CHDD
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INSIDE

Social Interest is Crucial: Understanding Obstacles to Language Learning in Children with ASD

In the experimental lab, the four-year-old turns her head to the right or left to play sounds. A head turn in one direction starts a recording of women speaking in motherese, the stretched sing-song speech that people use the world over when speaking to babies. A head turn in the other direction triggers a warbling, computer-synthesized sound with the same pitch and rhythm as the motherese. If the girl has autism spectrum disorder (ASD) she is more likely to select the machine sounds, a preference that makes it more difficult for her to learn language.

These and other experiments by Patricia Kuhl, Ph.D., are helping researchers understand the root causes of language deficits in children with ASD. "Children with autism may not be able to glean the information they need from the environmental signals, such as motherese, that typical children use to learn language," said Kuhl, a professor of speech and hearing sciences, co-director of the University of Washington Institute for Learning and Brain Sciences, and a research affiliate at the Center on Human Development and Disability (CHDD). Kuhl hopes to discover signals that do help children with autism learn language, information that may guide language interventions. In addition, because her tests of language perception do not require children to speak, they may be suitable for very early diagnosis of ASD.

Kuhl's ASD research builds on studies of language learning in typically developing children. "All across the planet, typically developing kids will follow a very consistent order in their acquisition of language," said Kuhl. "They coo at three months and babble at seven months and produce their first words at about twelve months. At around eighteen months to two years you have children the world over producing two-word utterances, beginning to show the ability to acquire a grammar and by three, they'll talk your leg off. They can speak in real sentences and have a conversation."

CHDD researchers are finding that children learn language with their eyes as well as their ears. Understanding how social interaction fuels language learning may improve interventions for children with autism spectrum disorders.

Kuhl analyzes this learning process to understand the step-by-step tasks and skills children must master to comprehend and express speech. She and her colleagues have found that three basic factors are critical to language learning: the ability to hear distinctions between speech sounds, the ability to analyze patterns in these sounds, and an interest in social interaction.

"You have to be able to hear the difference between *ba* and *pa* in order to hear the differences between the words *bat* and *pat*," said Kuhl. Sounds like these that change the meaning of words are called phonemes. No language uses all the possible phonemes. For example, English speakers make a distinction between the phonemes designated in English by the letters *r* and *l*. However, these sounds are actually very similar and are not distinct phonemes in Japanese. Therefore, Japanese speakers have a difficult time hearing the distinction between the English words rice and lice. In turn, English speakers have difficulty distinguishing the Spanish *b* and *p*, hearing both as the English *b*.

In addition to hearing distinctions in sounds, infants must also analyze sound patterns. They begin this process at the age of about six months. Until that time, all typically developing infants can distinguish all the phonemes in human speech, according to research by Kuhl and her colleagues. But from the ages of six to twelve months, infants begin performing complex statistical analyses to determine which phonemes they hear most frequently. They then focus on those phonemes and ignore the others. They are "forming their neural commitments, committing the circuits in the brain needed for their native language," said Kuhl. During this period, infants raised by Japanese speakers lose much of their ability to hear distinctions between non-Japanese phonemes, such as the English phonemes *r* and *l*, and infants raised by English speakers lose much of their ability to distinguish non-English phonemes, such as the Spanish *b* and *p*.

To calculate an accurate phoneme analysis, infants must accurately hear and categorize these units of language. In EEG

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studies, Kuhl has found that children with ASD, like children with language-learning impairments or dyslexia, have difficulty hearing differences among phonemes. "The abilities of these children to hear *ba-pa* or *da-ta* distinctions are simply not as good, right from the beginning," said Kuhl. "The inability to hear those distinctions will make it very difficult for these children to take accurate statistics on the language. It will mess up their frequency distributions."

