are due to the fact that the orbitofrontal cortex is extensively connected to the amygdala and subcortical structures, and can be considered as one of the elements of the limbic system.

In order to tap into their different functions, including language production, motor and executive functions, neuropsychological testing of frontal lobes is performed with a variety of tasks. A vast number of tests are available to test language production, including the Boston Naming Test. Neurological examinations are used to test for motor functions. Executive functions (EF) include skills required for action planning and execution, inhibition, organization, self-monitoring, cognitive flexibility, and set-shifting. EF tests include: Go/No-Go task, Train Making Test (TMT), Lexical Fluency, attention and concentration tests (e.g., serial 7), digit span, alternative sequence task, Tower of Hanoi (or of London), and Wisconsin Card Sorting task (WCST). In addition, a computerized battery for testing EF is available: the Cambridge Neuropsychological Test Automated Battery (CANTAB) (Robbins et al., 1994).

Below, we describe the findings of neuropsychological, anatomical, and functional studies in ASD.

Pathophysiology

Neuropsychological Studies

Executive functions: Abnormalities in executive functions are very common in autism, although their relationship with autistic symptomatology is complex (Joseph & Tager-Flusberg, 2004;

Kenworthy, Yerys, Anthony, & Wallace, 2008). In a recent review, Kenworthy et al. (2008) note that while there is a consensus about difficulties in various aspects of executive functions in daily life of individuals with autism, a number of questions remain in terms of their performance during neuropsychological testing. In particular, this review underlines the fact that while abnormal performance has been observed in tasks that required human administration and hence adherence to socially presented rules and interactions (such as the WCST, or the Tower tasks), less impairments are reported in ASD when tests are administered using CANTAB, a computerized battery.

Other confounding factors in EF studies in autism spectrum disorders (ASD) are IQ levels and age, with differential maturation trajectories of components of EF during adolescence. A robust finding, however, is the presence of spatial working-memory deficits. Ecologically valid EF control questionnaires (e.g., BRIEF) reveal that both children and adults with ASD have difficulties on unstructured tasks, but the potential confound with social demands on these measures still need to be better understood.

Mirror mechanisms: Mirror mechanisms were first described in primates and rapidly thereafter in humans by the group of Giaccomo Rizzolatti (Gallese, Fadiga, Fogassi, & Rizzolatti, 1996; Rizzolatti et al., 1996). The caudal part of the inferior frontal gyrus (corresponding to Broca's area on the left hemisphere) is part of the action mirror network. Mirror mechanisms translate sensory information during observation of an action done by others into that same action coded in he observer's motor system. The result of this sensory-motor transformation depends on the location of the mirror neurons: for neurons in the motor system, mirror mechanisms will make possible the understanding of action and imitation; for neurons located in the insula and the cingulate, mirror mechanisms will support emotional understanding and empathy. Language is very likely to have evolved from mirror mechanisms (Rizzolatti & Arbib, 1998).

The results of studies addressing imitation deficits in autism are sometimes contradictory (for review, see Sevlever and Gillis (2010)), probably partly due to the fact that the term "imitation" covers several categories of behaviors, including emulation, mimicry and true imitation, and that imitation tasks used in different studies are extremely variable.

Imitation performance in ASD depends on task type (Rogers, Hepburn, Stackhouse, & Wehner, 2003). Children with autism seem to be capable of elicited imitation, but do not show a tendency to spontaneously imitate (Ingersoll, 2008; Whiten & Brown, 1999; but see Bird, Leighton, Press, & Heyes, 2007). One common finding in ASD is the lack of automatic mimicry and spontaneous imitation of facial expressions (reviewed in Sevlever and Gillis (2010)). A recent theory, the EP-M model (emulation and planning-mimicry; Hamilton, 2008) suggests that while the EP route is intact, the M route, responsible for automatic mimicry and spontaneous imitation may be impaired in children with ASD.

Recent data show that training of motor imitation can reduce other symptoms (e.g., improve language) in ASD (Ingersoll & Lalonde, 2010).

Joint attention (*JA*), *theory-of-mind* (*ToM*), *social orienting*: Deficits in joint attention are one of the first symptoms to appear autism (for review, see Mundy (2003)). Capacity for joint attention normally emerges at 6 months of age, and is a prerequisite for social learning. ToM is the capacity to attribute mental states to oneself and others, and deficits in ToM are one of the current theories in autism ((Baron-Cohen, Leslie, & Frith, 1985), but see Tager-Flusberg (2007)). Additional controversies exist between supporters of ToM and those of a simulation theory, positing that other people's mental states, rather than being analytically processed by a ToM, are represented by a spontaneous perspective adoption supported by mirror mechanisms (Gallese & Goldman, 1998). JA and ToM may share common processes subserved by the medial prefrontal and anterior cingulate cortices, in conjunction with other parts of the "social brain" (Brothers, 1990). Impairment in the medial prefrontal/anterior cingulate may be related to the difficulties seen in autism to initiate joint attention, to self-monitor, and to orient toward social stimuli.

Anatomical Studies

Brain Volume and Cortical Thickness

Anatomical differences in ASD brain are reviewed in Amaral, Schumann, and Nordahl (2008). A common theory posits that ASD brains undergo a period of precocious growth during early postnatal life, followed by a deceleration (for review, see Courchesne, Redcay, and Kennedy (2004)) related to a disproportionate increase in white matter (Herbert et al., 2003). Greatest increases in volume have been observed in the dorsolateral prefrontal and medial frontal cortices (Carper, Moses, Tigue, & Courchesne, 2002; Herbert et al., 2004), whereas no differences have been reported in the orbitofrontal cortex. A recent longitudinal study reports that by age 2.5, both cerebral gray and white matter are significantly enlarged in toddlers with ASD, with the most severe enlargement occurring in frontal, temporal, and cingulate cortices (Schumann et al., 2010). Abnormalities in gyrification of the inferior frontal gyrus, as well as cortical thickness decreases in the inferior frontal cortex have been described (Ecker et al., 2010; Hadjikhani, Joseph, Snyder, & Tager-Flusberg, 2006). In the dorsolateral prefrontal cortex, Casanova et al. have observed abnormal minicolumns (Casanova, Buxhoeveden, Switala, & Roy, 2002). A recent study reports increased neuron number and size in the prefrontal cortex of children with autism (Courchesne et al. 2011).

Diffusion Tensor Imaging (DTI) Studies

Multiple studies have described white matter alteration in the frontal lobes in ASD. Most DTI studies report reduced fractional anisotropy (FA) in frontal white matter in adolescents and adults with ASD (Alexander et al., 2007; Barnea-Goraly et al., 2004; Keller, Kana, & Just, 2007; Thakkar et al., 2008) as well as in children (Kumar et al., 2010; Sundaram et al., 2008).

Opposite findings (increased FA in the frontal lobe) are reported in children, suggesting the role of a developmental component, with an early and accelerated abnormal maturation of the white matter (Ben Bashat et al., 2007). Increased myelination of the frontal lobe was also described in a study using T2-weighted imaging in young children with ASD (Carmody & Lewis, 2010).

Functional Imaging

Activation Studies

Activation studies have addressed different functions of the frontal lobes.

Language: Semantic processing is associated with stronger activation in Broca's area in adolescents with ASD, who also exhibit less laterality for this area (in the context of similar behavioral performance) (Knaus, Silver, Lindgren, Hadjikhani, & Tager-Flusberg, 2008). In adults, other studies have shown reduced activation in Broca's area during semantic processing; however, behavioral performance was not equal between groups, making the interpretation of the data difficult.

Motor functions: Very few functional studies have examined the imaging correlates of motor dysfunctions in autism. Mostofsky et al. (2009) describe greater activation of the supplementary motor area (SMA) during finger tapping task in children with autism, compared to controls who tend to engage the cerebellum for this type of task.

Executive functions: Three main caveats need to be underlined in functional imaging of EF in ASD. The first critical issue is the necessity to compare brain activation in tasks where behavioral performance is similar between the groups being compared. A second issue is that the fact that age may play a role in the synchronization of frontal and fronto-striatal brain regions involved in response inhibition, and that in consequence age groups need to be strictly defined and homogeneous. Finally, one has to bear in mind that an abnormal developmental trajectory may also be the basis of the differences observed in children and adolescents with ASD.

The majority of functional magnetic resonance imaging (fMRI) studies of EF in ASD have demonstrated diminished brain activation compared to controls, with some exceptions (reviewed in Solomon et al. (2009)).

Mirror mechanisms: Several functional studies using different imaging techniques including

fMRI, electroencephalography (EEG) and magnetoencephalography (MEG) have reported abnormalities in mirror mechanisms in ASD. In neurotypicals, action observation is typically associated with a suppression of the mu rhythm in the EEG signal – a phenomenon that is absent in ASD (Oberman, Ramachandran, & Pineda, 2008). Using transcranial magnetic stimulation (TMS), Theoret et al. (2005) have demonstrated that TMS applied over the primary motor cortex, did not modulate the excitability of the primary motor cortex in children with ASD, contrary to controls.

fMRI studies in children and in adults have reported weaker activation of the frontal operculum during face or body emotion expression perception (Dapretto et al., 2006; Hadjikhani, Joseph, Snyder, & Tager-Flusberg, 2007; Hadjikhani et al., 2009).

However, recent studies report normal (Dinstein et al., 2010) or even enhanced (Martineau, Andersson, Barthelemy, Cottier, & Destrieux, 2010) activation of mirror system areas during action observation, leaving the question of atypical activity of the MNS in autism open.

Reward Processing: One of the current hypotheses about social impairments in autism is a lack of social motivation, due to a decreased reward value for social stimuli. Very few studies have examined the neural correlates of reward processing in autism, but recently, diminished neural responses to both social and monetary reward, but not to objects of interest, have been described in children (Scott-Van Zeeland, Dapretto, Ghahremani, Poldrack, & Bookheimer, 2010) and adults (Dichter et al., 2010) with ASD. However, increased anterior cingulate activation during monetary reward achievement has also been described in adults with high functioning ASD (Schmitz et al., 2008).

Social emotion, joint attention, theory-of-mind (ToM): Reduced anterior cingulate activation has been observed in tasks of ToM (Happe et al., 1996; Wang, Lee, Sigman, & Dapretto, 2007), but normal activation has been reported in middle prefrontal cortex during social emotional tasks (Schulte-Ruther et al., 2010).

Response monitoring: Abnormal (increased) anterior cingulate activation has been shown in response to correct trials during response monitoring in ASD (Thakkar et al., 2008). In summary, functional activation studies remain controversial and a better understanding of frontal functions in autism will require further investigation.

Functional Connectivity Studies

A common finding of functional connectivity studies has been the presence of underconnectivity in ASD between large-scale networks and over-connectivity in small-scale networks. In most studies specifically addressing connectivity of the motor system (Mostofsky et al., 2009) and of the EF system (Just, Cherkassky, Keller, & Minshew, 2004; Kana, Keller, Minshew, & Just, 2007; Koshino et al., 2005; Solomon et al., 2009; Thakkar et al., 2008), underconnectivity was reported. However, other groups show no differences (Lee et al., 2009) or even increased (Shih et al., 2010) connectivity in the frontal regions in ASD (superior frontal and anterior cingulate) – concluding to atypical connectivity of the imitation network with an enhanced role of the dorsal prefrontal cortex.

Nevertheless, one major caveat needs to be kept in mind for functional connectivity studies: while many studies described decreased functional connectivity in ASD, the majority of these studies also describe a difference in the magnitude of activation between groups that may bias the connectivity results toward finding group differences (discussed in Lee et al. (2009)). In addition, a recent study has pointed to the risk that slightly different motion magnitudes may induce spurious differences between groups, and this factor will need to be specifically addressed in future studies (Van Dijk, Sabuncu & Buckner, 2012).

Perspectives

Because of the developmental trajectory of executive functions and of the frontal lobes in general,

future studies will need to have larger number of participants, with homogeneous age groups and cognitive abilities (IQ). In addition, functional studies will need to be designed so that behavioral performance is equal between groups, if brain activations need to be compared for a specific task.

See Also

- ▶ Executive Function (EF)
- ▶ Mirror Neuron System

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Frontal Lobe Functions

► Frontal Lobe Findings in Autism

Frontal Lobe Syndrome

Nicole Rinehart, Peter Enticott and John Bradshaw
Faculty of Medicine, Nursing and Health Sciences, Monash University, Melbourne, VIC, Australia

Synonyms

Disorder of executive dysfunction; Dysexecutive syndrome; Frontostriatal disorder

Short Description or Definition

The frontal lobes are the slowest regions of the central nervous system to fully develop, rendering them more susceptible to neurodevelopmental disorders, such as autism spectrum disorder, than any other region (Bradshaw, 2001). The frontal lobes form part of key brain regulatory circuitry where multiple cortico-striato-thalamo-cortical loops connect the frontal lobes to the rest of the brain. Frontal lobe syndromes have in common abnormalities in structure and chemical balance in the frontal lobes, basal ganglia, and thalamus. Abnormalities in these structures and chemical imbalances are proposed to lead to deficiencies in the domains of cognition, emotions, and motor control. A mixture of these deficiencies can explain the symptomatology observed in classic frontal syndromes of childhood, for example, autism, Asperger's disorder (henceforth to be referred to as "autism spectrum disorder"), and attention deficit hyperactivity disorder (ADHD), and neurodegenerative frontal disorders, for example, Parkinson's and Huntington's disease. The common basis accounts for why there is high comorbidity within neurodevelopmental and neurodegenerative sub-groupings of frontal syndromes; for example, it is common for children with autism to also have clinical manifestations of ADHD. Such comorbidity may also point to common heritability factors across syndromes.

Categorization

Disorders which arise from damage to the frontal lobes are often referred to under the umbrella term of "frontal lobe syndrome." Frontal lobe syndrome can result as the consequence of: (1) developmental problems with the frontal lobe, as is the case with autism spectrum disorder, ADHD, obsessive compulsive disorder (OCD), Tourette's disorder (TD) schizophrenia, and depression, (2) degeneration of the frontal lobes, for example, Parkinson's disease, Alzheimer's disease, and Huntington's disease, or (3) Acquired brain injury, for example, stroke or traumatic brain injury.

While autism is defined in DSM-IV-TR (American Psychiatric Association, 2000) as a psychiatric disorder, it is commonly referred to as a disorder which crosses the psychiatry-neurology divide, involving both "psychiatric" and "neurological" symptoms. When we consider autism under the umbrella of a "frontal lobe syndrome," we group autism with other disorders where vital brain pathways which connect the frontal lobes to the rest of the brain are disrupted, either through genetic abnormalities, or environmental insult. While some of these disorders fall within the categorization of "neurological" disorders, for example, Parkinson's disease, others fall under the categorization of psychiatric disorders, for example, autism and ADHD. It has been argued that the division between neurology and psychiatry is in many ways arbitrary; in the case of autism, frontal-related symptoms such as "repetitive motor mannerisms" could be considered as a neurological "motor disorder" or as a psychiatric "perseverative" phenomenon.

The question remains whether the presence of a particular frontal lobe syndrome is a necessary precursor for the development of autism and AD, given that some autistic behaviors (e.g., stereotyped or ritualistic activities) are present in other childhood psychiatric disorders, such as TD, OCD, and ADHD. It may be considered that autism, TS, OCD, and ADHD are five clusters of behaviors that represent disorders in several discrete dimensions, such as communication, social skills, or stereotyped/ritualistic/obsessive behavior. Autism and AD may be thought of as occurring at the same end of the social ability continuum, but as occurring at different ends of the communication ability continuum, while OCD and TS may lie toward the less-impaired ends of these dimensions. Alternatively, on the stereotyped movements-obsessive behaviors dimension, autism and TS may be thought of as occurring at the "stereotyped movement" end of the continuum, with OCD occurring at the "obsessive behaviors" end. These diagnostically distinct disorders may simply reflect different combinations of a finite set of behavioral disturbances (Bradshaw, 2001).

