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The Gregarious Brain

By DAVID DOBBS

If a person suffers the small genetic accident that creates Williams syndrome, he'll live with not only some fairly conventional cognitive deficits, like trouble with space and numbers, but also a strange set of traits that researchers call the Williams social phenotype or, less formally, the "Williams personality": a love of company and conversation combined, often awkwardly, with a poor understanding of social dynamics and a lack of social inhibition. The combination creates some memorable encounters. Oliver Sacks, the neurologist and author, once watched as a particularly charming 8-year-old Williams girl, who was visiting Sacks at his hotel, took a garrulous detour into a wedding ceremony. "I'm afraid she disrupted the flow of this wedding," Sacks told me. "She also mistook the bride's mother for the bride. That was an awkward moment. But it very much pleased the mother."

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Another Williams encounter: The mother of twin Williams boys in their late teens opened her door to find on her stoop a leather-clad biker, motorcycle parked at the curb, asking for her sons. The boys had made the biker's acquaintance via C.B. radio and invited him to come by, but they forgot to tell Mom. The biker visited for a spell. Fascinated with how the twins talked about their condition, the biker asked them to speak at his motorcycle club's next meeting. They did. They told the group of the genetic accident underlying Williams, the heart and vascular problems that eventually kill many who have it, their intense enjoyment of talk, music and story, their frustration in trying to make friends, the slights and cruelties they suffered growing up, their difficulty understanding the world. When they finished, most of the bikers were in tears.

These stories are typical of those who have Williams syndrome. (Some people with the disorder as well as many who work with them simply call it Williams.) Williams syndrome rises from a genetic accident during meiosis, when [DNA](#)'s double helix is divided into two separate strands, each strand then becoming the genetic material in egg or sperm. Normally the two strands part cleanly, like a zipper's two halves. But in Williams, about 25 teeth in one of the zippers — 25 genes out of 30,000 in egg or sperm — are torn loose during this parting. When that strand joins another from the other parent to eventually form an embryo, the segment of the DNA missing those 25 genes can't do its work.

The resulting cognitive deficits lie mainly in the realm of abstract thought. Many with Williams have so vague a concept of space, for instance, that even as adults they will fail at six-piece jigsaw puzzles, easily get lost, draw like a preschooler and struggle to replicate a simple T or X shape built with a half-dozen building blocks. Few can balance a checkbook. These deficits generally erase about 35 points from whatever I.Q. the person would have inherited without the deletion. Since the average I.Q. is 100, this leaves most people with Williams with I.Q.'s in the 60s. Though some can hold simple jobs, they require assistance managing their lives.

The low I.Q., however, ignores two traits that define Williams more distinctly than do its deficits: an exuberant gregariousness and near-normal language skills. Williams people talk a lot, and they talk with pretty much anyone. They appear to truly lack social fear. Indeed, functional brain scans have shown that the brain's main fear processor, the amygdala, which in most of us shows heightened activity when we see angry or worried faces, shows no reaction when a person with Williams views such faces. It's as if they see all faces as friendly.

People with Williams tend to lack not just social fear but also social savvy. Lost on them are many meanings, machinations, ideas and intentions that most of us infer from facial expression, body language, context and stock phrasings. If you're talking with someone with Williams syndrome and look at your watch and say: "Oh, my, look at the time! Well it's been awfully nice talking with you . . .," your conversational partner may well smile brightly, agree that "this is nice" and ask if you've ever gone to Disney World. Because of this — and because many of us feel uneasy with people with cognitive disorders, or for that matter with anyone profoundly unlike us — people with Williams can have trouble deepening relationships. This saddens and frustrates them. They know no strangers but can claim few friends.

This paradox — the urge to connect, the inability to fully do so — sits at the center of the Williams puzzle, whether considered as a picture of human need (who hasn't been shut out of a circle he'd like to join?) or, as a growing number of researchers are finding, a clue to the fundamental drives and tensions that shape social behavior. After being ignored for almost three decades, Williams has recently become one of the most energetically researched neurodevelopmental disability after [autism](#), and it is producing more compelling insights. Autism, for starters, is a highly diverse "spectrum disorder" with ill-defined borders, no identified mechanism and no clearly delineated genetic basis. Williams, in contrast, arises from a known genetic cause and produces a predictable set of traits and behaviors. It is "an experiment of nature," as the title of one paper puts it, perfect for studying not just how genes create intelligence and sociability but also how our powers of thought combine with our desire to bond to create complex social behavior — a huge arena of interaction that largely determines our fates.