A third key factor in language learning is interest in human faces and voices. "To effectively learn language, we believe infants must have social interest in motherese, as well as social skills that allow them to gather information from social interactions," said Kuhl. Attraction to motherese is strongly tied to language learning. Worldwide, adults speaking motherese enunciate more slowly and clearly, stretching and emphasizing the phonemes in their language so they are easier to understand, and so easier for a baby to categorize and imitate. Children who avoid this kind of speech miss important cues in learning language. In the motherese preference experiments described at the beginning of this article, the more that children preferred recordings of computer warbles to recordings of motherese, the more difficulty they had in distinguishing phonemes. The degree to which children preferred the computer warbles also predicted the severity of their ASD symptoms and their delay in their verbal scores.

Exaggerated sound is not the only important characteristic of motherese. Motherese speakers also exaggerate their facial expressions. "Motherese is like theater," says Kuhl. "People's eyebrows go up, their eyes are wide open." These animated facial expressions may entice infants to engage in social interactions, which Kuhl and her colleagues have shown are critical to language learning.

In one group of experiments, infants from English-speaking homes learned phonemes of Mandarin Chinese by listening to a live Mandarin Chinese speaker, but not from voice or video recordings. In another set of experiments, infants who interacted more actively with a Spanish-speaking tutor learned more. "When a tutor would hold up a new toy, some babies just stared at the toy," said Kuhl. "And some babies just stared at the face of the tutor. But other babies looked back and forth between the toy and the Spanish tutor. It's like they were taking into account how the tutor was referencing an object with language." The more infants alternated their gaze, the more Spanish phonemes they learned.

Numerous studies have shown that children with ASD are not attracted to social interaction and may find animated faces and voices distressing, said Kuhl. "We've seen babies cover their eyes when we presented them with animated speaking faces." The hyper-animation of motherese may be especially upsetting to these children. This avoidance of animated social interactions, such as motherese, combined with their difficulty in hearing distinctions between phonemes, can make it extremely difficult for children with ASD to learn language in conventional ways.

To develop language interventions that are more effective for children with ASD, researchers need to learn "what kinds of sounds these children will listen to, what kinds of faces they will watch," said Kuhl. For example, she said, it's possible that children with ASD may prefer interactions that are devoid of emotion. The ultimate goal is to "create an auditory, visual stimulus that presents language in a way that interests children with ASD enough to help them get language into their brains," said Kuhl. "If we could invent a methodology that works to the strengths of children with ASD, we might be onto something really important." ♦

Patricia Kuhl

X vs. Y: Sex Chromosomes and Brain Development

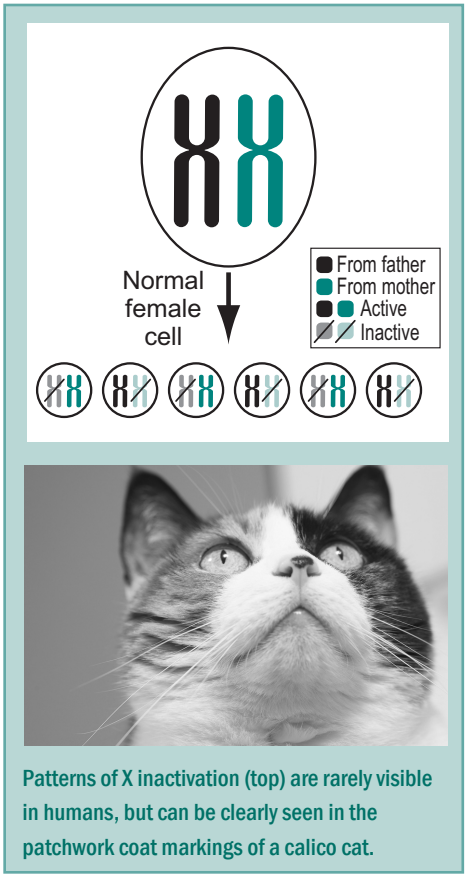
It has long been known that mutations on the X chromosome can cause developmental disabilities, including Fragile X and Turner Syndrome. Only recently have researchers begun to understand why and how X-linked mutations have such a profound impact on the brain. Christine Disteche, Ph.D., professor of pathology and CHDD research affiliate, is a leader in the study of the X chromosome and its role in brain development.