Epidemiology

ASDs are highly prevalent with the incidence estimated at between 60 and 90 per 10,000 individuals (Autism & Developmental Disabilities Monitoring Network Surveillance Year, Principal Investigators, Centers for Disease Control and Prevention, 2009; Fombonne, 2009), although a recent study of students aged between 5 and 9 years in the United Kingdom sets this estimate to approximately 160 in 10,000 (Baron-Cohen et al., 2009). It is likely that the apparent increase in prevalence is due to better diagnosis and awareness in the general public. The apparent "epidemic" of ASD is similar to the increased ascertainment of ADHD in the 1990s. For this reason, "ASD" is sometimes referred to in the context of being the "new ADHD."

Although the precise cause of executive dysfunction in ASD is not known, researchers have used neuroimaging and electrophysiological techniques to demonstrate atypical activation across a range of frontostriatal cortical and subcortical structures. This includes, for example, hypoactivation of inferior frontal gyrus, orbitofrontal cortex, and ventral medial prefrontal cortex, and hyperactivation of supplementary motor area, dorsal anterior cingulate, and middle frontal gyrus. This is presumably underpinned by various neurochemical abnormalities that have demonstrated relevance to both executive function and ASD, including abnormal serotonergic, dopaminergic, glutamatergic, and GABAergic function. More recent evidence also points to the role of impaired neural connectivity within frontostriatal circuitry, which includes reduced integrity of white matter pathways. There are, however, no consistent results in ASD demonstrating structural abnormalities (e.g., lesions) that reflect what is typically seen in frontal lobe syndrome (FLS) associated with acquired or neurodegenerative presentations.

Natural History, Prognostic Factors, Outcomes

While autism manifests in the first 3 years of life, the full extent of the disorder unfolds as the slowly

developing frontal lobes reach maturity; this in part contributes to the changing clinical manifestation of autism throughout childhood and adolescence (see Clinical Presentation below).

The term most commonly used to describe the impairments which arise from disruption to the frontal lobes is "executive dysfunction." Executive dysfunction can be thought of as a loss or disruption to skills which we require to interact in a flexible way with the environment, solve problems, plan, engage efficiently in goal-directed behavior, and defer or inhibit responses. Overall, frontal lobe syndrome results in a deficit in maintaining appropriate problem-solving set for the attainment of future goals (Bradshaw, 2001).

Pennington et al. (1997) proposed the "executive dysfunction hypothesis" of autism spectrum disorder on the basis that there is severe, early disruption to the frontal areas, disrupting the ability to plan complex behaviors. Working memory deficits are also noted as being important to this theory. Poor performance on tests of executive functioning is common in children with autism.

Neuropsychological research has shown that two core aspects of frontal lobe function are disturbed in autism: (1) reduced cognitive flexibility, which refers to the ability to shift one's focus of attention from one feature, or cognitive set, to another and (2) perseveration, which refers to a tendency to repeat a verbal or nonverbal behavior beyond what is required. These frontal syndrome deficits underpin the daily challenges a child with autism experiences with planning and organizing tasks required for everyday life. The ability to sustain attention and inhibit responses has been shown to be generally intact in individuals with autism using standardized neuropsychological tests (see evaluation and differential diagnosis below). Using more novel experimental paradigms, there is some evidence to suggest that one aspect of inhibitory function may be impaired in autism, verbal inhibition (see Rinehart, Bradshaw, Moss, Brereton & Tonge, 2006). Such deficits are also seen in patients with neurodegenerative "frontal lobe" syndrome, Parkinson's and Alzheimer's disease, and neurodevelopmental disorders such as schizophrenia.

The difficulties an individual with autism has with cognitive flexibility persist into adult life. In contrast to children with autism, adults with autism can develop better skills for managing executive dysfunction, either through further acquirement and practice of techniques to scaffold executive difficulties (see Treatment below), via greater insight into their own inflexibility, or through greater desire to follow the rules and behaviors necessary for successful adult life.

There is some evidence that children with autism experience a "second wave" of executive deficits around puberty due to a divergence in the developmental trajectory of the frontal lobes (Minshew & Williams, 2007). At around this time children with autism are also likely to suffer from depression; this may manifest as mood disturbance and irritability, sleep and appetite disturbance, and thoughts of suicide (Tonge & Rinehart, 2006). Organizational and planning deficits can become more pronounced when children are depressed or anxious. In addition to pubertal brain development, particularly impacting on the frontal lobes, the increased vulnerability to depression during adolescence may be associated with increased insight into the disorder. Graduation from Primary School, or other change in educational placement, which tends to coincide with puberty, is often a major stress for children with autism. When individuals with autism exhibit self-injury and aggression, in addition to executive dysfunction, as often occurs with individuals who have associated intellectual disability, this can result in socially inappropriate and "disinhibited" behavioral patterns which can reduce successful participation in the school and community settings, and may require more restricted care plans.

Comorbidity with other neurodevelopmental disorders, which fall under the umbrella term of "frontal lobe syndromes," may also impact on quality of life. This comorbidity may be genetically determined, as is likely the case with ADHD, a stress-induced vulnerability, or both, as is the case for Tourette's disorder. For example, clinically significant symptoms consistent with ADHD are reported to be highly prevalent in children with autism spectrum disorder. (Note

that despite this overlap in diagnostic symptoms, our major diagnostic classification system (DSM-IV-TR) currently precludes a dual-diagnosis of autism and ADHD.). There is some empirical evidence that children who present with autism and clinically significant levels of ADHD have higher levels of emotional-behavioral disturbances (Gargaro, Rinehart, Bradshaw, Tonge & Sheppard, 2010). In the case of Tourette's disorder, a frontal syndrome vulnerability, together with environmental stress, possibly related to biological or situational changes (see above), may trigger the comorbid presentation of Tic disorder or full-blown Tourette's disorder.

Clinical Expression and Pathophysiology

Executive dysfunction, in part, gives way to one of the most striking paradoxes of autism, the contrasting and uneven development of skills, sometimes characterized by peaks in certain areas of intellect, artistic, or numerical ability, which occur in the context of an impoverished ability to manage and organize the tasks necessary for daily life. In general, the impact of executive dysfunction on an individual with autism varies according to the individuals' intellectual ability, capacity for independent functioning, and provision of social, emotional, financial, and later, occupational support. It has been suggested that individuals with associated intellectual disability and epilepsy are at greater risk for deterioration during early adulthood, while there may be some improvement in functioning in normally intelligent young adults with ASDs.

Frontal lobe syndrome or executive dysfunction is commonly linked to the repetitive, stereotyped behavioral patterns which are core diagnostic features of autism (Turner, 1999). The repetitive behavioral patterns may be due to a difficulty inhibiting previously relevant response patterns, or may be due to a problem with generating new ideas and responses (Turner, 1999). Common repetitive, restricted, and stereotyped behavioral patterns include: lining up toys or objects, being preoccupied with special objects such as stones, repeating play sequences, obsessions with train timetables or dinosaurs. Individuals with autism may have a number of rituals associated with daily life, such as a fixed order for bathing and dressing, or an insistence on wearing the same clothes or taking the same route to a familiar place. These rituals go beyond what is normally seen in typically developing children. There are usually some motor mannerisms, such as hand flapping, or tiptoe walking and gait abnormality.

The symptoms of frontal lobe syndrome in autism cannot be neatly separated out from the broader network of behavior-brain disruptions which characterize autism. For example, children with autism may study the detail in a picture book or closely observe spinning wheels, or a ceiling fan; this may be due to problems with disengaging from interests (executive dysfunction), or it might be due to an overly developed visual-perceptual system which enhances the experience of looking at small objects (Plaisted, Swettenham & Rees, 1999). Similarly, a child with autism may have difficulty interacting with the peer group, not primarily because of social impairments related to "theory of mind" deficits, but perhaps because of an inability to shift attentional set from his or her own topics of interest. Therefore, a child with autism may engage in a monologue of speech about his favorite interests rather than a reciprocal dialogue with his peers either due to executive-based inflexibility, or due to a poor understanding that "turn taking" is required in productive social conversations (theory of mind deficit).

The clinical manifestation of frontal lobe syndrome may differ in girls with autism in a way which is consistent with gender-related differences in the typically developing population of male and female children. For example, while boys may have an intense preoccupation with vehicles, such as trains and cars, girls may develop their special interests in areas such as teddy-bear or doll collections, and spend unusual amounts of time on craft and art to the exclusion of other types of play. Gender-neutral activities, such as "drawing," may form the "special interest" for either males or females with autism. It has been

suggested that females tend to have fewer special interests than males with autism (Gillberg & Coleman, 2000), although this may relate to male interests being more circumscribed (e.g., trains, cars) than female interests (e.g., craft, doll play), and thus more clinically salient.

Pathophysiology

Development of the frontal lobes is guided by regulatory genes which lay down and prune brain pathways (referred to as apoptosis), and set the timing of how the brain will be "wired" (referred to as synaptic connectivity). There is a disturbance in these regulatory genes in autism which results in atypically developing frontal regions. The current literature points to the developmental time course of brain development being the salient disturbance, rather than the "final product" of brain development in autism (Amaral, Schumann & Nordahl, 2008, p. 137). The unfolding neurobiological disruptions which characterize autism may contribute to the changes in core symptoms of autism we see over time, such as stagnation and regression in social-communicative symptoms, and changes in the focus of obsessions and nature of repetitive behavioral patterns.

Bradshaw's (2001) frontostriatal theory provides the most comprehensive review of the pathophysiology of "frontal lobe" syndrome in the context of neurodevelopmental disorders. According to this theory, the frontostriatal system involved in neurodevelopmental disorders comprises the dorsolateral prefrontal cortex, the lateral orbitofrontal cortex, the anterior cingulate, the supplementary motor area, and the basal ganglia. These areas are involved in a number of functions, such as executive functions (e.g., functions such as self-monitoring, planning, organization, flexibility of thinking, and inhibition), motivation, control of complex behaviors, and sequencing movements. A number of neurotransmitters are implicated in the frontostriatal system, including dopamine, noradrenaline, acetylcholine, GABA (inhibitory), glutamate (excitatory), and serotonin.

Evaluation and Differential Diagnosis

Frontal lobe syndrome (FLS) resulting from acquired brain injury involves impairment in cognitive and/or mood domains that are presumed to be largely governed by neural circuitry involving the frontal lobe. This may include difficulties with planning, organization, daily living skills, attention, and social cognition. Individuals with FLS may be disinhibited and impulsive, and this can include aggression or inappropriate sexual, financial, or other behaviors. Depression, anxiety, and apathy are often also present. FLS in the purest sense is thought to arise from a lesion, be it from disease or trauma. This lesion can occur anywhere within fronto-striatal-thalamic circuitry, and the nature and degree of impairment is related to the site of injury. As noted above, FLS can also relate to neurodevelopmental, e.g., ASD and neurodegenerative disorders, e.g., Parkinson's disease.

In addition to deficits in executive function, FLS syndrome can also involve impaired social functioning, including poor emotion recognition and theory of mind, and perseverative and repetitive behaviors. Individuals with FLS can also be quite rigid and concrete in their thinking. For these reasons, ASD is often referred to under the FLS umbrella. In addition, there is much evidence for executive deficits among individuals diagnosed with an ASD, and this can include problems with planning, time estimation, attentional set-shifting, and response inhibition.

Obtaining a thorough and careful developmental history is critical for differential diagnosis. Symptoms of FLS related to lesions typically have a relatively rapid onset, and are recognized because they are uncharacteristic of the individual. ASD, on the other hand, generally must first present within certain time periods, and there is emerging evidence to suggest that some symptoms are present in infancy. When assessing individuals later in life (e.g., adolescence onward), it is crucial to involve family members in the diagnostic process, and to conduct a structural magnetic resonance imaging (MRI) brain scan.

Assessment for autism spectrum disorder (ASD) should include neuropsychological testing,

which at a minimum would involve an age-appropriate, standardized cognitive assessment, but perhaps also tests of executive function. This might include inhibitory controls (e.g., word-color Stroop test), attentional set-shifting (e.g., Wisconsin Card Sorting Test), and planning/organization (e.g., Tower of Hanoi, Trail making test). Verbal fluency assessment may also be used (e.g., FAS). This will characterize an individual's strengths and weaknesses, and allow clinicians to devise an appropriate treatment plan that meets the individual's needs. For very young children, or those for whom standardized assessment is not possible, parent and teacher report can provide insights into relative strengths and weaknesses, including problematic behaviors.

Treatment

The best outcomes for managing clinical symptoms associated with executive impairment in autism are produced by a combination of psychological, educational, and medical approaches if indicated. Treatment should be designed to target the specific needs of an individual child and also to meet the needs of parents and schools (Tonge & Rinehart, 2006).

Psychosocial

Training in problem solving and organizational skills can be useful for targeting difficulties with generating new or novel behaviors and moving the individual away from rigid thinking styles. Interventions which capitalize on a child's special interests, for example, technology, may improve compliance. Reminders, schedules, visual prompting (e.g., when I finish playing trains, I can choose from a list of these activities to do), and executive-function "scaffolding" by care-givers, (e.g., breaking down tasks into smaller components, helping individuals to structure daily events to prevent becoming submerged in one activity during the day, assisting with school-based tasks) are often helpful.

Pharmacological

Drugs might be prescribed where psychological and behavioral interventions have not been successful in the management of disturbed emotions and behaviors, in particular, repetitive self-injurious behaviors which place children at greater psychological and physical risk. There is some evidence that neuroleptic medication (e.g., haloperidol, risperidone) reduces repetitive and stereotyped behaviors; however, it may produce side effects including sedation, dystonic reactions, and increased weight gain. Unlike the original neuroleptic medications, new atypical drugs are not associated with the risk of irreversible tardive dyskinesia. Tricyclic antidepressants (e.g., imipramine, clomipramine) are effective anxiolytics, and may also reduce repetitive behaviors, but may have potential cardiotoxic effects (Tonge & Rinehart, 2006). Parents may be stressed by the uncertainty of potential long-term effects of placing their child on medication. Currently there is a lack of good long-term evidence for the effectiveness of stimulant medication on learning but they may decrease the risk of later substance abuse (Tonge & Rinehart, 2006).

See Also

- ▶ Frontal Lobe Findings in Autism
- ▶ Prefrontal Cortex

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Frontostriatal Disorder

▶ Frontal Lobe Syndrome

FTT

▶ Finger-Tapping Test

Full Physical Prompt

▶ Hand-Over-Hand Assistance

Functional Analysis

Mark Palmieri
Feeding Clinic, Center for Children with Special Needs, Glastonbury, CT, USA

Definition

Assessing behavior through data-based collection procedures is a necessary component of applied behavior analysis (Cooper, Heron, & Heward, 2007). The primary goal is to establish a reliable relationship between the treatment and behavior change. This concept forms the basis for the assessment of challenging behavior in applied behavior analysis. Functional analysis is the procedure by which environmental conditions are manipulated to reliably evoke a target behavior (Carr & Durand, 1985; Cooper & Harding, 1993; Iwata, Dorsey, Slifer, Bauman, & Richman, 1982/1994; Iwata, Pace, Kalsher, Cowdery, & Cataldo, 1990; O'Reilloy, Rispoli, Davis, Machalick, Lang, Sigafoos, 2009; Roscoe, Kindle, & Pence, 2010). Based upon the results of these assessments, maladaptive behavior is conceptualized as being motivated by a particular function, thus allowing appropriate interventions to be developed. An important component of functional analytic methodology is that behavior must be understood by its consequences within the environment and not solely by its topography or form (Cooper & Harding). The majority of functional analysis procedures currently used are based upon the seminal work of Iwata, Dorsey, et al. (1982/1994).