Julie R. Korenberg, a neurogeneticist at Cedars-Sinai Medical Center and at the [University of California](#), Los Angeles, who has helped define the Williams deletion and explore its effects, believes the value of Williams syndrome in examining such questions is almost impossible to overstate. "We've long figured that major

behavioral traits rose in indirect fashion from a wide array of genes,” Korenberg says. “But here we have this really tiny genetic deletion — of the 20-some-odd genes missing, probably just 3 to 6 create the cognitive and social effects — that reliably creates a distinctive behavioral profile. Williams isn’t just a fascinating mix of traits. It is the most compelling model available for studying the genetic bases of human behavior.”

Korenberg’s work is part of a diverse research effort on Williams that is illuminating a central dilemma of human existence: to survive we must relate and work with others, but we must also compete against them, lest we get left behind. It’s like the TV show “Survivor”: we want to keep a place in the group — we must — and doing so requires not only charming others but also showing we can contribute to their success. This requires a finely calibrated display of smarts, savvy, grit and hustle. Show too little, and you’re voted off the island for being subpar. Show too much, and you’re ousted as a conniving threat.

Where is the right balance? A partial answer lies in the mix of skills, charms and deficiencies that is Williams syndrome.

Williams syndrome was first identified in 1961 by Dr. J. C. P. Williams of New Zealand. Williams, a cardiologist at Greenlane Hospital in Auckland, noticed that a number of the hospital’s young cardiac patients were small in stature, had elfin facial features and seemed friendly but in some ways were mentally slow. His published delineation of this syndrome put Dr. Williams on the map — off which he promptly and mysteriously fell. Twice offered a position at the prestigious [Mayo Clinic](#) in Rochester, Minn., he twice failed to show, disappearing the second time, in the late ’60s, from London, his last known location, with the only trace an unclaimed suitcase later found in a luggage office.

The rarity of Williams syndrome — about 1 in 7,500 people have it, compared with about 1 in 150 for autism or 1 in 800 for Down syndrome — rendered it obscure. Unless they had the syndrome’s distinctive cardiovascular problems (which stem from the absence of the gene that makes blood vessels, heart valves and other tissue elastic and which even today limit the average lifespan of a person with Williams to around 50), most people with Williams were simply considered “mentally retarded.”

This ended in the late 1980s, when a few researchers in the emerging field of cognitive neuroscience began to explore Williams. Among the most earnest was Ursula Bellugi, the director of the Laboratory for Cognitive Neuroscience at the Salk Institute for Biological Studies in La Jolla, Calif. Bellugi, who specializes in the neurobiology of language, was drawn to the linguistic strength that many Williamses displayed in the face of serious cognitive problems. The first person with Williams she met, in fact, came by referral from the linguist [Noam Chomsky](#).

“The mother of that Williams teenager later connected me with two more, both in their teens,” Bellugi said. “I didn’t have to talk to them long to realize something special was going on. Here they had these great cognitive deficits. Yet they spoke with the most ardent and delightful animation and color.”

To understand this uneven cognitive profile, Bellugi gave an array of language and cognitive tests to three groups: Williams children and teenagers, Down syndrome kids with similar I.Q.’s and developmentally average peers. “We would do these warm-up interviews to get to know them, ask about their families,” said Bellugi, who, less than five feet tall and with a ready smile and an animated manner, is somewhat elfin and engagingly gregarious herself. “Only, the Williams kids would turn the tables. They’d tell you how pretty you look or ask, ‘Do you like

opera?’ They would ornament their answers in a way other kids didn’t. For instance, you’d ask an adolescent, ‘What if you were a bird?’ The Down kids said things like: ‘I’m not a bird. I don’t fly.’ The Williams teens would say: ‘Good question! I’d fly through the air being free. If I saw a boy I’d land on his head and chirp.’”

Bellugi found that this fanciful verbosity was accompanied by infectious affability. To measure it she developed a questionnaire and gave it to parents of Williams, Down and normal children. It asked about things like friendliness toward strangers, connections to familiar people, different social scenarios. At every age level, those with Williams scored significantly higher in sociability than those in the other groups. Having long studied the human capacity for language and its biological basis, Bellugi assumed that some extraordinary urge to use language drove this hypersociability: “The language just seemed to be erupting out of them.”