Disteche researches gene expression, the copying of genes to make proteins, especially the cellular mechanisms that ensure that expression among chromosomes is balanced, or equivalent. These balancing mechanisms are called dosage compensation. Disteche's team was the first to discover a specific form of dosage compensation necessary to balance expression of the X chromosome with that of the 22 pairs of non-sex chromosomes, known as autosomes.

Expression of the X chromosomes is inherently unbalanced because of differences in the numbers of this chromosome in males versus females. Males have one X sex chromosome and one Y sex chromosome (XY); females have two X chromosomes and no Y (XX). In contrast, the autosomes occur in pairs that carry the same genes and, for the most part, express genes from both copies. Without regulation, the one-to-two ratio of X chromosomes between males and females, as well as the one-to-two ratio of the X chromosome in males to the autosomes in males, would upset the balance of gene expression.

The number and type of genes differ between the sex chromosomes. The X chromosome is much larger, carrying about 1,100 genes compared to about 100 genes in the Y, and so has roughly ten times as many genes to express as the Y. The Y chromosome carries genes necessary for male sex characteristics, such as the development of the testes. The X chromosome carries genes necessary for both male-specific (e.g. testes) and female-specific (e.g. ovarian) development.

The X chromosome also carries genes critical for other functions, especially brain development. Disteche and her colleagues found that about 40 percent of the genes on the X chromosome are expressed in the brain. About 25 percent of X genes, or 10 percent of the total, have been linked to intellectual disabilities. This makes genes linked to intellectual disabilities three times more common on the X chromosome than on any other chromosome. Mutations in X-linked genes important to brain development can impact males more than females. If a gene on a male's single X chromosome is damaged, there is no back-up. For example, Fragile X Syndrome (FXS) is caused by a single mutation on the X chromosome (see article on FXS, page 4).



Patterns of X inactivation (top) are rarely visible in humans, but can be clearly seen in the patchwork coat markings of a calico cat.

Organisms use a variety of dosage compensation strategies to balance the expression of the X chromosome with the Y chromosome and the autosomes. For example, fruit flies (Drosophila) double the activity of the single X chromosome in males so that males have the functional equivalent of two X chromosomes. Disteche and her colleagues discovered that mammalian evolution resulted in a similar doubling of expression of X-linked genes, a mechanism they called X up-regulation. However in mammals, such as humans and mice, X up-regulation occurs in both males and females. Without further dosage compensation, this would give males the functional equivalent of two X chromosomes and females the functional of four X chromosomes. To achieve balance, yet another mechanism halves X expression in females. This mechanism, called X inactivation, was first discovered by the British geneticist Mary Lyon in 1961. X inactivation silences the expression of one of the two X chromosomes in females through structural modifications. One X chromosome in each pair is wrapped in proteins that block access to the DNA. As a result, each human female cell has one active X chromosome (Xa) and one inactive X chromosome (Xi).

X inactivation occurs very early in the development of the female embryo. In roughly half of the embryo's cells, the X chromosome from the father is inactivated, leaving the mother's X active (maternal Xa); in the other half the X from the mother is inactivated and the X from the father is active (paternal Xa). Each of these embryonic cells divides and develops into a part of the body that will express either maternal or paternal Xa. This pattern is illustrated mostly clearly by the patchwork coloration of a calico cat. The contrasting patches of fur, for example black versus ginger stripes, represent areas of skin that express either maternal or paternal Xa. This patchwork pattern of X inactivation also occurs in human females, although it is rarely evident to the unassisted eye.