Functional analysis methodology has been applied to a variety of treatment settings. The original research on these procedures was conducted in highly controlled treatment settings (Carr & Durand, 1985; Cooper & Harding, 1993;

Iwata, Dorsey, et al., 1982/1994; Iwata et al., 1990). In these environments, experimenters have exposed participants to repeated treatment conditions in order to establish a reliable relationship between environmental contingencies and the occurrence of a target behavior (Carr & Durand, 1985; Iwata, Dorsey, et al., 1982/1994; Iwata et al., 1990). Research attention has expanded toward evaluating the use of functional analytic procedures in outpatient settings with less precise and sustained control (Cooper & Harding, 1993). This research has provided a great deal of support for brief functional analysis as a means of assessing challenging behavior (e.g., Cooper et al., 1992; Derby et al., 1994; Derby, Wacker, Sasso, et al., 1992). These assessments follow the same conceptual guides as does extended functional analysis; however, their structure and duration allows for them to be more successfully integrated into outpatient settings.

Historical Background

Carr (1977) established the conceptual foundation for functional analysis. The model for clinical practice is associated with the work of Iwata, Dorsey, et al. (1982/1994) and Carr and Durand (1985). The methodology developed in these studies has since been widely examined and used in the applied behavior analysis literature (Hanley, Iwata, & McCord, 2003). The initial study by Iwata, Dorsey, et al. (1982/1994) utilized four assessment conditions to evaluate self-injurious behavior in nine individuals diagnosed with developmental disability, ranging in age from 19 months to 17 years 2 months. The conditions included in the functional analysis were social disapproval, academic demand, unstructured play, and alone. During the assessments, conditions lasted for 15 min and were randomly ordered for each participant. The functional analysis continued until stable levels of responding were observed in each condition, or until 12 days of assessment were completed.

The social disapproval condition was designed to replicate contingencies for positive reinforcement in the form of attention for engaging in self-injury. The participant was instructed to play with toys while the experimenter worked. If the participant engaged in self-injury, the experimenter provided physical and vocal attention. The academic demand condition tested for the presence of negative reinforcement contingencies in the form of escape from work for engaging in self-injury. The experimenter ran academic programs appropriate to each participant's ability level. Social praise was delivered after each response whether or not the response was correct. If the participant engaged in self-injurious behavior, the experimenter turned away and terminated the learning trial. The alone condition was designed to assess for self-injurious behavior maintained by automatic reinforcement. The participant was left alone without access to attention or tangible items. The experimenter did not provide a consequence for an occurrence of self-injury. Finally, the unstructured play condition served as a control condition for the functional analysis. In this condition, the experimenter provided noncontingent attention and gave no demands. Again, no consequence was provided contingent upon an occurrence of self-injury.

To address individual differences in the topography of self-injury, operational definitions were provided for the self-injury that each participant experienced. Interobserver agreement was calculated to ensure that all observers were able to reliably identify all the topographies of challenging behavior. The results of the Iwata, Dorsey, et al. (1982/1994) study demonstrated that similar topographies of behavior can serve different functions. In their study, the level of responding varied from individual to individual across the assessment conditions. As a result of these data, Iwata, Dorsey, et al. (1982/1994) supported functional analysis as a means of systematically evaluating the stimuli maintaining behavior and subsequently the use of individualized assessment and intervention procedures for self-injurious behavior.

Carr and Durand (1985) echoed the Iwata, Dorsey, et al. (1982/1994) results, showing that similar forms of challenging behavior can be maintained by different contingencies in each individual. The study evaluated a number of topographies of challenging behavior experienced by four participants, ages 7 to 14 years, with either developmental disabilities or brain damage. The functional analysis conditions were designed to assess escape and attention motivations each target behavior. The "easy 100" condition served as the control condition for the analysis. In this condition, the experimenter provided easy demands and attention during 100% of the condition's intervals. In the "easy 33" condition, the experimenter again utilized easy demands but only provided attention during 33% of the intervals. This condition was used to assess for an attention motivation for each target behavior. During the "difficult 100" condition, the participants were given challenging demands and attention during 100% of the condition's intervals. It was expected that this condition would assess for an escape function maintaining any target behavior.

Consequences were provided for all topographies of behavior in the same manner during each condition. All behavior except darting and responses that risked physical injury was placed on extinction (Carr & Durand, 1985). If the participant darted from work and did not return in 10 s, she or he was physically guided back to the table. In cases where physical risk was a concern, the participant's hands were restrained for 5–10 s while the experimenter followed through with the work demands. The results of the functional analyses suggested that the various forms of challenging behavior of the participants were maintained by different environmental contingencies. The data from the Carr and Durand study supported those obtained by Iwata, Dorsey, et al. (1982/1994). That is, functional analysis was endorsed as a means for evaluating challenging behavior.

Carr and Durand (1985) further supported functional analysis as a means of assessing and treating challenging behavior by implementing functional communication training for each of the participants. The targets of the training were mands for attention and help, consistent with the attention and escape motivations for the forms of challenging behavior. By providing consistent reinforcement for appropriate requests for attention and help, all participants' challenging behavior decreased. Functionally equivalent interventions were thereby supported as the optimal treatment for challenging behavior.

Current Knowledge

A great deal of research has been conducted on functional analysis methodology since the seminal studies by Carr and Durand (1985) and Iwata, Dorsey, et al. (1982/1994). Systematic reviews of the literature on functional analysis have consistently supported the procedure's efficacy in identifying the function or functions of challenging behavior (e.g., Hanley et al., 2003; Iwata, Pace, et al., 1994; O'Reilloy et al., 2009). Hanley et al. reviewed 575 functional analysis studies, 96% of which rendered usable outcomes. While the functional analysis procedures utilized in typical studies are rarely identical, the basic premise of controlled antecedents and consequences as defined by environmental manipulations has aided in the development of functionally equivalent interventions that have shown a decrease in problem behavior and an increase in targeted replacement skills (Hanley et al.; Iwata, Pace, et al., 1994).

Hanley et al. (2003) reviewed the literature to identify trends for best practices in functional analysis methodology. Their review supported the use of functional analysis to study many topographies of challenging behavior in individuals with disabilities of varying severities. While functional analysis has been applied primarily to learners with pervasive developmental disorder or intellectual disability, it is important to note that a variety of other mental disorders and mild behavior problems have been included in analyses (e.g., Cooper, Wacker, Sasso,

Reimers, & Donn, 1990; Doggett, Edwards, Moore, Tingstrom, & Wilczynski, 2001).

The experimental conditions most prevalent in the literature are developed based upon those used by Iwata, Dorsey, et al. (1982/1994). These conditions, social positive reinforcement (attention), social negative reinforcement (escape), automatic reinforcement (alone), and control, appear as they were described above. In addition, a tangible condition has been applied to in a number of studies (e.g., Fisher, O'Connor, Kurtz, DeLeon, & Gotjen, 2000; Moore, Mueller, Durbard, Roberts, & Sterling-Turner, 2002; Mueller, Wilczynski, Moore, Fusilier, & Trahant, 2001; Shirley, Iwata, & Kahng, 1999). In this condition, the individual is given access to a highly preferred item for one minute at which point the experimenter removes the item and places it out of reach (Mueller et al.). The participant is told that the target item is unavailable and directed toward other low-preference items. Upon the occurrence of the target behavior, the experimenter grants access to the high-preference item.

A number of concerns are common in developing the conditions for a functional analysis. These concerns primarily focus on the presence of confounds in the assessment conditions (Hanley et al., 2003; Moore, Mueller, et al., 2002; Shirley et al., 1999). Hanley et al. note that the research on functional analysis has yet to render a unified set of rules for assessment implementation; however, components have been identified that can be considered among best practices. Included here are topics such as limiting assessment to a manageable number of responses, considering the influence of establishing operations on the contingencies active in each condition, relatively short sessions, brief designs that can be expanded on an individual basis, and programming for consequences (Hanley et al.).

Concerns regarding session confounds were illustrated by Moore, Mueller, et al. (2002) regarding the influence that attention can play during a tangible condition. A functional analysis was conducted on a child's self-injurious behavior (SIB). The results of the analysis suggested that SIB was a multi-operant behavior maintained by positive reinforcement in the form of attention and access to preferred items (Moore, Mueller, et al., 2002). In a follow-up analysis, the level of attention provided during the tangible condition was evaluated. By reducing the amount of attention paired with the presentation of the tangible, the rate of SIB was decreased. Moore, Mueller, et al. (2002) suggested that the attention inadvertently delivered during the tangible condition was acting as a confound and evoking SIB. Weakening the contingency between the target behavior and access to attention (e.g., delivering attention noncontingently) may serve to control for the influence of confounds (Moore, Mueller, et al., 2002). If the contingent presentation of attention does confound the tangible condition, it stands to reason that all independent variables should be carefully controlled during the development of functional analysis sessions.

Another methodological concern was identified by Shirley et al. (1999) in a study on incidental maintenance in the tangible condition. A functional analysis conducted on an individual's hand mouthing suggested that the behavior was maintained by automatic reinforcement and access to tangible items. Observations of the behavior indicated that the preferred items used in the assessment were almost never provided as a natural consequence. Therefore, the functional analysis may have identified a tangible function that was not actually maintaining the supported as a means for could have if presented contingently (Shirley et al.). Shirley et al. suggest caution when using the results of a preference assessment without collecting some form of data on the natural environment.

Session duration is an important topic in functional analysis methodology. A number of studies have demonstrated the use of functional analysis in less controlled settings such as schools and outpatient clinics (Cooper et al., 1992; Cooper & Harding, 1993; Iwata et al., 2000; Moore, Edwards, Sterling-Turner, Riley, DuBard, & McGeorge, 2002; Umbreit, 1995). To be applied within these settings, topics related to the efficient application of functional analysis

procedures must be considered. A study by Wallace and Iwata (1999) considered the influence of session duration on determining function. Forty-six individuals participated in functional analyses based on the model described by Iwata, Dorsey, et al. (1982/1994). Tangible conditions were also run for those individuals whose indirect assessment suggested that access to tangible items might evoke the target behavior. The sessions were videotaped and three sets of data were prepared for each participant, by using the first 5, 10, and 15 min of the sessions. Trained independent raters evaluated data from each video. The results rendered strong agreement between the 15- and 10-min sessions and only three disagreements between 15- and 5-min sessions. As a result, shorter session duration was supported as a means for increasing the practical application of functional analysis methodology (Wallace & Iwata).

Future Directions

The majority of research on functional analysis has been conducted in controlled settings where naturally occurring environmental events are much less likely to influence assessment conditions. One potential result of this structure is that the functional analysis may suggest a relationship that does not exist in the natural environment (Hanley et al., 2003). This phenomenon may compromise the ecological validity of the findings. In addition, most individuals referred for treatment are not admitted directly to inpatient facilities; typically, intervention attempts on an outpatient basis constitute the first stage of treatment (Cooper et al., 1990). By developing a model compatible with an outpatient treatment facility and using parents during a functional analysis, Cooper et al. (1990) was able to identify the functions maintaining different topographies of challenging behavior and develop successful treatment interventions. A growing body of research has demonstrated the use of functional analysis procedures in a variety of treatment settings such as outpatient clinics, schools, and homes (Cooper & Harding, 1993; Cooper et al., 1990; Doggett et al., 2001; Umbreit, 1995).

Doggett et al. (2001) and Umbreit (1995) tested the application of functional analysis methodology in classroom environments. Their studies focused on developing a process in which the conceptual foundations of applied behavior analysis were incorporated with an efficient use of classroom resources. Similar to Cooper et al. (1992) and Cooper and Harding (1993), Doggett et al. and Umbreit stressed the importance of using indirect data collection procedures, as well as descriptive analyses and observations, to aid in the interpretation of functional analysis data.

In the Doggett et al. (2001) study, behavioral consultants assisted general education classroom teachers in conducting an entire functional assessment. The functional analysis component of the assessment was implemented during periods of general classroom instruction. Behavioral consultants trained and supervised the entire assessment procedure, ensuring that the teachers played a primary role in hypothesis development and data analysis. Similarly, in Umbreit (1995), a teacher was supported in the implementation of a functional analysis that proved successful in identifying a function of the student's challenging classroom behavior. The successful implementation of functional analysis in a typical classroom setting further demonstrates the technology's use outside of controlled inpatient clinics (Cooper & Harding, 1993; Doggett et al.; Umbreit).

Functional analysis is considered among the most efficient assessment technologies as it provides a data on demonstrated relationships between environmental events and target behavior. This allows for the development of behavior intervention and teaching plans that are precisely crafted to the needs of the individual. This technology enables investigators to control the many confounding environmental variables that make direct observation-based assessments often very difficult. For example, difficulties discriminating whether escaping a difficult

demand or gaining access to direct teacher attention is the most relevant feature of a student's noncompliant behavior. Using functional analysis, these variables are precisely controlled. The demonstrated relationship between environmental triggers and behavior leads to therapeutic interventions that are functionally driven and evaluable.

See Also

- ▶ Analog Condition Functional Analysis
- ▶ Functional Behavior Assessment

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Functional Analysis Screening Tool

Robert LaRue
Douglass Developmental Disabilities Center, Rutgers, The State University of New Jersey, New Brunswick, NJ, USA

Synonyms

FAST

Description

The FAST is a type of indirect functional assessment method. It is comprised of a self-report checklist designed to identify whether maladaptive behavior is maintained via attention and tangibles, escape, sensory stimulation, or pain attenuation.

Prior to the rating scale section of the instrument, the FAST has two information sections: Informant-Client Relationship and Problem Behavior Information. The Informant-Client Relationship section contains four questions regarding the informant-client relationship (e.g., relationship to the client, how often, and in what situations they interact). The Problem Behavior Information section contains eight questions regarding the topography, frequency, and severity of the target behavior.

The checklist section contains 16 items. Each question is marked as either "yes," "no," or "not applicable." Items endorsed with a "yes" are then categorized into one of the four possible sources of reinforcers for problem behavior: attention/preferred items (social positive reinforcement), escape (social negative reinforcement), sensory stimulation (automatic positive reinforcement), and pain attenuation (automatic negative reinforcement). Behavior function is inferred by calculating a summary score for the four possible maintaining variables. The FAST interview process is 10–15 min in length.

Historical Background

The FAST was originally developed in 1995, as an adjunct to functional analysis procedures. The scale underwent several revisions before most recent version, which was released in 2005. The initial scale included 34 items. After field-testing the FAST with over 300 individuals, the item pool was reduced to the present 16 items.

Psychometric Data

Psychometric data for the FAST are generally sparse. Zaja, Moore, van Ingen, and Rojahn (2011) evaluated and compared the psychometric properties of the Questions About Behavioral Function (QABF) scale (Matson & Vollmer, 1995), the Functional Assessment for Multiple Causality (FACT) scale (Matson et al., 2003), and the FAST for the indirect assessment of self-injurious, stereotypic, and aggressive/destructive behavior. The FAST subscales generally showed the lowest interrater agreement with correlations ranging from poor to good relative to the QABF and FACT. Zaja et al. also found that the FAST had unacceptably low internal consistency especially for the social attention and social escape subscales. The authors indicated that the weaker psychometrics properties (reliability and validity) for the FAST were likely related to its length, limited response format ("yes"/"no"/"NA"), and restricted range of subscales (4 instead of 5).

Clinical Uses

The FAST is an indirect functional assessment procedure. The purpose of the instrument is to provide preliminary information regarding the cause (of function) of maladaptive behavior. The information gathered from the FAST can be incorporated into direct assessment methods. Given the questionable reliability of most indirect assessment measures (such as interviews and rating scales), it is often recommended that these methods be employed in conjunction with direct observation and/or functional analysis procedures and not as the sole means to determining behavioral function (Zarcone, Rodgers, Iwata, Rourke, & Dorsey, 1991).

See Also

► Functional Behavior Assessment

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Functional Assessment

► Functional Behavior Assessment

Functional Assessment and Curriculum for Teaching Everyday Routines

John Molteni
Institute for Autism and Behavioral Studies, University of Saint Joseph, West Hartford, CT, USA

Definition

The Functional Assessment and Curriculum for Teaching Everyday Routines (FACTER) is a criterion-referenced assessment and curriculum for teaching daily routines and activities of daily living. It is designed to address the needs of individuals with developmental disabilities. FACTER provides task-analyzed skills that cover a variety of skills needed to navigate an individual's daily environment. There are two versions of FACTER, an elementary level and secondary level. Both consist of formative and summative assessments of student learning and provide a framework for developing and tracking instruction across routines.