Then she attended a meeting of Williams families that included infants and toddlers. “That was about a year into my research project,” she says. “The room was full of little ones — babies, toddlers who weren’t speaking yet. And when I came in the room all the young children old enough to walk ran to the door to greet me. No clinging to Mom; they just broke away. And when I would talk to mothers holding infants — literally babes in arms — some of these babies would almost dive out of their mothers’ arms to meet me.

“I knew then I was wrong. The language wasn’t driving the sociability. If anything, it was the other way around.”

Developmental psychologists sometimes call the social urge the “drive to affiliate.” It seemed clear early on that the Williams deletion, which was definitively identified in the mid-1990s, either strengthened this drive or left it unfettered. But how do missing genes steer behavior toward gregariousness and engagement? How can a deletion heighten a trait rather than diminish it?

I got a hint when I met Nicki Hornbaker, who is 19, at Bellugi’s office in La Jolla. Nicki, whose Williams was diagnosed when she was 2, has been participating as a subject in Bellugi’s research for 15 years. She and her mother, Verna, drove down from Fresno that day to continue testing and to talk with me about living with Williams syndrome. Like most people with Williams, Nicki loves to talk but has trouble getting past a cocktail-party-level chatter. Nicki, however, has fashioned at least a partial solution.

“Ever since she was tiny,” Verna Hornbaker told me, “Nicki has always especially loved to talk to men. And in the last few years, by chance, she figured out how to do it. She reads the sports section in the paper, and she watches baseball and football on TV, and she has learned enough about this stuff that she can talk to any man about what the 49ers or the Giants are up to. My husband gets annoyed when I say this, but I don’t mean it badly: men typically have that superficial kind of conversation, you know — weather and sports. And Nicki can do it. She knows what team won last night and where the standings are. It’s only so deep. But she can do it. And she can talk a good long while with most men about it.”

In the view of two of Bellugi’s frequent collaborators, Albert Galaburda, a Harvard Medical School professor of neurology and neuroscience, and Allan Reiss, a neuroscientist at the Stanford School of Medicine, Nicki’s learned facility at sports talk illustrates a central lesson of Williams and, for that matter, modern genetics: genes (or their absence) do not hard-wire people for certain behaviors. There is no gene for understanding calculus. But genes do shape behavior and personality, and they do so by creating brain structures and functions that favor certain abilities and appetites more than others.

Reiss and Galaburda's imaging and autopsy work on Williamses' brains, for instance, has shown distinct imbalances in structure and synaptic connectivity. This work has led Galaburda to suspect that some of the genes missing in the Williams deletion are "patterning genes," which direct embryonic development and which in this case dictate brain formation. Work in lab animals has shown that at least one patterning gene choreographs the developmental balance between the brain's dorsal areas (along the back and the top of the brain) and ventral areas (at the front and bottom). The dorsal areas play a strong role in vision and space and help us recognize other peoples' intentions; ventral areas figure heavily in language, processing sounds, facial recognition, emotion, music enjoyment and social drive. In an embryo's first weeks, Galaburda says, patterning genes normally moderate "a sort of turf war going on between these two areas," with each trying to expand. The results help determine our relative strengths in these areas. We see them in our S.A.T. scores, for example: few of us score the same in math (which draws mostly on dorsal areas) as in language (ventral), and the discrepancy varies widely. The turf war is rarely a draw.

In Williams the imbalance is profound. The brains of people with Williams are on average 15 percent smaller than normal, and almost all this size reduction comes from underdeveloped dorsal regions. Ventral regions, meanwhile, are close to normal and in some areas — auditory processing, for example — are unusually rich in synaptic connections. The genetic deletion predisposes a person not just to weakness in some functions but also to relative (and possibly absolute) strengths in others. The Williams newborn thus arrives facing distinct challenges regarding space and other abstractions but primed to process emotion, sound and language.

This doesn't mean that specific behaviors are hard-wired. [M.I.T.](#) math majors aren't born doing calculus, and people with Williams don't enter life telling stories. As Allan Reiss put it: "It's not just 'genes make brain make behavior.' You have environment and experience too."

