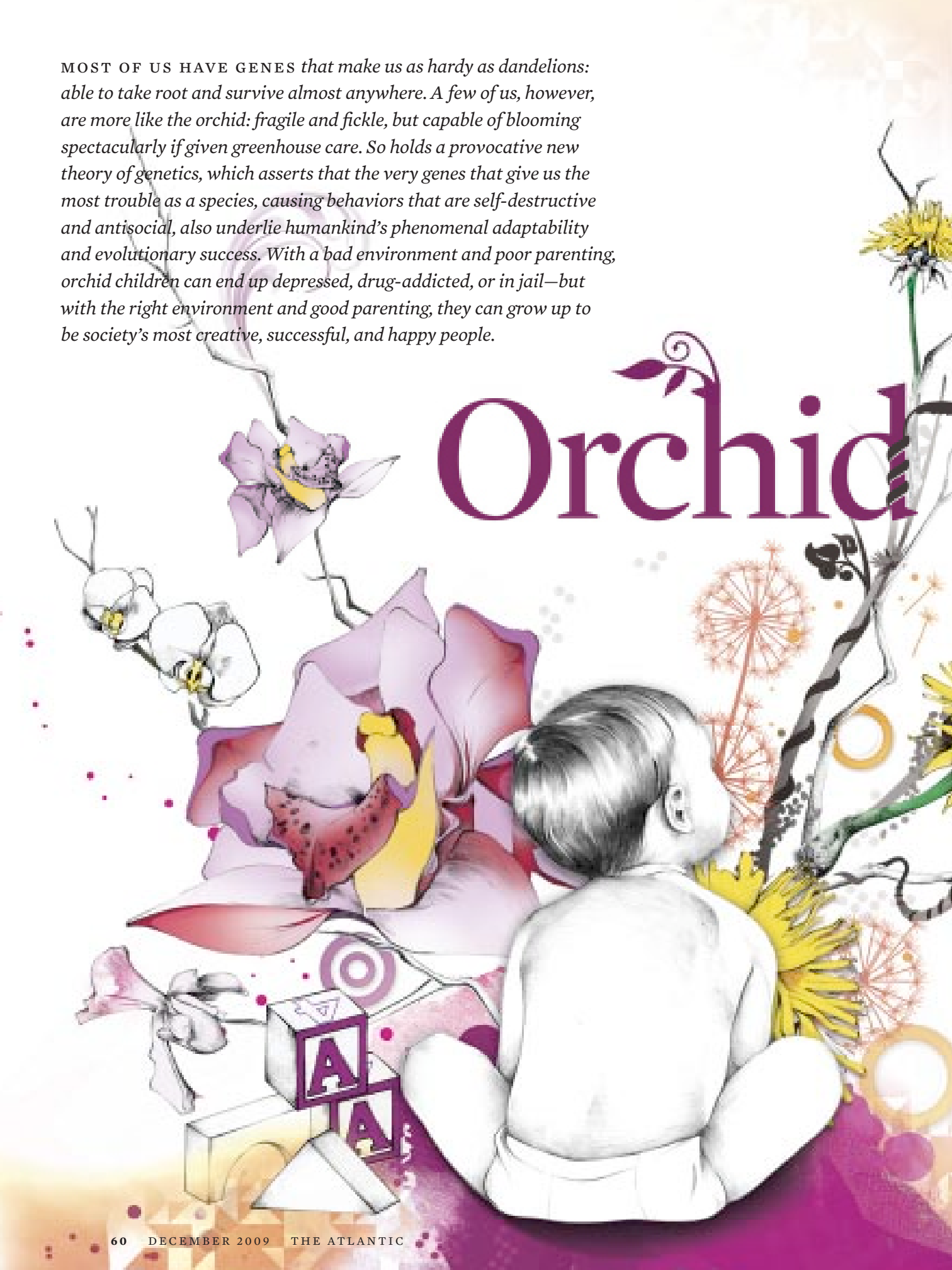
MOST OF US HAVE GENES *that make us as hardy as dandelions: able to take root and survive almost anywhere. A few of us, however, are more like the orchid: fragile and fickle, but capable of blooming spectacularly if given greenhouse care. So holds a provocative new theory of genetics, which asserts that the very genes that give us the most trouble as a species, causing behaviors that are self-destructive and antisocial, also underlie humankind's phenomenal adaptability and evolutionary success. With a bad environment and poor parenting, orchid children can end up depressed, drug-addicted, or in jail—but with the right environment and good parenting, they can grow up to be society's most creative, successful, and happy people.*

Orchid



Children

By David Dobbs

In 2004, Marian Bakermans-Kranenburg, a professor of child and family studies at Leiden University, started carrying a video camera into homes of families whose 1-to-3-year-olds indulged heavily in the oppositional, aggressive, uncooperative, and aggravating behavior that psychologists call "externalizing": whining, screaming, whacking, throwing tantrums and objects, and willfully refusing reasonable requests. Staple behaviors in toddlers, perhaps. But research has shown that toddlers with especially high rates of these behaviors are likely to become stressed, confused children who fail academically and socially in school, and become antisocial and unusually aggressive adults.