Historical Background

The history of FACTER is outlined in the Program Manual (Arick, Nave, Hoffman, & Krug, 2000). FACTER was developed to meet the requirements of the Individuals with Disabilities Education Act Amendments of 1997. The first iteration, named the Extended Career and Life Role Assessment System (Arick, Nave, & Hoffman, 2000), was designed to assess individuals who were not participating in statewide or district assessments and meet the state standards for career-related learning. There were several features of the Extended CLRAS that allowed the state of Oregon to meet IDEA mandates for reporting assessment results.

The first version of FACTER was an extension of the Extended CLRAS that added the instructional component. Individualized lesson plans were added to assist educators in developing instructional programs that complemented the

assessments conducted for program planning and assessing progress on intervention goals and objectives. Progress monitoring was added to support ongoing instructional development and modification and track the effectiveness of interventions. Skills targeted for assessment and instruction were derived from special education standards and areas of functioning referenced in the literature and clinical practices as important for independent living.

Rationale or Underlying Theory

The primary rationale for FACTER is the development of skills that will maximize independent skill development, a primary goal of special education. Assessment of skills and instructional practices are developed to maximize student independence and allow for a complete assessment to goal development and instructional planning to implementation of instruction. The underlying instructional theory is that of direct instruction utilizing task analytic approaches to instruction. Additional strategies utilized during instruction include visual supports, tactile systems, and social stories. Preteaching of routines and instruction during the routine are implemented during instruction. Criterion-referenced assessment is a critical feature of this program with skills that are broken down into smaller component skills that allow for analysis and progression through the sequence of behaviors necessary to independently perform the skill.

Goals and Objectives

The goals and objectives of FACTER are related to the instruction of routines and underlying skills related to those routines to enhance a student's independent living skills. Individualized goals and objectives are developed based on results obtained in the FACTER assessment phase. Subsequently, lesson plans based on these objectives are developed and implemented utilizing direct instructional approaches. Ongoing data collection is utilized to track individual's progress as well as report the results of larger groups of students. Individual student tracking allows for adjustment of instructional programming in response to student performance. Summative assessment utilizing criterion-referenced assessment allows for reporting on Individualized Educational Plans and for statewide and district-wide assessments.

Treatment Participants

FACTER is designed for individuals who require special education services with a particular focus on individuals in need of life skills training and those that are not participating in statewide or district-wide assessments. A particular focus is on developing skills that maximize student independence and align with state educational standards for post-secondary skills that support career, independent living, and involvement within society as a responsible citizen.

Treatment Procedures

There are three components outlined in the FACTER manual related to treatment procedures. These are:

Assessment Phase

This phase consists of utilizing the Program Manual of FACTER to assess the individual's ability across a variety of domains. These include Living Skills, Transition, Academic, Leisure, and Community at the elementary level with the addition of Career as a domain in the secondary level. During the assessment phase, the educator obtains baseline ratings of routines on a 5-point scale from 0 (does not complete with physical assistance) to 4 (completes independently). An N can be used to indicate Not Applicable which may be due to physical or medical limitations that prevent the student from engaging in the routine or indicates that the routine is deemed inappropriate by the educational team or is not supported by the school environment. Baseline ratings are completed by individuals who are familiar with the student being assessed.

Following the baseline ratings, routines are selected for performance assessment. Corresponding skills related to completion of the routine are also selected. Skill areas include expressive communication, receptive communication, problem solving, teamwork/social skills, motor skills, and functional academics. These are subsequently broken down from basic to complex. All selections in this step are made on the basis of IEP goals and objectives developed by the educational team. The criterion for selection is that if completed, these skills will lead to increased independence by the student. Data are collected via direct observation on these routines and skills and are charted in the student booklets included with the Program Manual.

Instruction Phase

This phase includes a variety of instructional strategies that can be utilized by educators to instruct students on independence of routines selected in the assessment phase. Routines are typically taught in inclusive settings, with transition routines integrated between routines from other domains. That is, when a routine in the classroom involves an academic task, then a transition to group instruction, the educator strings these three routines (academic, transition, social) together. Taken as a whole, increasing independence on these routines allows the student to be better able to navigate a typical environment with as little assistance as possible. This includes strategies of prompting and prompt fading to increase independent functioning. Instruction before the routine is executed (pre-routine instruction) and instruction within the routine follows. Location of this instruction is relevant to the individual's ability to execute the skills in the natural environment.

Steps described by the authors include prioritizing instructional objectives, providing instruction and assessing progress, and conducting performance assessment of routines following instruction. During the first step, educators choose teaching strategies to utilize with the student with specific setup, and teaching suggestions are outlined in the Program Manual. Instructional strategies are grouped into categories such as creating cues, creating pictures, fine and gross motor skills practice, modeling, picture storybook, role playing, social stories, tactile systems, and visual systems. Ongoing assessment occurs with direct observation data collection which indicates what strategies were used and what outcomes were achieved by the student. Assessments are conducted after several teaching sessions in a probe format. The final step involves a performance assessment of the student's performance on the routine. This is the fourth assessment of the routines following instruction and ongoing performance assessment.

Evaluation Phase

This phase involves determining whether to continue instruction or return to the assessment phase. The Program Manual provides guidelines for determining which decision to make. Continuing with the instruction phase is warranted if the student has not met the criteria of mastery in the instructional phase. There are several conditions that can be used to terminate current instruction. These involve mastery of the routines and meeting the instructional goals of the student's individualized education plan that may not be at the level of independent functioning. That is, if a student has progressed by way of decreased dependence on teacher support, this may meet the goals set forth by the educational team. Finally, instruction may be discontinued in several instances, including student's lack of progress and environmental limitations that do not support ongoing instruction.

Efficacy Information

There are no empirical studies to date that outline the efficacy of FACTER in educational settings, although the Program Manual references some unpublished research. The Program Manual does provide data that was collected during field testing of FACTER in the state of Oregon through the Department of Education. Interrater reliability and test-retest reliability were reported with high correlation coefficients reported for elementary and secondary levels. These were somewhat

lower for secondary scores and ratings between teachers and assessment team raters.

Concurrent validity was assessed with the Vineland Adaptive Behavior Scales – Interview Edition and correlations between teacher ratings on FACTER and actual student performance. Correlation coefficients in both cases were adequate.

Outcome Measurement

Outcomes on FACTER are assessed for each individual on the routines that are the target of instruction. Assessments indicate level of independent performance. Please see the "Treatment Procedures" section for additional information on the assessment and evaluation procedures of FACTER.

Qualifications of Treatment Providers

FACTER is intended for use by special educators in a school setting, although no specific recommendations are made for individuals who may administer the FACTER or implement the teaching procedures. While the authors provide the necessary tools to utilize the curriculum, they do offer training and technical assistance to users.

See Also

- ▶ Criterion-Referenced Assessment
- ▶ Prompts
- ▶ Social Stories
- ▶ Task Analysis
- ▶ Visual Supports

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Functional Assessment Screening Tool (FAST)

Robert LaRue
Douglass Developmental Disabilities Center, Rutgers, The State University of New Jersey, New Brunswick, NJ, USA

Synonyms

FAST

Description

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See Also

▶ Functional Behavior Assessment

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Functional Behavior Assessment

Mark Palmieri
Feeding Clinic, Center for Children with Special Needs, Glastonbury, CT, USA

Synonyms

FBA; Functional assessment

Definition

Functional behavior assessment (FBA) procedures are used to investigate the maintaining variables associated with a target response and identify adaptive responses to be taught in order to allow the individual to better meet his or her needs. There are a variety of assessment procedures associated with the completion of a comprehensive FBA. These may be categorized into indirect and direct measures. Indirect procedures will capture archival information about the patient and allow those who are familiar with the individual and the target response to offer reports. Completion of indirect interviews and questionnaires allows investigators to capture information from many sources in a generally time-efficient fashion. The information gathered through indirect methods enables investigators to develop a comprehensive conceptualization of the target behavior for the assessment, as well as to capture information relevant to understanding the patient's needs, life context, learning history, motivators, and interpersonal support system. This information allows investigators to gain insight into common environmental variables associated with the target behavior, specifically including both high- and low-probability contexts. Indirect data collection tools will address environmental variables such as locations, times of day, individuals present, engagement, demand levels, access to preferred materials, and characteristics (e.g., sensory variables) of different environments. Direct data collection is a required feature of any FBA. The observations must be guided by an operational definition of the target response for the assessment, and all observers must be trained to levels of acceptable reliability on the target. Direct data should be captured across multiple contexts in order to capture a sufficiently comprehensive data set for analysis. This typically includes observations across multiple days, times of day, and settings (e.g., high- and low-demand environments and high-, low-, and divided-attention environments). Antecedent behavior consequence (ABC) data are among the most commonly collected direct observation data when completing an FBA. These data require the observer to record information on the general environmental context and the exact changes that occurred immediately before and after the occurrence of the target response. The data are analyzed in order to determine common classifications of antecedent and consequence events associated with the target behavior. This information is then reviewed with all other sources of information collected during the assessment in order to develop a functional hypothesis of the maintaining variables for the challenging behavior and to map replacement skills to teach during intervention phases.

See Also

- ▶ Analog Condition Functional Analysis
- ▶ Functional Analysis

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Functional Communication Training

Patricia Prelock
Communication Sciences & Disorders, Dean's Office, College of Nursing & Health Sciences, University of Vermont, Burlington, VT, USA

Definition

Functional communication training (FCT) is a systematic approach to intervention in which a more socially appropriate behavior is taught to replace a challenging behavior (Durand, 1990; Durand & Merges, 2008, 2009; Prelock, Paul, & Allen, 2011). It is presumed that the challenging behavior is an attempt to communicate; therefore, vocalizations, manual signs and gestures, or graphic symbols are used to replace the less desirable behaviors. Notably, FCT is used in combination with other interventions (Durand, 1999; Olive, Lang, & Davis, 2008).

Historical Background

Challenging behavior is a frequent occurrence for individuals with ASD, particularly when they do not possess the speech and language skills required to communicate their wants and needs. These behaviors may include self-injury, tantrums, aggression, etc. Research suggests that individuals with disabilities, and particularly those with ASD, are more likely to exhibit problem behaviors that are frequent, severe, and sustained over time. These behaviors interfere with family life and make it difficult for individuals with ASD to be integrated in their community. Further, such behavior problems are a strong predictor of school failure (Feldman et al., 2007). Therefore, behavioral strategies have been established to address these behavior problems. Durand, Carr, Dunlap, and colleagues recognized that these challenging behaviors are often a result of an inability to communicate intent including an individual's wants, needs, and desires (Carr & Durand, 1985; Dunlap, Kern-Dunlap, Clarke, & Robbins, 1991; Durand, 1990; Durand & Merges, 2008, 2009). FCT was designed as an intervention technique to address the reported challenging behaviors in individuals with ASD and other developmental disabilities by replacing the undesired behaviors with an alternative that is based on the perceived and assessed intent of the behavior. A variety of professionals (e.g., psychologists, speech-language pathologists, behavioral interventionists) and families use FCT across age, language skills, and developmental abilities in several contexts (Dunlap, Ester, Langhans, & Fox, 2006; Durand, 1999; Mancil, 2006; Petscher, Rey, & Bailey, 2009). FCT has strong support in the literature as an established skill-based intervention (Smith et al., 2007).

Rationale or Underlying Theory

There are several concepts that provide a theoretical framework for understanding why FCT is an effective intervention for reducing challenging behavior. Three concepts have been identified as crucial to understanding FCT and implementing it with a diverse population: functional equivalence, natural communities of reinforcement, and features of the alternative response (Carr, 1988; Durand, 1987, 1990; Durand & Merges, 2009).

Functional equivalence is used to explain how and why behavior changes in FCT. Assumptions are made about why a behavior occurs such as being reinforced by getting the attention of another or escaping from doing something. If this is true, then these challenging behaviors

could be adjusted by offering an alternative or new behavior that achieves the same function. Thus, FCT reduces problem behavior because it involves teaching and reinforcing a replacement behavior that serves the same function.

Using a communication strategy as a replacement for challenging behavior has an added advantage of being able to capitalize on "natural communities of reinforcement" (Durand, 1990). This means an individual learns to ask for a reinforcer from a variety of communication partners, and those partners do not require specific training from an interventionist.

A third component underlying the conceptual framework of FCT that is important to the success of this intervention is the opportunity for creating an alternative response (Durand & Merges, 2009). An alternative response is one that is functionally equivalent to the challenging behavior and includes several features like how closely it matches the function of the behavior, how successful and efficient it is likely to be, how recognizable and acceptable it is as a response, and how it works in the natural environment.

An important principle in understanding the approach and value of FCT is that behavior problems are not viewed as undesirable occurrences that must be eliminated. Instead, challenging behaviors are viewed as functionally equivalent to other modes of communication. This view recognizes that although the behaviors may be unacceptable, what is being communicated is not (e.g., getting attention, asking for help). This is a critical concept because the intervention goal changes. Instead of eliminating the behavior and providing no alternative for accessing what an individual needs or wants, an alternative and more acceptable communication behavior is taught so the desired reinforcer can be achieved.

Goals and Objectives

FCT is primarily designed to address a range of challenging behaviors that interfere with an individual's ability to fully participate in learning, and family and community life. Appropriate targets may include self-injury, aggression, tantruming, stereotypies, and inappropriate sexual behavior, to name a few (Fyffe, Kahng, Fittro, & Russell, 2004).

Treatment Participants

FCT can be implemented for anyone who displays problematic behavior. It is an appropriate strategy to address the behavior challenges reported for individuals with ASD as well as other disorders (e.g., ADHD, schizophrenia, traumatic brain injury) in which behavior problems are a frequent occurrence (Durand & Merges, 2009). Research also suggests that FCT can be used to address the difficult behaviors of individuals of all ages with a range of language and cognitive abilities (Bird, Dores, Moniz, & Robinson, 1989; Mancil, 2006; Petscher et al., 2009).

Treatment Procedures

FCT requires the implementation of three primary steps: (1) assessing the function of the challenging behavior to be addressed, (2) selecting an appropriate alternative to the challenging behaviors, and (3) teaching the alternative response and fading prompts to use the alternative response in the environment.

To begin FCT, the interventionist identifies the antecedents (what comes before) and consequences (what follows) of a problem behavior in an effort to assess the function the behavior serves. When the function is determined, individuals are taught to make a request in a more acceptable manner. Selecting an alternative communication modality to make this request is a critical next step. Consideration is given to those modes of communication the individual has already demonstrated some skill (e.g., signing, graphic symbols, writing, verbalization).

Once an alternative mode of communication is identified, teaching situations are created. First, the environment is arranged to provide multiple opportunities for using the new communication strategy. Situations are established that are most

likely to elicit the interest of the individual. Training opportunities are interspersed through the day in the environment where the individual is expected to communicate. This approach increases the likelihood of intervention effects that are generalized and maintained.

Several language training techniques are required to teach individuals to communicate their intent using an acceptable means as a replacement for their challenging behavior (Durand, Mapstone, & Youngblade, 1999). Prompts are used to teach the new response and then are faded as quickly as possible. Prompts may be full physical prompts or partial physical, gestural, or verbal prompts. Delayed prompting is also used to help fade the prompting and encourage spontaneous use of the desired communication behavior (Halle, Baer, & Spradlin, 1981; Schwartz, Anderson, & Halle, 1989). Once an individual demonstrates success in using the new communicative behavior in three of five consecutive opportunities, prompt fading begins to eliminate prompt dependency.

The context for training occurs on a continuum from more artificial settings like a therapy room to more natural settings like the community. The context is largely dependent on where the individual is expected to use the new trained communication response. If training begins in a more restricted setting like a therapy room, once the response is learned, performance should be encouraged in the settings where the behavior is expected to occur to ensure generalization and maintenance.

