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Sociability traced to particular region of brain

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People with a genetic condition called Williams syndrome are famously gregarious. Scientists, looking carefully at brain function in individuals with Williams syndrome, think they may know why this is so. The researchers at the Stanford University School of Medicine showed that parts of a particular brain region known as the amygdala react more powerfully in Williams syndrome patients than in developmentally normal subjects — or in subjects with delays in development not caused by Williams syndrome — when exposed to facial expressions conveying positive emotions.

The study will be published Jan. 28 in the *Journal of Neuroscience*. Biopsychologist Brian Haas, PhD, a postdoctoral researcher at Stanford, shares first authorship of the study with Debra Mills, PhD, of Bangor University in Gwynned, Wales. Haas conducts research in the laboratory of Allan Reiss, MD, the Howard C. Robbins Professor of Psychiatry and Behavioral Sciences at Stanford, who is the paper's senior author. The work is part of an ongoing multicenter collaboration.

Williams syndrome, a rare genetic disorder affecting perhaps one in 10,000 individuals in whom a specific and well-defined chunk of DNA in one chromosome is missing, manifests in a distinctive pattern of physical and behavioral abnormalities including greatly reduced spatial and mathematical reasoning, but relatively less loss of certain verbal abilities or capacity to read others' emotions.

"If you give people with Williams syndrome a picture of a bicycle to

copy, they are able to draw the individual components of the bike — the wheels, the handlebars and so forth — but these components will be all over the page. It wouldn't look like a bike," said Haas. "But if you give them pictures of faces and ask them to describe the expressions, or ask them to talk about a story they've heard, they not only show just as much skill as you or I, but in some cases use even more socially and emotionally descriptive language. It's been speculated that they may even be better than the rest of us at picking up social information from facial expressions. We aimed to study the neurological underpinnings of social functioning in these people."

Sociability is one trait emphatically not lacking in people with Williams syndrome. On the contrary, they are invariably sociable — so much so that they will not uncommonly approach and strike up conversations with total strangers. Indeed, these individuals' famous gregariousness can be so pronounced as to occasionally place them in harm's way.

The investigators reasoned that the link between stereotypical sociability associated with Williams syndrome and the characteristic genetic deletion causing the condition might be mediated by a region deep within the brain called the amygdala. This almond-shaped, peanut-sized structure is known to be key to social and emotional processing — reading facial expressions or voices, for example. Lesions of the amygdala can cause a person to lose the ability to make quick "friend or foe" assessments, which have undoubtedly had life-or-death implications in human evolution.

Using two different techniques for objectively measuring brain response, the team showed that when 14 individuals with Williams syndrome looked at photos of faces judged by an independent team of normal reviewers to be especially reflective of a positive emotional state — like happiness — their amygdalas responded much more forcefully than did those of 13 age-matched normally developing subjects. One of those

techniques, functional magnetic resonance imaging, was able to localize the increased activity to specific nerve clusters in the amygdala, while another technique, involving monitoring the brain's electrical signals with a device placed on subjects' heads, charted the course of this activity over time.

Earlier work by others had already shown that Williams syndrome patients' amygdalas respond less vigorously to negatively charged stimuli (such as a face exhibiting fear) than do those of developmentally normal subjects. The new study both confirmed that finding and showed, for the first time, that exposure to a positive facial expression triggers a jump in signaling within the amygdala in these individuals, but not in healthy control subjects.

To rule out the possibility that the different response merely reflected IQ differences between normal and Williams syndrome subjects, the researchers also compared the latter with 15 other developmentally challenged subjects whose IQs matched those of the Williams syndrome group. They saw the same result here, as well.

Their reduced amygdala response to negatively charged facial expressions may provide a physiological basis for Williams syndrome patients' relative lack of reticence about approaching and engaging strangers, said Haas. Likewise, he said, the heightened response to positively charged facial expressions suggests that exposure to these smiling faces may be profoundly rewarding to these patients and, therefore, enhance their sociability. It may also mean that social rather than, say, monetary rewards may prove to be better incentives for training persons with the syndrome to compensate for their deficits, Haas speculated.

This differential processing in the amygdala appears to have its roots in Williams syndrome's defining DNA deletion, implying a genetic basis

for the difference — and, perhaps, for individual differences among normal people.

In some ways, both the deficits and strengths of the syndrome are the polar opposites of those that typify the far more common syndrome called autism, in which mathematical and visuospatial skills may be not only unimpaired but sometimes pronouncedly enhanced, while functions such as eye contact or gregarious behavior are markedly diminished. But in contrast to Williams syndrome, which is always attributable to the same distinct genetic lesion, "autism is an umbrella covering many different conditions with similar symptoms but a wide range of causes," said Reiss, complicating brain-function analysis of the sort his lab is doing. However, he said, another genetic disorder, called Fragile X syndrome, stems from a genetic abnormality as distinctive and well-characterized as Williams syndrome but often produces the symptoms of autism.

Reiss has applied for funding to study and compare brain function in very young children with these two conditions, in the hope of determining how the loss of specific genes results in specific behavioral changes, how and where these changes are mediated in the brain and what role the environment plays in modifying outcome.

This research holds implications for neurologically normal individuals, too. Studies have shown that more-extroverted people's amygdalas are more responsive to happy faces than less-extroverted people's are, suggesting the involvement of physiological hardwiring in shaping personality traits. "The more we understand about what makes us more or less social beings," said Reiss, "the better we may be able to tolerate one another's differences."

Source: Stanford University Medical Center