At the outset of their study, Bakermans-Kranenburg and her colleagues had screened 2,408 children via parental questionnaire, and they were now focusing on the 25 percent rated highest by their parents in externalizing behaviors. Lab observations had confirmed these parental ratings.

Bakermans-Kranenburg meant to change the kids' behavior. In an intervention her lab had developed, she or another researcher visited each of 120 families six times over eight months; filmed the mother and child in everyday activities, including some requiring obedience or cooperation; and then edited the film into teachable moments to show to the mothers. A similar group of high-externalizing children received no intervention.

To the researchers' delight, the intervention worked. The moms, watching the videos, learned to spot cues they'd missed before, or to respond differently to cues they'd seen but had reacted to poorly. Quite a few mothers, for instance, had agreed only reluctantly to read picture books to their fidgety, difficult kids, saying they wouldn't sit still for it. But according to Bakermans-Kranenburg, when these mothers viewed the playback they were "surprised to see how much pleasure it was for the child—and for them." Most mothers began reading to their children regularly, producing what Bakermans-Kranenburg describes as "a peaceful time that they had dismissed as impossible."

And the bad behaviors dropped. A year after the intervention ended, the toddlers who'd received it had reduced their externalizing scores by more than 16 percent, while a non-intervention control group improved only about 10 percent (as expected, due to modest gains in self-control with age). And the mothers' responses to their children became more positive and constructive.

ART/PHOTOGRAPHY CREDIT

Few programs change parent-child dynamics so successfully. But gauging the efficacy of the intervention wasn't the Leiden team's only goal, or even its main one. The team was also testing a radical new hypothesis about how genes shape behavior—a hypothesis that stands to revise our view of not only mental illness and behavioral dysfunction but also human evolution.

Of special interest to the team was a new interpretation of one of the most important and influential ideas in recent psychiatric and personality research: that certain variants of key behavioral genes (most of which affect either brain development or the processing of the brain's chemical messengers) make people more vulnerable to certain mood, psychiatric, or personality disorders. Bolstered over the past 15 years by numerous studies, this hypothesis, often called the "stress diathesis" or "genetic vulnerability" model, has come to saturate psychiatry and behavioral science. During that time, researchers have identified a dozen-odd gene variants that can increase a person's susceptibility to depression, anxiety, attention-deficit hyperactivity disorder, heightened risk-taking, and antisocial, sociopathic, or violent behaviors, and other problems—if, and only if, the person carrying the variant suffers a traumatic or stressful childhood or faces particularly trying experiences later in life.

This vulnerability hypothesis, as we can call it, has already changed our conception of many psychic and behavioral problems. It casts them as products not of nature or nurture but of complex "gene-environment interactions." Your genes don't doom you to these disorders. But if you have "bad" versions of certain genes and life treats you ill, you're more prone to them.

Recently, however, an alternate hypothesis has emerged from this one and is turning it inside out. This new model suggests that it's a mistake to understand these "risk" genes only as liabilities. Yes, this new thinking goes, these bad genes can create dysfunction in unfavorable contexts—but they can also enhance function in favorable contexts. The genetic sensitivities to negative experience that the vulnerability hypothesis has identified, it follows, are just the downside of a bigger phenomenon: a heightened genetic sensitivity to *all* experience.

"Bad" genes can create dysfunction—but also enhance function. Genetic sensitivities to negative experience are the downside of a heightened sensitivity to *all* experience.

The evidence for this view is mounting. Much of it has existed for years, in fact, but the focus on dysfunction in behavioral genetics has led most researchers to overlook it. This tunnel vision is easy to explain, according to Jay Belsky, a child-development psychologist at Birkbeck, University of London. "Most work in behavioral genetics has been done by mental-illness researchers who focus on vulnerability," he told me recently. "They don't see the upside, because they don't look for it. It's like dropping a dollar bill beneath a table. You look under the table, you see the dollar bill, and you grab it. But you completely miss the five that's just beyond your feet."