Efficacy Information

There are more than 200 studies documenting the effectiveness of FCT for addressing challenging behaviors for a range of individuals (Durand & Merges, 2008, 2009; Mancil, 2006; Mirenda, 1997; Petscher et al., 2009). Research also suggests that FCT successfully decreases problematic behavior and increases communication in individuals with ASD. Some of the communicative responses that have been successfully taught as a replacement to difficult behaviors include mands (Brown et al., 2000; O'Neill & Sweetland-Baker, 2001) and augmentative and alternative communication (AAC) strategies such as signing, communication books, and voice output devices (Mirenda, 1997). The use of these replacement behaviors has led to increased spontaneous and appropriate verbal communication (Mancil, 2006) that is maintained and generalized to untrained activities (Olive et al., 2008; Wacker et al., 2005).

Petscher et al. (2009) reviewed over 80 studies describing FCT as a treatment to address problematic behaviors and found it met their preestablished criteria for a well-established intervention. This supports the findings of the National Standards Project (2009) which also identified FCT as an established behavioral intervention that has successfully decreased problem behaviors and restricted repetitive behaviors, and increased academic, communication, and social behaviors in individuals with autistic disorder and pervasive developmental disorder-not otherwise specified ranging in age from very young to 21 years.

Although there is extensive research examining the effectiveness of FCT, most studies use single subject designs with small numbers of participants. Some larger studies do exist (e.g., Durand & Carr, 1992; Wacker et al., 1998), but no randomized clinical trials have been reported that compare treatments or outcomes to no treatment.

Durand and Rost (2005) suggest that there is some question about the representativeness of the literature examining behavioral interventions including FCT. They suggest it is unclear if the current intervention research systematically excludes certain individuals which would influence the results and applications to specific populations.

Outcome Measurement

Functional behavioral assessment (FBA) is a key element in the implementation of FCT (Durand, 1990). FBA is used to determine the function of problematic behavior that eventually leads to

selecting a more appropriate alternative communication behavior. The selection of the alternative behavior must serve the same function as the challenging behavior.

Informal observations, antecedent-behavior-consequence (ABC) charts, functional analyses, and a variety of rating scales are frequently used to determine the function of a behavior. Functional analysis is often identified as the best method to determine the function of a behavior. It is a process by which aspects of the environment are manipulated to assess behavior change (Hanley, Iwata, & McCord, 2003), although there are challenges in gaining accessibility to manipulate the environment. There are also some influences that may not be conducive to manipulation.

Assessment typically begins with interviews of those who know the individual best as well as informal observations. Once this information is gathered, more formal assessments are completed to validate the impressions made from the informal assessment. Available instruments include the *Motivation Assessment Scale*, the *Motivation Analysis Rating Scale*, the *Functional Analysis Interview Form*, the *Functional Assessment Checklist*, the *Functional Analysis Checklist*, and the *Questions About Behavioral Function*.

Data collection requires establishing goals, analyzing behavior patterns, assessing environmental obstacles, and monitoring results (Durand & Hieneman, 2008a, 2008b). To establish goals, there needs to be sufficient information to define the problem and determine the desired responses across settings. Different forms of functional assessment are then employed to analyze behavior patterns. The learning environment is then examined to identify what alternative communicative responses could be supported. Those responsible for collecting data are then identified and trained to tally responses (Durand & Hieneman, 2008a, 2008b).

Qualifications of Treatment Providers

Individuals who implement FCT are usually highly trained in behavioral strategies and recognize the role of behavior in communication. FCT is best implemented using a team approach in collaboration with a psychologist who has experience with functional behavior assessment and a speech-language pathologist who can help to identify the function and appropriate replacements for expressing communicative intent considering an individual's cognitive and communication abilities. Children with ASD often require a behavioral plan, so once this is developed, training needs to occur for all those who come into contact with the individual who is exhibiting challenging behavior. Adequate training in behavioral principles is necessary to ensure that regression does not occur and behavior problems don't increase (Durand & Carr, 1991, 1992).

See Also

- ▶ Applied Behavior Analysis
- ▶ Communication Assessment
- ▶ Communication Interventions
- ▶ Functional Analysis
- ▶ Functional Behavior Assessment
- ▶ Functional Goals

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Functional Connectivity

Ralph-Axel Müller
SDSU Department of Psychology, San Diego State University, San Diego, CA, USA

Definition

Functional connectivity (FC) has been defined as "temporal correlations between spatially remote neurophysiological events" (Friston, Frith, & Frackowiak, 1993), a definition that is considered authoritative even today. The reference to correlation highlights the distinction from the concept of *effective connectivity*, which invokes causality and has been defined as "the influence one neural system exerts over another" (Friston et al., 1993). Correlations can exist on widely differing time scales, specifically high frequencies in the gamma band (>25 Hz), as detected in electroencephalography (EEG) and magnetoencephalography (MEG), and low frequencies (<0.1 Hz) in functional connectivity MRI (fcMRI). A more encompassing recent definition of FC refers to "the statistical dependence between remote neural processes" (Honey, Kotter, Breakspear, & Sporns, 2007).

Historical Background

In the first FC study of ASD, Horwitz et al. (1988) used positron emission tomography (PET) to examine the correlations of glucose metabolic rates between numerous brain regions of interest (ROIs). Correlations were reduced in young men with ASD for ROI pairings within and between frontal, parietal, subcortical, and cerebellar ROIs. This early interest in connectivity was stimulated by later findings of atypical white matter growth patterns in children with ASD (Courchesne et al., 2001), which may suggest early-onset axonal anomalies. It is also consistent with a view of ASD as a disorder of functional networks, rather than one or a few local sites (Belmonte et al., 2004; Müller, 2007; Rippon, Brock, Brown, & Boucher, 2007).

Current Knowledge

Underconnectivity Theory

Recent interest in FC was prompted by a study by Just et al. (2004), who reported that correlations of the blood oxygen level–dependent (BOLD) fMRI signal during sentence comprehension were reduced between multiple ROIs in participants with ASD (compared to a typically developing [TD] control group matched for age, but not handedness). The "underconnectivity theory," as proposed by these authors, has found support in a large number of subsequent studies that examined FC for finger movement (Mostofsky et al., 2009), visuomotor coordination (Villalobos, Mizuno, Dahl, Kemmotsu, & Müller, 2005), face processing (Kleinhans et al., 2008), verbal working memory (Koshino et al., 2005), sentence comprehension (Kana, Keller, Cherkassky, Minshew, & Just, 2006), response inhibition (Kana, Keller, Minshew, & Just, 2007; Lee et al., 2009), embedded figures (Damarla et al., 2010), problem solving (Just, Cherkassky, Keller, Kana, & Minshew, 2007), cognitive control (Solomon et al., 2009), self-representation (Lombardo et al., 2010), and tasks tapping into theory of mind (Kana, Keller, Cherkassky, Minshew, & Just, 2009; Mason, Williams, Kana, Minshew, & Just, 2008). General underconnectivity has in fact been presented as a potential "first firm finding" in ASD (Hughes, 2007). The evidence is further strengthened by associations between reduced FC and other markers of neurological, cognitive, or diagnostic abnormality in ASD. For example, two studies observed a correlation between frontoparietal FC and size of the callosal genu in adults with ASD that was not seen in control participants (Just et al., 2007; Kana et al., 2006). Kleinhans et al. (2008) found that reduced FC between fusiform face area and amygdala was associated with symptom severity on the ADOS Social score (Lord, Rutter, DiLavore, & Risi, 1999) in adults with ASD. Association between reduced frontoparietal connectivity and attention deficit was observed by Solomon et al. (2009) in adolescents with ASD. One recent study (Dinstein et al., 2011) found reduced interhemispheric

synchronization of activity in language-related brain regions (inferior frontal and superior temporal gyri) in toddlers with ASD scanned during natural sleep. Inferior frontal synchronization was correlated with an expressive language score.

Partial Overconnectivity

Often overlooked is the fact that in the original study by Just et al. (2004), reduced correlations were detected in only 10 out of 186 ROI pairs. In addition, a number of studies have reported results that appear inconsistent with the underconnectivity theory. Welchew et al. (2005) examined BOLD correlations for a large matrix of 90 cortical and subcortical ROIs. Although they focused on reduced connectivity in the medial temporal lobe in adults with high-functioning autism or Asperger's syndrome, they also detected many ROI pairs that showed greater FC in ASD compared to a TD control group. Subsequent studies presented findings on FC of thalamus (Mizuno, Villalobos, Davies, Dahl, & Müller, 2006) and caudate nucleus (Turner, Frost, Linsenbardt, McIlroy, & Müller, 2006) that suggested diffusely increased FC in ASD, also inconsistent with the underconnectivity theory. One of these (Mizuno et al., 2006) and a further study on cortico-cortical connectivity of networks related to source memory (Noonan, Haist, & Müller, 2009) suggested that effects of apparent "overconnectivity" in ASD were characterized by widespread low-threshold correlations ("noisy connectivity") not seen in control groups. A few other recent studies observed overconnectivity or mixed findings (underconnectivity between some regions, overconnectivity between others) in adults and adolescents with ASD related to imitation (Shih et al., 2010), emotional processing (Ebisch et al., 2011; Wicker et al., 2008), and the default mode network (Monk et al., 2009). One further study (Shih et al., 2011) found that overconnectivity of posterior superior temporal sulcus, which plays a role in biological motion perception, face processing, joint attention, auditory-visual integration, and language perception (Redcay, 2008), was associated with reduced functional differentiation within this region in children and adolescents with ASD.

Resting State and the Default Mode Network

The study by Monk and colleagues (2009) mentioned above belongs to a growing literature on FC associated with task-free resting states in ASD. Cherkassky et al. (2006) reanalyzed resting blocks (fixation cross only) from several task-activation studies in 12 ROIs attributed to the default mode network (DMN) and found underconnectivity for an overwhelming majority of ROI pairs in young adults with ASD. Kennedy and Courchesne (2008) acquired continuous resting state fMRI data for 7 min and found that averaged correlation maps for three seeds considered nodes of a task-negative network (equivalent to the DMN) were strongly reduced in adolescents and adults with ASD, overall consistent with the findings by Cherkassky et al. On the other hand, FC within a task-positive network (regions that frequently activate across different task domains) was similar between ASD and TD groups, and differences in effects also included some connectivity clusters seen only in the ASD, but not in the TD group. Monk et al. (2009), using only a single seed in posterior cingulate cortex (PCC), found more mixed results of partial under- and overconnectivity within the DMN in adults with ASD. These authors also found that impaired social functioning (from the ADI-R; Rutter, Lord, & LeCouteur, 1995) was associated with reduced connectivity between PCC and medial prefrontal regions and that restricted and repetitive behaviors from the same measure were positively correlated with connectivity between PCC and right parahippocampal gyrus. In a subsequent study, the same group (Wiggins et al., 2011) found that posterior regions of the DMN, as detected in data-driven self-organizing maps for each individual participant, had reduced FC with two right-hemispheric inferior parietal and superior frontal sites. Another study examining default mode networks derived from independent component analysis identified several sites in frontal and parietal lobes where reduced connectivity was associated with symptom severity

in adolescents and young adults with ASD (Assaf et al., 2011). Resting state data have also been applied to questions other than the DMN. Anderson et al. (2011) investigated the BOLD signal correlation between each brain voxel and the homotopic voxel in the contralateral hemisphere and found interhemispheric underconnectivity in adolescents and young adults with ASD in several perisylvian and parietal regions. Ebisch et al. (2011) used the insula, considered a crucial area for "emotional awareness," as fcMRI seed and reported several sites with reduced connectivity, such as amygdala and PCC.

Methodological Issues

While a clear majority of fcMRI studies in ASD have reported findings in support of the underconnectivity theory, relatively little attention has been paid to methodological questions in the context of partially inconsistent results. Thai et al. (2009) raised methodological concerns regarding the potential impact of task factors and increased variability in ASD cohorts. In a comparative methodological fcMRI study, Jones et al. (2010) indeed showed that underconnectivity effects in ASD (compared to a matched TD group) almost completely disappeared when modeled task effects were regressed out (see below). This study further suggested that inverse findings of overconnectivity in ASD might be related to global signal regression, which relates to a recent discussion in the fcMRI methods literature about the treatment of global signal fluctuations (i.e., changes in signal intensity seen across the whole brain). While it is generally accepted that some of these BOLD fluctuations may be noise, it has been argued that their removal through global signal regression results (by mathematical necessity) in potentially spurious anti-correlations (Murphy, Birn, Handwerker, Jones, & Bandettini, 2009). It is further likely that some components in the global signal may actually reflect true neuronal fluctuations (Fox, Zhang, Snyder, & Raichle, 2009; Schölvinck, Maier, Ye, Duyn, & Leopold, 2010).

A recent survey of 32 fcMRI studies in ASD published by late 2010 (Müller et al., 2011), however, showed that global signal regression alone is unlikely to explain differences in FC findings on ASD described above. This survey suggested that studies were most likely to generate underconnectivity findings in ASD when task effects were not regressed out, data were not low-pass filtered, and results were considered only for task-related ROIs (rather than the whole brain). Removal of task effects and low-pass filtering is related to the concept of *intrinsic connectivity* (Van Dijk et al., 2010), which refers to low-frequency (<0.1 Hz) fluctuations in the BOLD signal that may reflect fluctuations in local field potentials (Leopold, Murayama, & Logothetis, 2003; Schölvinck et al., 2010). These fluctuations are spontaneous (i.e., independent of online cognitive, task-related processing) and have been related to Hebbian plasticity and the sculpting of neuronal connections based on long-term experience (Lewis, Baldassarre, Committeri, Romani, & Corbetta, 2009; Sadaghiani, Hesselmann, Friston, & Kleinschmidt, 2010). It is remarkable that many ASD studies that were fully adapted to isolating such intrinsic connectivity through task regression and low-pass filtering yielded evidence incompatible with the underconnectivity theory, whereas many studies in support of underconnectivity examined task-activation effects or a mixture of such effects and intrinsic fluctuations (for details, see Müller et al., 2011).

Electrophysiological Studies

Although EEG and MEG can detect signal correlations in a much wider frequency range than fcMRI due to their superior temporal resolution, the current EEG and MEG literature on FC in ASD remains surprisingly small. Several studies have examined the temporal coherence of EEG or MEG signals. Coherence in the gamma band is considered to reflect binding during perceptual processing across brain regions (Nase, Singer, Monyer, & Engel, 2003; Roelfsema, Engel, König, & Singer, 1997), presumably based on axonal connectivity. Several studies in children and adults with ASD suggest abnormalities in gamma band activity related to face perception (Grice et al., 2001), viewing of illusory shapes (Brown, Gruber, Boucher, Rippon, & Brock, 2005), and auditory perception of clicks

(Wilson, Rojas, Reite, Teale, & Rogers, 2007), arguably reflecting impaired perceptual binding. Interhemispheric coherence of visual evoked potentials was reduced in a small sample of children with ASD (Isler, Martien, Grieve, Stark, & Herbert, 2010), but this effect was found only in theta and lower bands (<8 Hz), not in the gamma band.

However, not all studies have reported results consistent with underconnectivity. One EEG study (Orekhova et al., 2007) found atypically increased spectral power in the gamma band for task-free visual conditions, which was correlated with developmental delay in boys with ASD aged 3–8 years. Murias et al. (2007) examined EEG coherence during rest, with complex findings grossly suggesting decreased long-distance and increased short-distance connectivity (especially in temporal lobes) in adults with ASD for frequency bands between 3 and 17 Hz (see also Barttfeld et al., 2011, for partially consistent results). Conversely, an EEG study of adults with ASD during REM sleep reported reduced short-distance right frontal coherence in the theta band, but increased long-distance left fronto-occipital coherence in the delta band (Léveillé et al., 2010).