By environment, Reiss means less the atmosphere of a home or a school than the endless string of challenges and opportunities that life presents any person starting at birth. In Williams, he says, these are faced by someone who struggles to understand space and abstraction but readily finds reward listening to speech and looking at faces. As the infant and toddler seeks and prolongs the more rewarding experiences, already-strong neural circuits get stronger while those in weaker areas may atrophy. Patterns of learning and behavior follow accordingly.

"Take the gaze," Reiss told me. Everyone who has worked with Williams children knows the Williams gaze, which in toddlers is often an intense, penetrating eye contact of the sort described as "boring right through you." The gaze can seem like a hard-wired expression of a Williams's desire to connect. Yet the gaze can also be seen as a skill learned at the end of the horrible colic that many Williams infants suffer during their first year and before they start to talk well. This window is longer than that for most infants, as Williams children, oddly, start talking a year or so later than most children. It's during this window that the gaze is at its most intense. Until she was 9 months old, for instance, Nicki Hornbaker rarely slept more than an hour at a time, and when she was quiet she tended to look vaguely at her mother's hairline. Then her colic stopped, she started sleeping and "almost overnight," her mother told me, "she became a happy, delightful, extremely social child, and she couldn't get enough eye contact." Later, when talk gave Nicki a more effective way to connect, the intensity of the eye contact eased. Nicki's eyes now meet yours, warm and engaging, but they don't bore through you.

To Reiss, the gaze is one of several things Williams people learn in order to pursue social connections. "They want that connection," he said, "and they learn all these things to get it: the gaze and the gregariousness, the smiles and

language and narrative skills, in succession as they're able to. What they learn is shaped by the inclinations and abilities their genes create.

“Look at the difference between Williams kids and fragile X.” Fragile X, another developmental syndrome, produces similar cognitive defects but a pronounced social reticence or aversion to looking at faces. If a Williams wants to lock eyes, a fragile X child will literally twist himself sideways to avoid eye contact. “Nothing could be more different from a Williams,” Reiss continued. “But the thing is, fragile X kids don't do that when they're a year old. They'll still look at you at that age. And Williams kids don't have that intense gaze yet at that age. It's only over the next year or two that they take this incredible divergence. In both cases you have a genetically inclined pattern of behavior that is reinforced.”

This is a genetic version of Bellugi's observation that sociability drives language. The child gravitates toward the pathways that offer smoother going or more interesting experiences — at least until she finds other pathways more rewarding (sports talk, for example). In fragile X, those pathways tend to keep a child close to himself. In Williams they lead headlong toward others.

As an experiment of nature, Williams syndrome makes clear that while we are innately driven to connect with others, this affiliative drive alone will not win this connection. People with Williams rarely win full acceptance into groups other than their own. To bond with others we must show not just charm but sophisticated cognitive skills. But why? For vital relationships like those with spouses or business partners, the answer seems obvious: people want to know you can contribute. But why should casual friendships and group membership depend on smarts?

One possible answer comes from the rich literature of nonhuman primate studies. For 40 years or so, primatologists like Jane Goodall, Frans de Waal and Robert Sapolsky have been studying social behavior in chimps, gorillas, macaques, bonobos and baboons. Over the past decade that work has led to a unifying theory that explains not only a huge range of behavior but also why our brains are so big and what their most essential work is. The theory, called the Machiavellian-intelligence or social-brain theory, holds that we rise from a lineage in which both individual and group success hinge on balancing the need to work with others with the need to hold our own — or better — amid the nested groups and subgroups we are part of.

It started with fruit. About 15 or 20 million years ago, the theory goes, certain forest monkeys in Africa and Asia developed the ability to digest unripe fruit. This left some of their forest-dwelling cousins — the ancestors of chimps, gorillas and humans — at a sharp disadvantage. Suddenly a lot of fruit was going missing before it ripened.

To find food, some of the newly hungry primate species moved to the forest edge. Their new habitat put more food in reach, but it also placed the primates within reach of big cats, canines and other savanna predators. This predation spurred two key evolutionary changes. The primates became bigger, giving individuals more of a fighting chance, and they started living in bigger groups, which provided more eyes to keep watch and a strength of numbers in defense.

But the bigger groups imposed a new brain load: the members had to be smart enough to balance their individual needs with those of the pack. This meant cooperating and exercising some individual restraint. It also required understanding the behavior of other group members striving not only for safety and food but also access to mates.