As a result of X inactivation, X expression is theoretically equalized in human male and female cells. However, some Xi genes "escape" silencing. A subset of these escaped Xi genes has copies on the Y chromosome. "So in the male you have expression from Xa, plus expression from Y. And in the female you have expression from Xa, plus from some escaped genes from Xi," said Disteche. The amount of product expressed by the escaped Xi gene is roughly the same as from the Y gene. "So expression in males and females is fairly similar in the end," said Disteche. "But that's not true for all these genes and that's what makes it interesting."

Some Xi escapees appear to be similar in function to Y genes. Others are not. "For some of these genes it seems that the copy on the Y chromosome has a different function

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Gene Repeat Research May Guide Therapies for the X-Linked Neurodegenerative Disorder FXTAS

The symptoms start when you are about to turn sixty: tremors and lack of coordination that make it difficult to walk. Ten years ago you might have been misdiagnosed with Parkinson's disease. Instead, you learn that you have a newly discovered progressive neurodegenerative disorder called Fragile X-Associated Tremor/Ataxia Syndrome (FXTAS) and that you may develop dementia. The worst news is that your children's children could be born with the developmental disability Fragile X syndrome (FXS), the most common inherited cause of intellectual disability. FXS is associated with a range of learning and intellectual disabilities, as well as behavioral symptoms including attention deficits, and difficulties with speech. About one third of individuals with the full Fragile X mutation are also diagnosed with autism. Like many X-linked disorders, FXS is more common in males than females.

Individuals with FXTAS may have descendents with FXS because both disorders are caused by changes in the Fragile X gene (FMR1). The genetic variation that can lead to FXTAS is an expansion, referred to as a premutation, of a normal variation in FMR1. For reasons researchers don't yet fully understand, males with the Fragile X premutation are much more susceptible to FXTAS than females. Females with the premutation may be at risk of Fragile X-Associated Primary Ovarian Insufficiency, which is associated with infertility and early menopause.

The FMR1 expansion can increase in length as it is passed through the generations of a family, culminating in the full mutation that causes FXS, known as the Fragile X mutation. "Individuals with FXTAS don't have a developmental disability, but they're at risk of passing a developmental disability on to their children or grandchildren," said Albert La Spada, M.D., Ph.D., an associate professor of laboratory medicine, director of the University of Washington Center for Neurogenetics and Neurotherapeutics, and an associate director of the CHDD Intellectual and Developmental Disabilities Research Center. La Spada and other CHDD research affiliates are analyzing the molecular and cellular effects of the Fragile X premutation, information that may eventually contribute to treatments for FXTAS.

Over the generations, the genetic repeat that causes Fragile X-Associated Tremor/Ataxia Syndrome (FXTAS) can expand into the full Fragile X mutation, which causes Fragile X syndrome (FXS). In this family, the grandfather (left) has FXTAS and his grandchildren (right) have the full FXS mutation.
Photo credit: The National Fragile X Foundation

"Individuals with FXTAS don't have a developmental disability, but they're at risk of passing a developmental disability on to their children or grandchildren."

- Albert La Spada

FXTAS was discovered by Randi Hagerman, M.D., and her colleagues at University of California (UC) Davis through their clinical work with family members of patients with FXS. "Her discovery shows the importance of careful clinical evaluation and being open-minded to what you observe when you see patients," said La Spada. Hagerman directs the UC Davis Fragile X Research and Treatment Center, which works closely with the CHDD Fragile X Research Center of Excellence. She is also a co-investigator of the UC Davis NeuroTherapeutics Research Institute (NTRI) directed by Paul J. Hagerman, M.D., Ph.D. La Spada is a co-investigator of the NTRI, which was funded by the National Institutes of Health to investigate and develop treatments for neurogenetic disorders, including FXTAS.

FXS and FXTAS, like Huntington's disease, belong to a group of disorders caused by a genetic error resulting from the expansion of a genetic repeat. Genetic repeats are repetitions of short stretches of genetic code, which consists of variations in the order of the four nucleotide bases that make up DNA (adenine, cytosine, guanine, and thymine). In Huntington's disease, the damaging repeat occurs in the part of a gene that carries the code for a protein. The repeats change the protein's structure, resulting in malformations that damage neurons.