A few studies have used alternative approaches to investigate FC in ASD. Pollonini et al. (2010) used Granger causality and graph theory to analyze resting state MEG data from a small sample of young adults with ASD, reporting an atypically increased characteristic path length, which refers to the number of edges [grossly equivalent to causal links] that must be traversed to go from one node [grossly equivalent to a brain region] to another (Bullmore & Sporns, 2009). An additional finding was a reduced clustering coefficient in ASD, that is, a reduction in tightly interconnected local clusters of nodes. Both of these findings were replicated in a resting state EEG study using graph theory by Barttfeld et al. (2011). Overall, these findings suggest that a typical "small-world" architecture, in which efficient connectivity is achieved by strong local clustering of connections balanced with long-distance connections between hubs (Bullmore & Sporns, 2009), may be impaired in ASD (Barttfeld et al., 2011).

In summary, the electrophysiological literature tentatively suggests impaired FC in ASD as well as general aberrations of the typical modular "small world" organization of networks.

Future Directions

Whereas the EEG and MEG literature remains limited, the fcMRI literature in ASD has grown rapidly in recent years. However, the quantity of fcMRI findings has not always been matched by the quality of methods. Sharpened methodological awareness will be crucial for a better interpretation of fcMRI findings in ASD. An exemplary issue concerns head motion. The vast majority of fcMRI studies in ASD did not test for group differences in head motion, although it is known that head motion is likely to affect the interregional correlation of the BOLD signal (Auer, 2008). As indicated earlier, several other methodological choices may crucially affect the pattern of connectivity disturbances observed in ASD that may be grossly characterized as general underconnectivity versus partial over-connectivity (Müller et al., 2011). FCMRI studies directly testing the impact of differing processing pipelines will contribute to a more precise understanding of FC in ASD. At present, a tentative interpretation of the literature would suggest that BOLD correlations tend to be reduced in ASD when task-activated data (or data from uncontrolled cognition during resting states) are examined for ROIs identified based on expected or known activation in the TD population. On the other hand, BOLD correlations are often found to be atypically increased outside such domain-specific networks, especially if time series are low-pass filtered and effects of task are removed in order to isolate intrinsic low-frequency BOLD fluctuations that are thought to best reflect functional network organization (Cordes et al., 2001; Fox & Raichle, 2007).

Assuming that such an interpretation holds generally true in light of future methodologically more rigorous studies, results from both types of fcMRI studies may actually reflect interesting and complementary aspects of network dysfunction in ASD. The emergence of network

connectivity can be understood with respect to the interplay between experience-driven constructive and regressive processes (Johnson, 2011; Kandel, Jessell, & Sanes, 2000). Within-network underconnectivity, observed in many task-related studies, may reflect disturbances in the constructive processes of synaptic stabilization and axonal myelination. On the other hand, out-of-network diffuse overconnectivity, as seen in some intrinsic fcMRI studies, may reflect disturbances of synaptic pruning due to diminished interactive experience related to the autistic condition itself, or traces of early white matter overgrowth in ASD (Courchesne et al., 2001). Forthcoming diffusion tensor imaging (DTI) studies better characterizing the axonal organization associated with early overgrowth in infants and toddlers will be instrumental in answering this latter question (for first findings see Sundaram et al., 2008; Weinstein et al., 2011).

Aside from improved awareness of methodological implications in future fcMRI studies, other techniques will be instrumental for a comprehensive picture. As mentioned, more electrophysiological studies with larger sample sizes will be important for examining FC in higher frequency bands. In addition, DTI advances may allow investigations to expand beyond the gross impairment of large fiber tracts, which has been well documented for ASD participants above 8 years of age (Alexander et al., 2007; Fletcher et al., 2010; Shukla, Keehn, & Müller, 2011), toward the detection of potential anatomical overconnectivity that may, for example, be reflected in increased presence of crossing fibers (cf. Lange et al., 2010). Multimodal approaches (e.g., combining fcMRI and DTI with EEG or MEG) promise to be more powerful than single-technique studies that have been common in the literature.

See Also

- ▶ Diffusion Tensor Magnetic Resonance Imaging
- ▶ Electroencephalography
- ▶ Functional MRI

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Functional Ecological Approach

Robert LaRue
Douglass Developmental Disabilities Center, Rutgers, The State University of New Jersey, New Brunswick, NJ, USA

Definition

The functional ecological approach is an approach to the assessment and intervention process that emphasizes environmental variables that may contribute to target behavior (Powers, 2005, 1997). The model stands in contrast to earlier conceptualizations of behavioral assessment focusing on the topographical aspects of behavior (i.e., what the behavior looks like) rather than focusing on the underlying function (or cause) maintaining behavior. In contrast, the functional ecological emphasizes the need to evaluate the individual's behavior in a broad developmental context, accounting for relevant antecedent and consequent conditions affecting behavior.

The process emphasizes the context in which behavior occurs, thus increasing the likelihood that the assessment process, intervention strategies, and outcomes lead to benefits across environments. The approach leads to (1) the modification of the environment in which the target behavior occurs, (2) the manipulation of the controlling contingencies fort target behavior, (3) the development of functional equivalent replacement skills, and (4) the development of functional and socially valid skill repertoires. These gains exert their effects in multiple settings and are maintained in the natural environment.

See Also

► Functional Behavior Assessment

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Functional Goals

Shaunessy Egan
The Center for Children with Special Needs, Glastonbury, CT, USA

Definition

A functional goal outlines a target skill to be acquired in measurable terms, while including a precise behavior to be accomplished and a specific criterion. It identifies the behavior or skill caregivers/instructors want the individual to

learn or accomplish, the context in which the skill will be taught, and a quantifiable level of mastery. Functional goals should be developed so that they are implemented and practiced within the individual's daily routine.

Functional goals include three components: an acquisition statement, a normalized context, and a criterion. An acquisition statement describes the target behavior in terms that are observable and measurable. A normalized context identifies the activities or daily routines in which the behavior is expected. A criterion states the desired level of the behavior for mastery of the skill.

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Functional Life Skills

Dawn Hendricks
Department of Special Education and Disability Policy, VCU Autism Center for Excellence, Virginia Commonwealth University, Richmond, VA, USA

Definition

Functional life skills are the variety of skills which are frequently demanded in natural domestic, vocational, and community environments. The skills involve those immediately applicable to daily life or may also include those which teach students to participate in future environments (Brown et al., 1979). Key qualities of functional skills include the following:

- It is performed within the context of a real activity.
- The activity is meaningful to the student.
- People without disabilities believe the activity serves a purpose.
- If the student is unable to perform the skill himself or herself, it would need to be completed by another person.
- The skill will be needed throughout the person's life.

Historical Background

The philosophy behind teaching functional life skills is full inclusion in all activities of family and community life, making adaptations where necessary. There is one absolute purpose of education for all students, including those with autism spectrum disorder (ASD): Preparation for adulthood. The Individuals with Disabilities Education Act (IDEA, 2004) was established to ensure all children with disabilities reach this collective goal and are provided opportunities for acquisition of knowledge and skills that lead to personal independence and social responsibility.

Research on the outcomes of adults with ASD yields sobering results. While some individuals with ASD are able to successfully achieve maximum independence and live self-fulfilling lives, many are faced with very significant obstacles in multiple areas. Even for those considered to be more skilled, they are unable to negotiate their way into college, work, community participation, and independent living. Experience has proven that individuals with all levels of ASD can work in a variety of businesses and industries (O'Brien & Daggett, 2006). A spectrum of employment options is now available which includes segregated training centers, supported employment, and competitive employment. Despite research documenting the benefit of community-based

employment, the vast majority of people with ASD continue to be unemployed or underemployed (National Organization on Disability, 2004; Wagner, Newman, Cameto, Garza, & Levine, 2005). Additionally, participation in secondary education may provide a suitable alternative to employment, however, limited research in this area indicates participation is low (Howlin, 2000). There is a continuum of living arrangements available to individuals with ASD which includes living independently, living with family, partially supported living, and fully supported living. While an array of residential options is available, research on living outcomes demonstrate a small percentage of adults with ASD live alone, some reside in long stay hospitals or institutions, but most continue to live at home with their parents (Billstedt, Gillberg, & Gillberg, 2005).

Such disappointing outcomes for adults with ASD have led to a shift in ensuring functional life skills are addressed during the educational years. Although it is important to focus on academic content and to make sure students with ASD are provided the opportunity to benefit from the general education curriculum, it has becoming increasing necessary to ensure they are also taught skills needed to achieve independence and community integration. This should not be viewed as a lessening of goals but instead as an opportunity to engage all students in real-life application of skills.

Current Knowledge

Each student is an individual and requires a specialized set of learning objectives specifically designed to meet his or her needs. In many classrooms across the United States, students' individualized education plans (IEP) are virtually identical in content. This is providing an injustice to many students, as not all will require the same skills to achieve maximum independence. Considerations regarding functional life skills designed for current and future success will include different learning objectives based on the student. Such skills are individualized and focus on the unique qualities, dreams, and desires of the student. They require planning for the future, providing a positive road map regarding what it may hold, and systematically addressing the skills needed to achieve it. Through such visionary planning, the student learns what is needed for enhanced functioning in the natural community.

Determining skills to teach involves cooperation between students and teachers. Perhaps the most important factor when selecting functional life skills to teach is the value of student and family preferences. Of crucial significance is whether the student is interested in the skill and has had a role in selecting the objective. If the student has been involved, he or she will be more motivated to learn it and are far more likely to use it outside of the school setting. Additionally, learning skills the student is vested in can lead to increasing self-concept and sense of accomplishment.

Another critical step to determining functional life skills requires examining a student's current and future environments (Brown, Nietupski, & Hamre-Nietupski, 1976). An ecologically oriented approach requires the student, teacher, and family to identify the environments and sub-environments where the student participates. For example, the student may participate in the school, home, synagogue, gym, and park. Next, the team discusses the student's high-priority activities for each of these major living environments. For example, if the student goes to a fast-food restaurant every Saturday, he may need to learn to take turns, wait, order food, and exchange money. There is no limit to the number of functional life skills identified using an ecological approach since any student with an autism spectrum disorder will have some deficits related to increasing independence that will need to be addressed (Schall, Doval, Targett, & Wehman, 2006).

Although targeting functional life skills is traditionally emphasized at the high school level, they should be carefully considered for elementary and middle school students as well. Certainly, as the student ages and needs evolve, the skill requiring instruction will change.

Acquisition becomes more essential as the student gets closer to graduation but should begin early for every student with ASD.

Although functional life skills will differ from individual to individual, there are key areas to be addressed. They include the following:

- Career education and work: Students need to learn skills that will allow them to identify a career path, obtain and hold a job, and understand the relationship between work responsibility, pay, and getting along with others (Wehman, 2001).
- Community participation: Community-based skills encompass a breadth of skills related to mobility, accessibility, and interactions. Listing the number of skills related to community is nearly impossible because there are so many. However, skills may include using a sidewalk, crossing a street, soliciting a retailer, or waiting in line at a movie theatre.
- Home living skills: Domestic skills are essential for community living. The optimal living environment for the student must be identified and then the skills essential for success targeted. Examples of home living skills include preparing lunch, cleaning the bathroom, and making the bed.
- Self-help: A key component of independence is self-care. Self-help skills allow a person to meet his own needs and be less reliant on another person. Self-help skills include such skills as eating, meal preparation, dressing, grooming, and toileting. They are critical skills as some are needed to survive (e.g., eating) while others are needed to leave the house and participate in the community (e.g., getting dressed). Self-help skills can be a major factor in accessibility to certain environments. For example, a young child with ASD who is not toilet trained may not be allowed in a child care center, while an adult with ASD who cannot use a public restroom may not be able to maintain a job.
- Transportation: A major aspect of independence in the community is related to getting to and from various locations. Skills related to transportation include not only using various modes of transportation such as driving a car or using a bus but also include planning travel based on desire as well as need to get to determined locations.
- Self-determination: Self-determination prepares students for adulthood teaching them to take control over their lives, understand their rights, and self-advocate. Too often, students with ASD become highly dependent on caretakers. This reliance then inhibits independence. Some examples of skills related to self-determination include self-awareness, goal setting, choice making, decision making, assertiveness, and problem solving (Wehmeyer & Lawrence, 1995).
- Personal health and safety: Acting responsibly and promoting the well-being of one's self as well as others is a vital functional skill. This focuses on attending to one's own health needs and demonstrating behaviors that ensure safety and may include such skills as applying a Band-Aid, calling 911, or monitoring medication.
- Financial planning and management: Economic self-sufficiency is a goal for most individuals with or without a disability. Activities may range from basic skills such as managing money to more complex activities including investment planning. Skill related to financial planning involves knowing how to shop by comparing prices, budgeting, saving for a purpose, investing, as well as avoiding scams and unfair marketing practices.
- Functional academics: Most education programs focus on academic content. For some students, learning the general education academic curriculum may not be possible. Therefore, functional academics may be advantageous. This involves teaching academic content in a way that uses real-life application and moves the student toward independence. For example, a student working on reading may learn to identify sight words he or she will see in the community. A student working on math may learn to count money and add the cost of two items purchased. An essential way to determine whether to teach functional academics is to ask whether the student will be able to use the information

currently or in the future or if the information is needed to obtain the student's educational goals. Increased learning of functional academics may impact the type of diploma received and may determine whether the student is able to pursue postsecondary education.

- Socialization: Appropriate social skills are essential for all people. Socialization deficits is the hallmark characteristic of ASD. Therefore, targeted instruction in this area is required for every student on the spectrum. The skills required for socialization are complex and are arguably the most difficult for an individual with ASD to learn. However, unless social skills are taught, full community integration and social fulfillment remain a challenge (National Research Council, 2001). Socialization skills include those related to peer interaction, friendships, and the range of relationships one may encounter. Additionally, socialization includes skills related to comprehending social stimuli, reciprocation, social rules, and affect and emotions.
- Sexuality: Sexuality is a natural part of the human experience, and it is a right for individuals with ASD to experience it. Skills related to understanding the facts regarding sexuality as well as the social rules surrounding its expression are typically required for a person on the spectrum (Stokes, Newton, & Kaur, 2007).
- Recreation and leisure: Students need to learn activities to perform in his or her free time. For many with ASD, leisure pursuits are rarely community based and are likely to be isolated activities such as video games and watching television (Jennes-Coussens, Magill-Evans, & Koning, 2006; Wagner et al., 2005). Skills related to recreation and leisure may include teaching independent play skills to reduce stereotypy, interactive games, or team activities.

Future Directions

With the passage of No Child Left Behind (NCLB, 2001) as well as the revision of IDEA (2004), access to the general education curriculum has become a more prominent focus for students with disabilities including those with ASD. However, as research has evolved and we have learned more about the autism spectrum, it has become increasingly clear that all students with this disorder require a focus on functional life skills in addition to other curricular content. Instruction is required related to community, employment, home, and leisure as well as academics related to the transition goals (Downing, 2005; Nuehring & Sitlington, 2003). Moreover, strategies for remedying communication, socialization, and behavioral deficits which profoundly and perpetually impact the individual are critical (Iovannone, Dunlap, Huber, & Kincaid, 2003). Future directions in this area include educating teachers on how to identify critical life skills students need to be successful as well as help them to integrate such skills into the general curriculum, so the student leaves school armed with the skills needed for adulthood.

See Also

- ▶ Adaptive Behavior
- ▶ Curriculum

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Functional MRI

▶ Event-Related Functional Magnetic Resonance Imaging (MRI)

Functional Play

▶ Play

Functional Routines (FR), Teaching

Katherin C. Holman
Department of Special Education, Towson University, Towson, MD, USA

Definition

Information-processing deficits in individuals with autism spectrum disorders (ASD) inhibit their ability to accurately attend to instructions, effectively participate in learning and social activities, and easily adapt to novel activities and change. Routines are effective at diminishing the information-processing load (Shatz, 1983) by providing an inherent organizational structure that reduces variation and supports individual's participation in them (Constable, 1986). The teaching of functional routines can assist individuals with ASD to be more successful in engaging in and completing everyday activities. Routines have the benefit of being sequential (McClannahan & Krantz, 1999; Siegel-Causey & Guess, 1989; Yoder & Davies, 1992) and predictable (Prizant, Wetherby, Rubin, Laurent, & Rydell, 2006; Synder-McLean, Solomonson, Mclean, & Sack, 1984) and are found in the natural environment (Pretti-Frontczak & Bricker, 2004; Sussman, 1999). Functional routines are predicable events that involve a chain of behaviors that are broken down into teachable units. They provide a reliable context that allows the

individual to more independently participate or complete a specified activity. For example, the behaviors of arriving to school, hanging one's coat up in the locker, traveling to desk, and taking out materials for the day are all integrated behaviors that must be completed to achieve the function of this routine. The routines will vary upon the setting in which they are taught. If it is during the school day, they could consist of transitioning and arrival, participating in circle-time or classroom activities, using the restroom, greeting a peer, eating lunch, etc. If the functional routines are being taught in the home environment, they may consist of improving daily living skills, such as independently dressing, eating meals, playing a social game with a caregiver, or making leisure choices.