Though this hypothesis is new to modern biological psychiatry, it can be found in folk wisdom, as the University of Arizona developmental psychologist Bruce Ellis and the University of British Columbia developmental pediatrician W. Thomas Boyce pointed out last year in the journal *Current Directions in Psychological Science*. The Swedes, Ellis and Boyce noted in an essay titled "Biological Sensitivity to Context," have long spoken of "dandelion" children. These dandelion children—equivalent to our "normal" or "healthy" children, with "resilient" genes—do pretty well almost anywhere, whether raised in the equivalent of a sidewalk crack or a well-tended garden. Ellis and Boyce offer that there are also "orchid" children, who will wilt if ignored or maltreated but bloom spectacularly with greenhouse care.

At first glance, this idea, which I'll call the orchid hypothesis, may seem a simple amendment to the vulnerability hypothesis. It merely adds that environment and experience can steer a person up instead of down. Yet it's actually a completely new way to think about genetics and human behavior. Risk becomes possibility; vulnerability becomes plasticity and responsiveness. It's one of those simple ideas with big, spreading implications. Gene variants generally considered misfortunes (poor Jim, he got the "bad" gene) can instead now be understood as highly leveraged evolutionary bets, with both high risks and high potential rewards: gambles that help create a diversified-portfolio approach to survival, with selection favoring parents who happen to invest in both dandelions *and* orchids.

In this view, having both dandelion and orchid kids greatly raises a family's (and a species') chance of succeeding, over time and in any given environment. The behavioral diversity provided by these two different types of temperament also supplies precisely what a smart, strong species needs if it is to spread across and dominate a changing world. The many dandelions in a population provide an underlying stability. The less-numerous orchids, meanwhile, may falter in some environments but can excel in those that suit them. And even when they lead troubled early lives, some of the resulting heightened responses to adversity that can be problematic in everyday life—increased novelty-seeking, restlessness of attention, elevated risk-taking, or aggression—can prove advantageous in certain challenging situations: wars, tribal or modern; social strife of many kinds; and migrations to new environments. Together, the steady dandelions and the mercurial orchids offer an adaptive flexibility that neither can provide alone. Together, they open a path to otherwise unreachable individual and collective achievements.

This orchid hypothesis also answers a fundamental evolutionary question that the vulnerability hypothesis cannot. If variants of certain genes create mainly dysfunction and trouble, how have they survived natural selection? Genes so maladaptive should have been selected out. Yet about a quarter of all human beings carry the best-documented gene variant for depression, while more than a fifth carry the variant that Bakermans-Kranenburg studied, which is associated with externalizing, antisocial, and violent behaviors, as well

as ADHD, anxiety, and depression. The vulnerability hypothesis can't account for this. The orchid hypothesis can.

This is a transformative, even startling view of human frailty and strength. For more than a decade, proponents of the vulnerability hypothesis have argued that certain gene variants underlie some of humankind's most grievous problems: despair, alienation, cruelties both petty and epic. The orchid hypothesis accepts that proposition. But it adds, tantalizingly, that these same troublesome genes play a critical role in our species' astounding success.

The orchid hypothesis—sometimes called the plasticity hypothesis, the sensitivity hypothesis, or the differential-susceptibility hypothesis—is too new to have been tested widely. Many researchers, even those in behavioral science, know little or nothing of the idea. A few—chiefly those with broad reservations about ever tying specific genes to specific behaviors—express concerns. But as more supporting evidence emerges, the most common reaction to the idea among researchers and clinicians is excitement. A growing number of psychologists, psychiatrists, child-development experts, geneticists, ethologists, and others are beginning to believe that, as Karlen Lyons-Ruth, a developmental psychologist at Harvard Medical School, puts it, "It's time to take this seriously."

WITH THE DATA gathered in the video intervention, the Leiden team began to test the orchid hypothesis. Could it be, they wondered, that the children who suffer most from bad environments also profit the most from good ones? To find out, Bakermans-Kranenburg and her colleague Marinus van Ijzendoorn began to study the genetic makeup of the children in their experiment. Specifically, they focused on one particular "risk allele" associated with ADHD and externalizing behavior. (An allele is any of the variants of a gene that takes more than one form; such genes are known as polymorphisms. A risk allele, then, is simply a gene variant that increases your likelihood of developing a problem.)

Bakermans-Kranenburg and van Ijzendoorn wanted to see whether kids with a risk allele for ADHD and externalizing behaviors (a variant of a dopamine-processing gene known as DRD4) would respond as much to positive environments as to negative. A third of the kids in the study had this risk allele; the other two-thirds had a version considered a "protective allele," meaning it made them less vulnerable to bad environments. The control group, who did not receive the intervention, had a similar distribution.