In FXS and FXTAS, the repeats occur in the promoter region of the FMR1 gene. The promoter region of FMR1, like that of all other genes, controls if and when a gene is expressed, or copied, to create proteins. Gene expression requires messenger RNA (mRNA), a molecule that copies genetic code from DNA and carries it out of the cell nucleus and into the cytoplasm to serve as a template for protein synthesis. Repeats in the promoter region of FMR1 affect the

expression of a protein called the fragile X mental retardation protein (FMRP). The role of FMRP is not fully understood, but researchers do know that it is synthesized in the brain and testes, as well as the liver, lungs, and other organs.

The FMR1 repeat linked to FXS and FXTAS consists of one cytosine and two guanines (CGG). Most typically developing individuals have 10 to 50 CGG repeats in the FMR1 promoter region. The repeat can expand when inherited. The Fragile X premutation occurs when the number of repeats reaches about 50 to 200. The extra repeats can trigger the production of too much mRNA. The excess mRNA appears to accumulate in the brain, causing toxicity and cell death. Messenger RNA from FMR1 has been found in inclusions, or clumps of protein, in the brains of persons with FXTAS.

The full Fragile X mutation occurs when the number of CGG repeats reaches a tipping point of about 200, silencing the FMR1 gene. Symptoms of FXS tend to be more severe in males because they have only one X chromosome and so only one copy of the FMR1 gene. If a male's single copy of the gene has the full Fragile X mutation, he does not produce any FMRP. Females typically have two X chromosomes and so two copies of the FMR1 gene. If only one copy has the Fragile X mutation, the other copy may still express some FMRP.

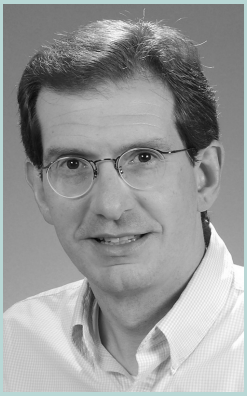
Repeat expansions may be caused by errors in DNA repairs, the complex molecular mechanisms that check the integrity of the genetic code and attempt to correct any errors, said La Spada. "It may be that repeats draw the repair machinery to a gene and a repair attempt is made but only makes things worse." The DNA repair system, which should remove the excess repeats, may duplicate them instead, exacerbating a mutation instead of correcting it.

To better study the cause and biochemical effects of repeats in FMR1, La Spada and his colleagues are developing a mouse model of FXTAS. This is proving to be a considerable challenge. Although gene repeats occur in other species, repeat-associated neurological disorders apparently do not. Repeats in the genes responsible for disorders such as FXTAS and Huntington's exist "in all mammalian species, and certainly in our non-human primate relatives," said La Spada. "However, the number of repeats does not normally get as large as they do in humans, and these neurological disorders don't occur naturally in any non-human mammal."

La Spada and his colleagues have already created mouse models of Huntington's and other related diseases caused by mutations in the coding regions of genes. The researchers are using these models to explore potential therapies including RNA-mediated interference (RNAi). RNAi involves engineering RNA molecules that block the mRNA produced by a mutated gene. If RNAi works, little or no mRNA survives to trigger the production of malformed proteins.

FXTAS, and other disorders linked to repeats in a gene's promoter region, present a different therapeutic challenge. In these disorders, the mutated gene expresses correctly formed mRNA, but in the wrong quantity. Excessive interference with mRNA production could cause serious side effects. La Spada and his colleagues plan to use their mouse model of FXTAS to test possible FXTAS treatments. The model will also be used to further study the molecular mechanisms of the disorder. For example, La Spada and his team will analyze how FXTAS affects different types of brain cells. "Understanding the cell types that are involved in the disease guides where you need to direct therapies," said La Spada. "We're really stepping back and trying to understand the cellular basis of this disease as a prelude to planning therapeutic interventions."