Historical Background

Routines have been described as a "scaffold" for children (Bruner, 1975). Bruner and his colleagues focused their research on the development of joint-action routines which promote shared attention, cooperative activity, and language development, for example, peekaboo, book sharing, or object exchange (Ninio & Bruner, 1978). This use of routines was later adapted by McLean and Snyder-McLean (1987) as part of a communication intervention that focused on highlighting the identifiable cycles and turn-taking opportunities that were present in everyday routines, such as feeding or bathing. The use of routines has historically been implicated in various language interventions (see Constable, 1986; Nelson, 1986).

Functional skills are described as those that will have a meaningful impact on the individual's life. The importance of explicitly teaching skills that are functional and using a routine as the platform for teaching these skills has been a critical intervention focus, particularly with individuals with severe disabilities (Brown, Branston, Nietupski, Pumpian, Certo, & Gruenewald, 1979; Snell, 1983; Wilcox & Bellamy, 1982). Historically, a task-analysis model was used to identify the skills and activities that are functional for a student and then further divide the activity or routine into teachable units of behavior. Brown, Evans, Weed, and Owen (1987) offered the component model of functional life routines, which provides a systematic structure for the specification of behaviors relevant to student competence. The model was proposed to provide a more comprehensive approach to the identification of behavioral sequences that will lead to a more successful and independent completion of routines by the individual with a disability.

Specific research related to the use of functional routines with individuals with ASD is scarce; however, the concept has evolved from both language interventions (as described above) and the documented need to provide predictability and routine (Dawson & Osterling, 1997) to promote participation and independence for individuals with ASD. Further, the use of routines is often a component in functional communication training, which is a common intervention used to improve the language and behavior of individuals with ASD (see Mancil, 2006).

Current Knowledge

Limited research is available regarding the sole use of functional routines in teaching individuals with ASD. Often, the use of functional routines is naturally embedded as part of a comprehensive intervention program or classroom model (Arick et al., 2003; Dawson et al., 2010; Landa, Holman, O'Neill, & Stuart, 2011). The use of functional routines varies on the setting in which they are being used. In the school or workplace setting, they can be used as a generalization strategy after specific behaviors have been taught in a highly structured context (such as one-on-one using discrete trial training), and now they might be chained together in a more natural context (such as circle time) to promote participation or engagement in the routine.

The STAR program, Strategies for Teaching Based on Autism Research, identifies functional routines as one of the three primary instructional strategies (the other two being discrete trial training and pivotal response training) that comprise the intervention. The STAR program

utilizes functional routines to assist the young child with ASD to independently participate in common school (e.g., participation in circle-time routines) and self-care routines (e.g., using the bathroom). The use of functional routines in this program assists with creating a structured learning environment. Arick and colleagues (2003) researched the use of functional routines in a preliminary outcome study of the program and noted it to be one of the most common one-to-one instructional strategies utilized in the population studied.

Rationale or Underlying Theory

The underlying theory for teaching functional routines is grounded in the need for creating a context that is predictable, repeated often (offering multiple opportunities to practice and participation), and comprised of smaller segments of functional activities that can be broken down and taught part by part. The behaviors taught as part of the routine are functional to ensure more independence in the individual's life. The routine can be conceptualized as a script (Bruner, 1975) for the individual with ASD and reduce the cognitive load and improve their successful and independent participation in or completion of the activity (Nelson, 1986).

Goals and Objectives

When identifying the goals and objectives associated with a functional routine, one must consider the functionality of the skills targeted within the curriculum or those required for completion of important activities in the life of an individual with ASD. Focus should be on those skills that (a) are most likely to be useful in the student's life to control his or her environment, (b) will increase the student's independence and quality of life, and (c) will increase the student's competent performance (Iovannone, Dunlap, Huber, & Kincaid, 2003). The goal will most likely be to independently and successfully complete or participate in the specified functional routine. Each functional routine can be task analyzed into a number of core steps or behaviors, and these behaviors can be identified as the objectives. Independent mastery of each objective in succession will lead to the functional and independent completion of the routine. The ASD individual's specific strengths and needs, the context, and the actual steps or components of the routine will determine the specific goals and objectives.

Treatment Participants

Traditionally, the teaching of functional routines has been used with individuals with cognitive impairments and those with ASD. They can be utilized with very young children, related to creating routines for caregivers and children (Kashinath, Woods, & Goldstein, 2006; Kern, Wolery, & Aldridge, 2007) or with older individuals who need assistance completing functional life skills (Iovannone et al., 2003; Ogletree, Oren, & Fischer, 2007).

Treatment Procedures

There is an inherent cycle for teaching functional routines, which includes (a) identification of a routine which is meaningful for the individual with ASD to complete independently, (b) task analysis of the steps and skills needed for a particular routine, (c) assessment of the individual's independent completion of each step or use of each skill to successfully participate or complete the routine, (d) direct instruction of each behavior/skill required in the functional routine, and (e) data collection on independence in participation or completion of the routine postinstruction. Further explanation of this cycle is below:

1. Identification of a functional routine. Determine what routine is meaningful for the individual with ASD to learn to complete independently. This could be to successfully arrive at work and prepare for the day, taking a bath at home, or participating in a

song-gesture routine at circle time. The specific routine will depend on the context and the individual needs of the person with ASD.
2. Task analyze the steps and skills needed for a particular routine. The adult who will be teaching the functional routine should break down the routine into specific sequential steps and skills and identify what behaviors need to be taught for successful participation or completion of the routine.
3. Assessment of the individual's independent completion of each step of the routine. Prior to teaching the functional routine, the adult instructor should complete a baseline measurement of the routine and measure the ASD individual's independence in completing each of the identified steps and skills in the functional routine. After collecting this information, the adult instructor should develop a lesson plan identifying the specific objectives (behaviors/skills) that need to be taught and the instructional procedures and strategies that will be used to teach the behaviors/skills for the routine.
4. Direct instruction of each behavior or skill in the functional routine. Depending on the needs of the individual with ASD, one might need to preteach each specific behavior individually using direct instruction. Once each step and behavior is mastered, then during the day, when the functional routine occurs, the instructor should provide the needed instruction and support for the individual to participate and complete each step of the routine.
5. Progress monitoring. Consistently monitor the ASD individual's successful and independent completion of the functional routine. If the individual has mastered the routine/related skills, restart the process and identify a new functional routine. If not, return to instruction and reevaluate the instructional strategies.

The teaching of functional routines is typically carried out one-to-one or in a caregiver-coaching model. The particular routine that is taught will be determined by the functional needs of the individual within the particular context (e.g., home, school, or community).

Efficacy Information

There is limited efficacy data available for teaching functional routines.

Outcome Measurement

Outcome measurement for functional routines is related to the specific behaviors that are needed to successfully complete or participate in the routine. These behaviors have typically been identified through the task analysis completed prior to teaching the functional routine. See further information described in the section "Treatment Procedures."

Qualifications of Treatment Providers

There are no specific qualifications for treatment providers. Treatment providers can be teachers, speech-language pathologists, caregivers, counselors, behavior therapists, job coaches, or other significant adults who work and who are familiar with the individual with ASD.

See Also

- ▶ Functional Assessment and Curriculum for Teaching Everyday Routines
- ▶ Functional Communication Training

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Functional Speech

► Verbal Communication

Functionally Equivalent Alternative Behavior

Kristen D'Eramo
The Center for Children with Special Needs, Glastonbury, CT, USA

Synonyms

Replacement behavior

Definition

Functionally equivalent alternative behaviors, or functionally equivalent replacement behaviors, are desirable/acceptable behaviors that achieve

the same outcome as a less desirable problem behavior. For example, an individual may shout in order to gain his or her parent's attention when the parent is otherwise occupied. An alternative behavior that could serve the same function might be to say "Excuse me." In order to determine an appropriate alternative behavior, one must first understand the function of the problem behavior. This requires completion of a functional behavior assessment. Once the function of the problem behavior is clearly articulated, an appropriate alternative response, or repertoire of alternative responses, can be selected that are equal in function to the targeted challenging behavior. Alternative responses should be selected based on their social validity and should be more efficient than the challenging behavior. For example, saying "excuse me" and raising one's hand are both socially acceptable ways to gain attention and thus represent potential alternative behaviors. On the other hand, circling a person three times silently may be another way to gain attention without shouting but should not be selected because the behavior lacks social validity. Once an alternative behavior is selected, the individual should be specifically taught to engage in the behavior and then reinforced for doing so. In the current example, the child would be taught to say "excuse me" and then reinforced for doing so. In order to make the alternative behavior more efficient than the problem behavior, the problem behavior should not be reinforced or should be reinforced less often. In the example cited, the parent would consistently provide attention to the child when he or she says "excuse me" but ignore him or her when he or she shouts. In another example, a learner who engages in hitting others (problem behavior) in order to escape work demands (function) may be taught a functionally equivalent alternative behavior of requesting a break. The behavior of requesting a break allows the individual to meet his or her need (escaping the demand) without engaging in a socially inappropriate response. Approaches to intervention that seek to eliminate problem behaviors without teaching functionally equivalent alternative behaviors are ill advised as the individual will ultimately select his or her own ways to meet the needs that the extinguished problem behavior was previously serving. For example, a child who shouts at his or her parent to gain attention could be taught to stop shouting for attention if the shouting is consistently ignored but over time he or she may begin hitting his or her parent in order to solicit attention if he or she is never taught what to do instead (e.g., say "excuse me"). Behavior support plans require explicit programming that addresses functionally equivalent alternative behaviors in order to best address individual needs. This includes specific teaching procedures for the alternative behaviors and plans to systematically reinforce the alternative behaviors when they occur.

See Also

- ▶ Differential Reinforcement
- ▶ Functional Behavior Assessment
- ▶ Functional Communication Training

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Fusiform Face Area

Susan Faja
Department of Psychiatry and Behavioral Sciences, University of Washington, Seattle, WA, USA

Definition

The fusiform face area (FFA) is a region of the cortex in the inferior temporal lobe of the brain

that has been shown to respond most strongly to faces compared with other types of input (e.g., objects) for typically developing individuals. Its activation has widely been investigated as a biological measure of face processing impairment among individuals with autism spectrum disorders (ASD). It is also referred to as the fusiform gyrus.

Historical Background

Processing of faces has been consistently linked to a particular pattern of neural activity, suggesting certain aspects of cortical networks in the visual stream are specifically adapted for these processes. Lesions affecting the fusiform gyrus have been associated with a disorder called prosopagnosia, which is characterized by an inability to recognize faces (e.g., Whiteley & Warrington, 1977). In the early and mid-1990s, a series of studies using positron emission tomography (PET) demonstrated that the activation of a portion of the fusiform gyrus was greater for faces compared with objects and nonsense pictures in healthy adults without autism (e.g., Kanwisher, McDermott, & Chun, 1997; Puce, Allison, Gore, & McCarthy, 1995; Sergent, Ohta, & MacDonald, 1992). In addition to activation within the fusiform gyrus, face-selective activation has also been found in the superior temporal sulcus and inferior occipital gyrus and is part of a larger network that is involved with processing information such as eye gaze and emotional expression (for reviews, see Haxby, Hoffman, & Gobbini, 2000; Johnson, 2005).

Within the fusiform gyrus, the middle lateral portion is typically the focus of activation to faces. Activation patterns vary between individuals. Typically, face-specific activation is functionally localized with a *face localizer task* in which the individual is asked to view faces and non-faces. Among some individuals, activation is found only in the right hemisphere, while for others activation may also be found in the left hemisphere or in both hemispheres.

In 2000, Schultz and his colleagues conducted the first investigation of the integrity of the fusiform gyrus among individuals with autism spectrum disorders (Schultz et al., 2000). They examined the brain activation of 14 high-functioning young adults with autism or Asperger syndrome versus 28 individuals with typical development using functional magnetic resonance imaging (fMRI). This first study found that activation in the fusiform gyrus was reduced for individuals with autism spectrum disorders compared with typically developing individuals when faces were presented. Instead, individuals with ASD exhibited greater brain activation in the inferior temporal gyrus, which was activated more strongly in the comparison group in response to objects. There were no differences detected between groups for activation to objects.

Current Knowledge

Initial work by Schultz paved the way for a series of fMRI studies that examined the activation of the fusiform face area among individuals with autism and ASD. In the past decade, there have been more than 15 published reports of reduced activation of the fusiform gyrus in response to faces by individuals with ASD compared with typically developing individuals. The majority of these studies have used adult participants. Most tasks involved active discrimination of faces, typically on the basis of identity or expression. Static photos of faces were used for nearly all studies, although reduced activation of the fusiform face area in ASD compared with controls has also been found in response to moving faces (e.g., faces with changing facial expressions, short naturalistic video clips with faces). Reduced activation in individuals with ASD has been found when viewing faces with neutral and emotional expressions as well as familiar and unfamiliar identities.

In contrast to these studies, there have been several reports of equivalent activation of the fusiform face area among individuals with ASD and typically developing individuals. There are several possible explanations for these inconsistent findings. These include the use of a task that places less of a processing demand on the

participant (i.e., viewing the face passively rather than discriminating faces), use of faces that may be more motivating or interesting to the person viewing them (i.e., familiar vs. unfamiliar faces), placement of a fixation cross on the face that may increase attention to the face – particularly visual attention to the eyes. As well, small sample sizes combined with variability of symptoms among individuals with autism spectrum disorders may contribute to mixed findings.

Understanding the variability in these findings is important. One factor that appears to influence the activation of the fusiform face area is the degree of familiarity of the face being presented. As Pierce and her colleagues have shown (e.g., Pierce & Redcay, 2008), both adults and children with autism and age-matched controls show similar levels of fusiform activation when viewing familiar faces. Secondly, Dalton, Davidson, and their colleagues have shown that attention affects activation of the fusiform gyrus. Specifically, increased activation appears to have a strong, positive association with visual attention to the eye region of the face. This effect has also been found in adolescents and adults, and the adolescent study demonstrated this effect when viewing familiar and unfamiliar faces (Dalton et al., 2005). More recently, when an experimental manipulation was used to vary the time individuals with autism spectrum disorders spent looking at the eyes, associated activation patterns appeared more typical in the fusiform face area (Perlman, Hudac, Pegors, Minshew, & Pelphrey, 2010). Thus, inclusion of a red cross to indicate where viewers should fixate on the face may have positively influenced attention in three studies that have found similar activation of the fusiform face area in individuals with ASD and controls (e.g., Hadjikhani, Joseph, Snyder, & Tager-Flusberg, 2007). Finally, it is important to recognize the heterogeneity of symptoms in autism spectrum disorders, including symptoms in the social domain. This may be an important consideration in interpreting mixed findings for the activation of the fusiform gyrus. This was recently illustrated by an investigation that assessed the influence of social anxiety on the activation of the network involved in face processing. For individuals with autism spectrum disorder who had greater social anxiety, activation of the fusiform face area was decreased (Kleinhans, Richards, Weaver, Johnson, Greenson, Dawson, & Aylward, 2010). Taken together, requiring viewers to more actively engage in face processing by discriminating or labeling, presenting faces that hold more personal interest, directing attention to the face, and including participants with fewer social difficulties (i.e., better eye contact, reduced social anxiety) would be consistent with an account that increased visual attention to the face increases activation of the fusiform gyrus in individuals with autism spectrum disorders.