And it called for comprehending and managing one's place in an ever-shifting array of alliances that members formed in order not to be isolated within the bigger group.

How did primates form and manage these alliances? They groomed one another. Monkeys and great apes spend up to a fifth of their time grooming, mostly with regular partners in pairs and small groups. This quality time (grooming generates a pleasing release of endorphins and oxytocin) builds strong bonds. Experiments in which a recording of macaques screaming in alarm is played, for instance, have shown a macaque will respond much more strongly to a grooming partner's cries than to cries from other members of the group. The large time investment involved seems to make a grooming relationship worth defending.

In this and other ways a group's members would create, test and declare their alliances. But as the animals and groups grew, tracking and understanding all those relationships required more intelligence. According to the social-brain theory, it was this need to understand social dynamics — not the need to find food or navigate terrain — that spurred and rewarded the evolution of bigger and bigger primate brains.

This isn't idle speculation; Robin Dunbar, an evolutionary psychologist and social-brain theorist, and others have documented correlations between brain size and social-group size in many primate species. The bigger an animal's typical group size (20 or so for macaques, for instance, 50 or so for chimps), the larger the percentage of brain devoted to neocortex, the thin but critical outer layer that accounts for most of a primate's cognitive abilities. In most mammals the neocortex accounts for 30 percent to 40 percent of brain volume. In the highly social primates it occupies about 50 percent to 65 percent. In humans, it's 80 percent.

According to Dunbar, no such strong correlation exists between neocortex size and tasks like hunting, navigating or creating shelter. Understanding one another, it seems, is our greatest cognitive challenge. And the only way humans could handle groups of more than 50, Dunbar suggests, was to learn how to talk.

"The conventional view," Dunbar notes in his book "Grooming, Gossip and the Evolution of Language," "is that language evolved to enable males to do things like coordinate hunts more effectively. . . . I am suggesting that language evolved to allow us to gossip."

Dunbar's assertion about the origin of language is controversial. But you needn't agree with it to see that talk provides a far more powerful and efficient way to exchange social information than grooming does. In the social-brain theory's broad definition, gossip means any conversation about social relationships: who did what to whom, who is what to whom, at every level, from family to work or school group to global politics. Defined this way, gossip accounts for about two-thirds of our conversation. All this yakking — murmured asides in the kitchen, gripefests in the office coffee room — yields vital data about changing alliances; shocking machinations; new, wished-for and missed opportunities; falling kings and rising stars; dangerous rivals and potential friends. These conversations tell us too what our gossipmates think about it all, and about us, all of which is crucial to maintaining our own alliances.

For we are all gossiped about, constantly evaluated by two criteria: Whether we can contribute, and whether we can be trusted. This reflects what Ralph Adolphs, a social neuroscientist at the [California Institute of Technology](#), calls the "complex and dynamic interplay between two opposing factors: on the one hand, groups can provide better security from predators, better mate choice and more reliable food; on the other hand, mates and food are available also to competitors from within the group." You're part of a team, but you're competing with team

members. Your teammates hope you'll contribute skills and intergroup competitive spirit — without, however, offering too much competition within the group, or at least not cheating when you do. So, even if they like you, they constantly assess your trustworthiness. They know you can't afford not to compete, and they worry you might do it sneakily.

Deception runs deep. In his book, "Our Inner Ape," Frans de Waal, a primatologist at [Emory University](#), describes a simple but cruel deception perpetrated by a female chimp named Puist. One day, Puist chases but cannot catch a younger, faster female rival. Some minutes later, writes de Waal, "Puist makes a friendly gesture from a distance, stretching out an open hand. The young female hesitates at first, then approaches Puist with classic signs of mistrust, like frequent stopping, looking around at others and a nervous grin on her face. Puist persists, adding soft pants when the younger female comes closer. Soft pants have a particularly friendly meaning; they are often followed by a kiss, the chimpanzee's chief conciliatory gesture. Then, suddenly, Puist lunges and grabs the younger female, biting her fiercely before she manages to free herself."

This "deceptive reconciliation offer," as de Waal calls it, is classic schoolyard stuff. Adult humans generally do a better job veiling a coming assault. The bigger the neocortex, the higher the rate of deceptive behavior. Our extra-big brains allow us to balance bonding and maneuvering in more subtle and complicated ways.