More information about FXTAS is available at the National Fragile X Foundation: http://www.fragilex.org/html/home.shtml ♦



Albert La Spada

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in males than the corresponding gene on the X chromosome does in females. Therefore the X version of that gene is truly more highly expressed in females," said Disteche. "I think that these differences in X-linked expression between male and female are probably going to turn out to be important for sex-linked differences," said Disteche.

The importance of escaped Xi genes is illustrated by Turner Syndrome. Turner syndrome results from the deletion of an entire copy of X chromosome. Females with Turner Syndrome have only one copy of the X chromosome (X0). Therefore, they have no expression from Xi escapees. This lack of Xi expression can result in a range of characteristics in females, including learning disabilities, small stature, delayed or diminished sexual development, and abnormalities in the kidney, heart, and other organs. "We have found high expression of escaped Xi genes in the ovary, so it makes sense that development of the ovary is deficient in individuals with Turner Syndrome," said Disteche. No males are born with Turner Syndrome; male embryos that lack an X chromosome (0Y) do not develop.

Disteche continues to research the molecular mechanisms of gene regulation on the X chromosome in different tissues including brain. Her laboratory is studying the mechanisms of X up-regulation, and escape from X inactivation. "We'd like to understand the characteristics of Xi genes that escape inactivation versus Xi genes that remain inactivated," says Disteche. "I think that understanding the mechanisms of any disorder can eventually help treatment to be better targeted. I think it's important to study the expression of these genes in the brain, X-linked versus Y-linked, to differentiate them and see what kind of role they play in development." ♦

Christine Disteche

Improving Inclusive Classrooms

A preschool teacher is trying to set out supplies for an art activity, but there's a book-sharing dispute in the reading corner, one three-year-old is having difficulty extracting herself from dress-up clothes, and a group of children want the teacher to see their big block tower. Then she notices the child with speech delay who's hovering near the blocks, wanting to play, too.

The teacher could use a variety of research-based strategies to help this child successfully join the group. But, first, she would have to be aware of these techniques. Then, she would need to know ways to integrate them into an already overloaded day. "And of course it's never just one child who has special needs. It's several. So the teacher has a large responsibility," said Susan Sandall, Ph.D., associate professor of special education and director of the Infant Toddler Program at the Experimental Education Unit (EEU).

Sandall develops and disseminates teaching strategies to ensure that children with developmental disabilities and other special needs receive the special instruction they need during the usual routine of a busy pre-school classroom. She and her colleagues train teachers using traditional classes and workshops, as well as developing and testing new training methods, such as those using technology to aid teachers in self-evaluation (see sidebar). Sandall is especially concerned with preparing teachers to use intervention strategies effectively in inclusive classrooms, which include children with and without developmental disabilities.

Children with disabilities in inclusive programs often make important gains in cognitive, physical, and language development. "They also have opportunities to develop friendships and the social skills that will be so important for their entire lives," said Sandall. Research shows that typically developing children in inclusive classrooms also learn important social skills through interacting with children who have a wide range of skills and interests.

Susan Sandall (left) and Janine Guanci (center), a masters student in speech-language pathology, guide and observe play in an inclusive classroom at the Experimental Education Unit. The children are Charlee (second from left), Ryan (second from right), and Zachary (far right).

Erika Edwards, a masters student in early childhood special education, encourages the play of Anna in an inclusive EEU classroom.

Sandall's goal is that children with developmental disabilities participate as fully in inclusive classrooms as possible. "Some children do need additional support and additional carefully planned instruction. But to the degree that we can, we don't want that additional support to be pull-aside or pull-out instruction. The goal is to embed planned instruction within typical classroom activities, such as free choice time."