While the region of the fusiform gyrus that is activated in response to faces is thought to be uniquely specialized for faces, an alternate viewpoint suggests that it instead is associated with processing any visual category for which the viewer has developed expertise (e.g., Gauthier, Skudlarski, Gore, & Anderson, 2000). That is, experts develop knowledge of individuals within a category (e.g., a white-breasted nuthatch rather than a bird in the case of a bird expert) and are able to perceptually distinguish individuals within that category (e.g., blue grosbeak vs. a field sparrow) as automatically as they distinguish between categories (e.g., bird vs. butterfly). Among typically developing individuals, greater activation in the fusiform face area has been found when experts view images within their category of perceptual expertise relative to other categories. Further, typical adults have been trained to expertly categorize a perceptual category and develop activation of the fusiform face area in response to that category. In this view, activation is related to expertise rather than viewing a face per se; faces are the most common and most robust source of activation because typically developing individuals learn to categorize faces at the individual level. Given the expertise of some individuals with autism for object categories, the possibility that the fusiform face area would be activated while viewing images related to their specialized interest was explored. A case study of an 11-year-old boy with autism with an intense interest in animated Japanese "Digimon"

characters found that he had enhanced activation of the fusiform face area for Digimon, but reduced activation for faces (Grelotti et al., 2005). This activation pattern was unique for the boy with expertise, and a comparison boy with autism and a typically developing boy did not have enhanced activation for Digimon. Both boys with autism had reduced activation of the fusiform for faces relative to the typically developing boy.

Most research to date has focused on adult participants, which provides information about the endpoint of development. However, understanding the development of face processing and recognition in autism is important given the possibility that activation of the fusiform gyrus may be experience driven. Several studies have specifically examined the activation of the fusiform gyrus during childhood and adolescence and have shown that children with autism spectrum disorders also activate the fusiform face area less robustly than comparison children on tasks that involve face and emotion recognition. Of interest, Pierce and Redcay (2008) tested children aged 6–12 years while they viewed familiar and unfamiliar faces of adults and children. They found similar activation patterns between children with autism and comparison children when they viewed familiar adult faces and children, but significantly reduced activation when viewing the faces of unfamiliar adults. One possible explanation for these findings is that children with autism may have had reduced attention or motivation while viewing the faces of unfamiliar adults.

Future Directions

Although the fusiform gyrus is activated in tasks that also involve emotion processing, it is primarily activated in response to recognition of faces. Nonetheless, Bölte, Hubl, Feineis-Matthews, Prvulovic, Dierks, and Poustka (2006) assessed whether providing training to individuals with ASD aimed at improving their emotion recognition skills would impact the activation of the fusiform gyrus. While they found changes in activation elsewhere, training did not appear to impact activation in the fusiform. One future direction will be to determine whether training in face recognition or other social skills training impacts the activation of the fusiform face area. A promising approach would be to target younger children with ASD, rather than adults, as children may have greater neural plasticity than adults.

A second direction that warrants future research attention is the interplay between the fusiform gyrus and the broader network of brain regions involved in social processing. Hadjikhani and her colleagues have twice reported activation of the fusiform face area that did not significantly differ from typically developing adult controls. However, they reported that the overall activation pattern was reduced in other social brain areas including the right amygdala, inferior frontal cortex, superior temporal sulcus, and premotor cortex (Hadjikhani et al., 2007). A similar pattern of reduced activation in a distributed network of structures involved in social processing, but not in the fusiform face area, was also found in children with autism spectrum disorders (Bookheimer, Wang, Scott, Sigman & Dapretto, 2008). Investigating the pattern of activation, as well as structural and functional connectivity between structures in this network throughout development, will shed light on the biological basis of social impairment in autism.

Third, there has been increasing interest in the structure of cells within the fusiform gyrus as well as its overall shape and volume. In one such recent study, thickness of the cortex in the fusiform gyrus was greater among adults with autism than healthy controls, and this thickness was specific to the fusiform (Dziobek, Bahnemann, Convit, & Heekeren, 2010). Interestingly, this thickness related to impaired face processing within the group with autism. Altered cortical volume and thickness of the fusiform may be related to the age of the individual with ASD. In addition, neuroimaging of pathways between the fusiform gyrus and structures such as the hippocampus and amygdala suggests the presence of structural abnormalities in the fibers connecting these regions.

Another direction of future investigation is exploring potential genetic contributions to the structure and function of the fusiform gyrus. Given that the fusiform face area is well-studied in autism spectrum disorders, it is a good candidate for investigation. An exploration of CNTNAP2 (*contactin-associated protein-like 2*) polymorphisms, which have been suggested to contribute to the genetic risk for autism spectrum disorders, and brain structure in typically developing individuals found an association between the volume of the fusiform and CNTNAP2 polymorphism (Tan, Doke, Ashburner, Wood, & Frackowiak, 2010).

Finally, it will be important to understand variability in fusiform structure and function beyond individuals with autism spectrum disorders. The fusiform face area and the related network of structures underlying social function have been implicated in other disorders, such as schizophrenia. And genetic variability even within the typically developing population may influence structure and function of the fusiform. Yet, investigation of face fusiform function has focused on comparison between groups of individuals with autism spectrum disorder and typical development. In the future, it will be especially important to compare autism with other disorders and to explore factors related to structure and function of the fusiform gyrus (e.g., with genetic expression, behavior) that are found in both clinical and nonclinical populations.

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Fusiform Gyrus (FG)

Danielle Perszyk
Yale Child Study Center, New Haven, CT, USA

Definition

The fusiform gyrus (FG) is a brain region consisting of occipitotemporal cortex (temporal lobe) that corresponds to Brodmann area 37. It is involved in high-level visual processing, specifically object recognition and category identification, of complex stimuli such as faces. Individuals with ASD demonstrate deficits in certain cognitive abilities for which the fusiform gyrus has been implicated – most notably and socially relevant being face processing – as well as abnormalities in the fusiform gyrus itself.

Historical Background

The fusiform gyrus is a brain region involved in high-level visual processing. Visual processing is organized hierarchically and involves many brain regions working together to transform information provided by the external world into internal representations. Early low-level visual processing occurs milliseconds after light hits the retina and distinguishes basic, isolated features such as orientation, spatial frequency, and color. Later high-level visual processing combines information from lower levels of processing and culminates in identification, recognition, and categorization of objects and actions. Visual processing begins in occipital cortex and proceeds anteriorly via ventral and dorsal pathways; the highest levels of processing occur in temporal cortex. The ventral and dorsal pathways are hypothesized to represent the processing of "what" and "where/how" information, respectively, and are correspondingly associated with "perception" and "action" (Underleider & Mishkin, 1982). The FG has been shown to be involved in numerous ventral stream processes, including high-level visual processes that entail semantic encoding.

The initial discoveries concerning specific functions of the FG arose from studies of primates (Van Essen, Anderson, & Felleman, 1992) and patients with brain lesions or psychological disorders (Mesulam, 1994), both of which have revealed the anatomy of the mammalian visual system. First, single-unit recordings from the inferotemporal cortex in macaques found populations of neurons that responded specifically to faces (Gross, Rochamir & Bender, 1972). Then, electrophysiological studies in humans suggested similar cortical specialization. Recordings from the same brain region in epilepsy patients with implanted subdural electrodes showed large N200 potentials elicited by faces but not scrambled faces, cars, or butterflies (Allison, Mccarthy, Nobre, Puce, & Belger, 1994). These studies were consistent with an older study of brain-injured patients that showed the same brain region seemed to be involved in recognizing faces but not houses (Yin, 1970). Additional studies of patients with damage to occipitotemporal cortex of the right hemisphere, who had selectively lost the ability to recognize faces, further supported the notion that this region is involved in face processing (DeRenzi & Saetti, 1997). At the same time, research in several areas, including cognitive psychology, neuropsychology, neurophysiology, and computational vision, converged to suggest the differentiation of face and object recognition as qualitatively different processes and perhaps even entailing distinct brain areas.

As research continued building an understanding of the function of the FG, a growing body of evidence indicated that face processing was not its only function. Neuroimaging became more popular as a technique to study brain function, and as a result, specific brain regions could be much more easily implicated in a wide range of cognitive processes, including processing shape and surface properties such as color and texture (Barrett et al., 2001), number and word recognition (Allison, Mccarthy, Nobre, Puce, & Belger, 1994), and within-category identification (Gauthier et al., 2000). Yet while these studies

showed that the FG is involved in myriad visual process, individuals with ASD do not characteristically demonstrate deficits in the lower level visual processes (e.g., shape and surface properties); rather, they often demonstrate difficulty integrating more basic features into configural representations and processing certain higher level visual categories, specifically the socially relevant category of faces. Early research investigating face processing in ASD showed that, unlike typically developing peers, children with ASD fail to show both a "face inversion effect" – a disproportionate difficulty recognizing upside down faces – and a face "decomposition effect" – reduced recognition of face components or partially obscured faces (Langdell, 1978). That is, children with ASD do not show more difficulty recognizing upside down faces than upright faces, nor do they show more difficulty recognizing face components or partially obscured faces than whole faces. These abnormalities in face processing suggest that, unlike typical counterparts, individuals with ASD do not process faces holistically (Freire, Lee, & Symons, 2000), but instead, they may process faces using feature-based strategies similar to how typical people process objects.

Given that ASD is at is core a social disorder and that face perception is among the most vital of social functions, it is especially noteworthy that individuals with ASD were observed to process faces atypically. But it remained uncertain whether these face-processing abnormalities stem from a primary brain abnormality, a primary behavioral difference (i.e., lowered attention to faces), or some combination and subsequent interaction thereof. Studies found that individuals with ASD already show lower attention to faces as young as 1 year of age (Osterling & Dawson, 1994); by childhood, they show more difficulty than typically developing children discriminating and recognizing (Boucher & Lewis, 1992) faces, and through adolescence and adulthood, they continue to show poorer recognition of faces than typically developing counterparts (McPartland, Dawson, Webb, Panagiotides, & Carver, 2004). In order to disentangle the possible origins of these behavioral deficits in face processing – whether they arise from primary brain abnormalities or primary behavioral differences – later studies used neuroimaging techniques to examine the brain bases of face processing in ASD. Functional magnetic resonance imaging (fMRI) studies showed that, when viewing neutral faces, individuals with ASD exhibit lowered fusiform gyrus activity compared to typical counterparts (Schultz et al., 2000). Furthermore, Schultz and colleagues (2000) suggested that individuals with ASD process faces more similarly to how typical counterparts process objects, with decreased FG activity and increased inferior temporal gyrus activity. Event-related potential (ERP) studies showed that children with ASD, but not typically developing children, fail to exhibit differential brain activity for familiar versus unfamiliar faces (Dawson et al., 2002); similarly, adolescents and adults with ASD, but not typical counterparts, fail to exhibit differential brain activity for upright versus inverted faces (McPartland, Dawson, Webb, Panagiotides, & Carver, 2004). Moreover, McPartland and colleagues (2004) found that individuals with ASD exhibit longer latencies to faces and that processing speed positively correlates with face recognition ability. These face-sensitive ERPs have been localized to the FG. Finally, one recent study found that individuals with ASD have smaller and fewer neurons in the FG (van Kooten et al., 2008).

Current Knowledge

Initial investigations of the FG revealed its involvement in face processing. While it has since been implicated in many additional roles, current investigations continue to focus on the role of the FG in face processing. There are presently three prevailing theories for the neural organization of face processing involving the FG. One is that the FG is specialized for face processing, which is modular and domain specific (Kanwisher, McDermott, & Chun, 1997). A second theory is that the FG is not specialized for faces per se but for objects with which one has experience or expertise; most humans have

extensive experience with and are thus "experts" of faces, making the FG specialized for faces by default. This theory proposes that the FG is domain general, and it may also serve to differentiate between specific levels of categorization, particularly subordinate levels of identification (Gauthier et al., 2000). A third theory is that the FG and, more generally, inferotemporal cortex are neither specialized for face nor object processing but for face-like features. That is, the brain region containing and surrounding the FG is topographically arranged according to visual features of objects, and the FG "happens to be" where types of visual features associated with faces are processed (Haxby, 1999).

It still remains uncertain as to which, if any, of these theories most accurately describes the role of the FG as it relates to face processing. Moreover, these theories do not comprehensively reflect face-processing research. Yet other studies suggest that face processing and the role of the FG are affected by the manner in which one conceives a visual percept. Specifically, thinking about a visual percept in a social context or imagining faces may be sufficient for FG activation. Schultz and colleagues (2003) found that the FG activated for geometric shape stimuli behaving in social but not mechanistic ways, suggesting that FG activity is dependent upon a social framework. While this interpretation is inconsistent with any of the above three theories, it is consistent with findings from other studies. These unreconciled findings have important implications for the source of social dysfunction in ASD. A major question is whether the observed abnormalities in face processing in ASD are causes or effects of the diagnostic social deficits in ASD. While some studies argue the former (Trepagnier, 1995), most evidence supports the latter; children with ASD may have experienced typical development with appropriate attention to faces early on followed by autistic regression, and individuals with ASD exhibit abnormalities in widely distributed brain systems, some of which are unrelated to face processing (Kemper & Bauman, 1998). A larger body of research suggests that face-processing difficulties stem from developmental abnormalities in social interest and attention. Individuals with ASD may exhibit face-processing deficits because they do not acquire adequate experience with faces due to a lack of social motivation (Dawson et al., 2002). This theory is consistent with both the domain-specific and domain-general theories of face processing aforementioned. Another explanation of face-processing abnormalities in ASD that has not been empirically supported is that they stem from early developmental abnormalities in the FG or, more generally, inferotemporal cortex.

Schultz and colleagues (2005) have proposed that face perception and social cognitive skills are part of a network supported by a system comprised of the amygdala and FG and that abnormalities in this brain system, which in turn cause deficits in the social perceptual network, may play a large role in the etiology of autism. They argue that the best neuroimaging evidence for brain-based differences in the ASDs thus far involves FG hypoactivation, and they describe three possible interdependent factors that might moderate the degree of FG activity in ASD. First, they suggest an attentional factor: lowered attention to a visual stimulus reduces FG activity. Second, they suggest a perceptual skill factor: chronically reduced attention to social stimuli, particularly faces, leads to reduced perceptual skill and reduced FG activity. Third, they suggest a social knowledge factor: the FG may encode social knowledge such that social judgment not necessarily involving faces may strongly activate the FG, and therefore, conversely, reduced social knowledge may reduce social judgments, which in turn may result in FG hypoactivation.

Two recent studies have provided a confound to this model. Dalton and colleagues (2005) found that when individuals with ASD view faces, amygdala and FG activation are strongly and positively correlated with time spend fixating the eyes. In addition, eye fixation is strongly and positively associated with amygdala activation, suggesting a heightened emotion response associated with eye fixation. Similarly, Morris, Pelphrey, and McCarthy (2007) found that when typically developing individuals view faces, the scanpath one follows alters FG activation such

that greater focus on the eyes (typical scanpath) as opposed to other face regions (atypical scanpath) elicits greater FG activation. These findings indicate that, in addition to degree of attention, the specific features attended to within social stimuli (i.e., faces) affect FG activation. These findings therefore imply that controlling gaze fixation for individuals with ASD when viewing social stimuli may serve to alter the neural mechanisms underlying their social deficits.

Future Directions

Further research concerning FG function will continue to disentangle the various models of face processing and, concerning the etiology of ASD, whether FG abnormalities are primary or whether deficits in social motivation and ability are primary to deficits in social skills. In addition, future neuroimaging studies will seek to investigate the effects on FG activation of various scanpaths for social stimuli in individuals with ASD. These studies will contribute to a refining of the understanding of both the causes of and potential treatments for ASD.

See Also

- ▶ Face Perception
- ▶ Face Recognition
- ▶ Occipital Lobe

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