People with Williams, however, don't do this so well. Generating and detecting deception and veiled meaning requires not just the recognition that people can be bad but a certain level of cognitive power that people with Williams typically lack. In particular it requires what psychologists call "theory of mind," which is a clear concept of what another person is thinking and the recognition that the other person a) may see the world differently than you do and b) may actually be thinking something different from what he's saying.

Cognitive scientists argue over whether people with Williams have theory of mind. Williams people pass some theory-of-mind tests and fail others. They get many jokes, for instance, but don't understand irony. They make small talk but tend not to discuss the subtler dynamics of interpersonal relationships. Theory of mind is a slippery, multilayered concept, so the debate becomes arcane. But it's clear that Williamses do not generally sniff out the sorts of hidden meanings and intentions that lie behind so much human behavior. They would reach for Puist's outstretched hand without hesitation.

To inquire into human behavior's genetic underpinnings is to ask what most essentially defines us. One of the most vexing questions raised by both Williams research and the social-brain thesis is whether our social behavior is ultimately driven more by the urge to connect or the urge to manipulate the connection.

The traditional inclination, of course, is to distinguish essential human behavior by our "higher" skills and cognitive powers. We dominate the planet because we can think abstractly, accumulate and relay knowledge and manipulate the environment and one another. By this light our social behavior rises more from big brains than from big hearts.

Andreas Meyer-Lindenberg, a psychiatrist and neurologist, sees it differently. Meyer-Lindenberg spent the last several years at the National Institute of Mental Health exploring neural roots of mood, cognitive and behavioral disorders — including Williams syndrome, which he has investigated as part of a team led by Karen Berman, a N.I.M.H. psychiatrist, clinical neurobiologist and imaging specialist. Working with Berman and Carolyn Mervis, a developmental psychologist at the [University of Louisville](#), Meyer-Lindenberg became convinced that we may

be overvaluing the cerebral.

“Cognitive social neuroscience tends to be very top-down,” Meyer-Lindenberg says. “It looks at lofty things like triadic intentionality — I’m conscious of you being conscious of me being conscious of you, things like that. Things that presuppose consciousness and elaborate intellectual procedures.” The Berman group’s work, however, was focused on brain networks operating, as Meyer-Lindenberg puts it, “at a lower hierarchical level.”

“And the most important abnormalities in Williams,” he says, “are circuits that have to do with basic regulation of emotions.”

The most significant such finding is a dead connection between the orbitofrontal cortex, an area above the eye sockets and the amygdala, the brain’s fear center. The orbitofrontal cortex (or OFC) is associated with (among other things) prioritizing behavior in social contexts, and earlier studies found that damage to the OFC reduces inhibitions and makes it harder to detect faux pas. The Berman team detected a new contribution to social behavior: They found that while in most people the OFC communicated with the amygdala when viewing threatening faces, the OFC in people with Williams did not. This OFC-amygdala connection worked normally, however, when people with Williams viewed nonsocial threats, like pictures of snakes, sharks or car crashes.

This appears to explain the amygdala’s failure in Williams to fire at the sight of frightening faces and suggests a circuit responsible for Williamses’ lack of social caution. If the results hold up, the researchers will have cleanly defined a circuit evolved specifically to warn of threats from other people. This could account not just for the lack of social fear in Williams, but with it the wariness that can motivate deeper understanding. It is possible, in short, that people with Williams miss social subtleties not just because they lack cognitive tools but because they also lack a motivation — a fear of others — that the rest of us carry to every encounter. To Meyer-Lindenberg, the primacy of such circuits suggests that human sociability rises from evolutionarily reinforced mechanisms — a raw yearning to connect; fearfulness — that are so basic they’re easy to undervalue.

The disassociation of so many elements in Williams — the cognitive from the connective, social fear from nonsocial fear, the tension between the drive to affiliate and the drive to manipulate — highlights how vital these elements are and, in most of us, how delicately, critically entwined. Yet these splits in Williams also clarify which, of caring and comprehension, offers the more vital contribution. For if Williams confers disadvantage by granting more care than comprehension, reversing this imbalance creates a far more problematic phenotype.

As Robert Sapolsky of the Stanford School of Medicine puts it: “Williams have great interest but little competence. But what about a person who has competence but no warmth, desire or empathy? That’s a sociopath. Sociopaths have great theory of mind. But they couldn’t care less.”

David Dobbs writes frequently about science and medicine. His last article for the magazine was about depression.

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