During free choice periods children can play as they choose in areas such as a book corner, block area, or housekeeping station. "For many children, the environment itself is so enticing that they participate and learn," said Sandall. "They figure out what to do with those blocks and they arrange them and rearrange them and they play out story lines and they cooperate with their friends to make the tower or the castle bigger and better. But children who have disabilities may face barriers that interfere with their ability to participate in play, to take full advantage of these interesting, rich classroom environments that teachers develop. For example, a child with a developmental disability might just stand and watch the other children. Or a child may try to get involved, but is not precise in his or her movements and interferes with the other children's play." Another child may not know how or not yet be able to ask for a block. Teachers need to be able to assist children with all these issues, said Sandall.

Appropriate interventions can vary greatly. Some children may only need direction and encouragement from a teacher who says, for example, 'Hey, come with me and help them build that tower.' Or a teacher could use a peer-mediated strategy, asking one of the children who is already engaged in an activity to extend an invitation to a child who is not. A child with fine motor delays might need special equipment or reinforcement to attempt to stack blocks or paint with a brush. A child with language challenges may need prompts to make verbal requests of other children, such as 'Hey, remember, ask.'

"Then you have to repeat that same embedded instruction over and over again

because once is not going to be enough," said Sandall. In her work to develop new intervention strategies, she has begun to study how many instances of embedded instruction are needed for effective intervention. In one project, 80 percent of children who received at least ten instances of embedded instruction per preschool session showed significant progress in their target behaviors in just two weeks. Embedded instruction needs to occur in different activities as well as at different times throughout the preschool session, said Sandall. For example, to progress in language skills, a child who needs a prompt to make a verbal request in the block area should also receive encouragement to use language during crafts, snack time, and song time.

Sandall also stresses the importance of collecting data to guide interventions. If an objective is important enough to be in a child's individual education plan (IEP), teachers should consider collecting data to learn if their teaching plans are having an impact, said Sandall. It's easier to prepare and motivate teachers to collect data when the target behavior is clear and important and a system is in place for easy data collection. For example, many teachers are accustomed to noting toilet training progress on a classroom wall chart. "But it can be challenging to figure out how to collect the right kind of data on some of the social behaviors that we want children to obtain: playing with other children, taking turns, continuing a playful interaction," said Sandall.

"When we talk about early childhood learning for all children, if we really mean all children, then we need to prepare all teachers for inclusive classrooms."

- Susan Sandall

Teachers can feel overwhelmed at the prospect of collecting data on one child's interactions while simultaneously encouraging 12-18 other children. "But if we don't collect data, children can slip through the cracks," said Sandall. Data collection techniques, as well as embedded instruction and other inclusive practices, are important skills for all teachers of young children and should be covered in all teaching preparation programs for early childhood education, not just those focusing on special education, said Sandall. "When we talk about early childhood learning for all children, if we really mean all children, then we need to prepare all teachers for inclusive classrooms," said Sandall. ♦

Training Teachers in Inclusive Techniques

To help ensure the needs of all children are met in inclusive classrooms, Susan Sandall, Ph.D., works to translate research on evidence-based teaching practices and evaluation techniques into procedures teachers can use in their daily work. Currently, Sandall is an investigator in three research projects designed to improve teacher effectiveness in inclusive classrooms.

Improving the Data-based Decision-making of Preservice Early Intervention Teachers, initial funding by Cure Autism Now. Investigators are Sandall; Ilene Schwartz, Ph.D., professor of special education and EEU director; and Mark Harniss, Ph.D., clinical assistant professor of rehabilitation medicine. This program develops training methods to increase teachers' mastery of data-based decision making. The program is also studying technologies that might ease the task of data collection and interpretation. The program provides access to equipment so participating teachers can evaluate their performance by recording and viewing video of themselves with students. Participating teachers are also experimenting with the use of a smart pen that can upload data into a spreadsheet program and produce graphs.

Impact of Professional Development on Preschool Teachers' Use of Embedded-Instruction Practices, funded by The Institute of Education Sciences, US Department of Education (DOE). Sandall is an investigator in a multi-site grant which is studying techniques for in-service teacher training. The study's ultimate goal is to increase the effectiveness of teacher training in embedded instruction techniques. The grant is directed by Patricia Snyder, Ph.D., professor of education at the University of Florida.

Preparation of Teachers for Early Intervention/Early Childhood Special Education, funded by the Office of Special Education Programs, DOE. Sandall and Schwartz direct this program, designed to increase the number of highly qualified teachers to serve infants, toddlers, and preschoolers with developmental disabilities. Master's students in the Early Childhood Special Education program in the College of Education receive tuition waivers, training at the EEU, and post-graduate support and mentoring. Through late 2007, all of the program's 17 graduates were teaching in birth-to-three or preschool classrooms.

Research Center Name Change Honors Eunice Kennedy Shriver

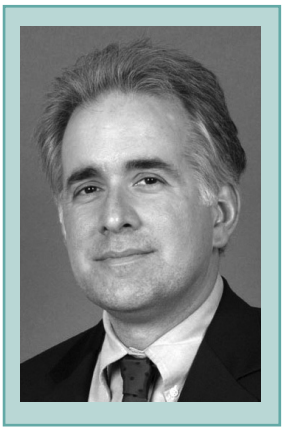
On March 3, 2008, the National Institute of Child Health and Human Development was renamed the Eunice Kennedy Shriver National Institute of Child Health and Human Development (NICHD) in recognition of Mrs. Shriver's critical role in founding the institute in 1963. In further recognition of Mrs. Shriver's significant interest in intellectual and developmental disabilities, the NICHD has renamed its 14 flagship research centers, which will now be known as the Eunice Kennedy Shriver Intellectual and Developmental Disabilities Research Center (IDDRCs). One of these centers composes one of the two major programs at the UW Center on Human Development and Disability (CHDD). This program's name has been changed from the Mental Retardation and Developmental Disabilities Research Center (MRDDRC) to the Eunice Kennedy Shriver Intellectual and Developmental Disabilities Research Center (IDDRC). ♦

Faculty Members Appointed as New CHDD Research Affiliates

Robert Andrews, M.D., an associate professor of pediatrics, studies the genetics of hematopoietic (blood-generating) stem cells. Currently, he researches factors that affect interactions of transplanted (allogeneic) CD34+ hematopoietic stem cells with progenitor cells. Hematopoietic stem cells generate all types of blood cells while progenitor cells are precursors of more specific types. His research may contribute to in-utero therapies for disorders that can cause developmental disabilities. For example, if transplanted CD34+ hematopoietic cells can be engrafted with progenitor cells in utero, they may be effective in treating leukodystrophies, rare degenerative brain disorders caused by defects in the growth or development of the myelin sheaths that insulate nerve fibers.

Annette Mercer Estes, Ph.D., is a research assistant professor of psychiatry and behavioral sciences. Her interests include the development of quantitative phenotypes for genetic studies of autism spectrum disorder (ASD) and changes in children with ASD throughout childhood. She also studies family adaptation and stress and the role of the family in promoting positive outcomes and reducing associated conditions in children with autism. Estes has recently begun studies with infant siblings and toddlers at risk for autism and is working to identify very early signs of autism risk and to test the effectiveness of very early intervention.

Nicholas Poolos, M.D., Ph.D., is an associate professor of neurology. He studies epilepsy, one of the most common causes of neurological disability in children. Current treatments do not reliably prevent seizures and associated neuronal damage in about 25 percent of individuals with epilepsy. In particular, Poolos and his colleagues study links between epilepsy and neuronal ion channel dysfunction. Dysfunctional ion channels can cause hyperexcitability in neurons, which in turn produces seizures. The team hopes to discover new molecular targets for antiepileptic drugs by dissecting the signaling pathways underlying ion channel dysfunction in epilepsy.



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