Both the vulnerability hypothesis and the orchid hypothesis predict that in the control group the kids with a risk allele should do worse than those with a protective one. And so they did—though only slightly. Over the course of 18 months, the genetically "protected" kids reduced their externalizing scores by 11 percent, while the "at-risk" kids cut theirs by 7 percent. Both gains were modest ones that the researchers expected would come with increasing age. Although statistically significant, the difference between the two groups was probably unnoticeable otherwise.

The real test, of course, came in the group that got the intervention. How would the kids with the risk allele respond? According to the vulnerability model, they should improve less than their counterparts with the protective allele; the modest upgrade that the video intervention created in their environment wouldn't offset their general vulnerability.

As it turned out, the toddlers with the risk allele blew right by their counterparts. They cut their externalizing scores by almost 27 percent, while the protective-allele kids cut theirs by just 12 percent (improving only slightly on the 11 percent managed by the protective-allele population in the control group). The upside effect in the intervention group, in other words, was far larger than the downside effect in the control group. Risk alleles, the Leiden team concluded, really can create not just risk but possibility.

CAN LIABILITY REALLY be so easily turned to gain? The pediatrician W. Thomas Boyce, who has worked with many a troubled child in more than three decades of child-development research, says the orchid hypothesis "profoundly recasts the way we think about human frailty." He adds, "We see that when kids with this kind of vulnerability are put in the right setting, they don't merely do better than before, they do the *best*"—even better, that is, than their protective-allele peers. "Are there any enduring human frailties that don't have this other, redemptive side to them?"

As I researched this story, I thought about such questions a lot, including how they pertained to my own temperament and genetic makeup. Having felt the black dog's teeth a few times over the years, I'd considered many times having one of my own genes assayed—specifically, the serotonin-transporter gene, also called the SERT gene, or 5-HTTLPR. This gene helps regulate the processing of serotonin, a chemical messenger crucial to mood, among other things. The two shorter, less efficient versions of the gene's three forms, known as short/short and short/long (or S/S and S/L), greatly magnify your risk of serious depression—if you hit enough rough road. The gene's long/long form, on the other hand, appears to be protective.

In the end, I'd always backed away from having my SERT gene assayed. Who wants to know his risk of collapsing under pressure? Given my family and personal history, I figured I probably carried the short/long allele, which would make me at least moderately depression-prone. If I had it tested I might get the encouraging news that I had the long/long allele. Then again, I might find I had the dreaded, riskier short/short allele. This was something I wasn't sure I wanted to find out.

But as I looked into the orchid hypothesis and began to think in terms of plasticity rather than risk, I decided maybe I did want to find out. So I called a researcher I know in New York who does depression research involving the serotonin-transporter gene. The next day, FedEx left a package on my front porch containing a specimen cup. I spat into it, examined what I'd produced, and spat again. Then I screwed the cap tight, slid the vial into its little shipping tube, and put it back on the porch. An hour later, the FedEx guy took it away.

OF ALL THE EVIDENCE supporting the orchid-gene hypothesis, perhaps the most compelling comes from the work of Stephen Suomi, a rhesus-monkey researcher who heads a sprawling complex of labs and monkey habitats in the Maryland countryside—the National Institutes of Health's

Laboratory of Comparative Ethology. For 41 years, first at the University of Wisconsin and then, beginning in 1983, in the Maryland lab the NIH built specifically for him, Suomi has been studying the roots of temperament and behavior in rhesus monkeys—which share about 95 percent of our DNA, a number exceeded only in apes. Rhesus monkeys differ from humans in obvious and fundamental ways. But their close resemblance to us in crucial social and genetic respects reveals much about the roots of our own behavior—and has helped give rise to the orchid hypothesis.

Suomi learned his trade as a student and protégé of, and then a direct successor to, Harry Harlow, one of the 20th century's most influential and problematic behavioral scientists. When Harlow started his work, in the 1930s, the study of childhood development was dominated by a ruthlessly mechanistic behavioralism. The movement's leading figure in the United States, John Watson, considered mother love "a dangerous instrument." He urged parents to leave crying babies alone; to never hold them to give pleasure or comfort; and to kiss them only occasionally, on the forehead. Mothers were important less for their affection than as conditioners of behavior.

With a series of ingenious but sometimes disturbingly cruel experiments on monkeys, Harlow broke with this cool behavioralism. His most famous experiment showed that baby rhesus monkeys, raised alone or with same-age peers, preferred a foodless but fuzzy terrycloth surrogate "mother" over a wire-mesh version that freely dispensed meals. He showed that these infants desperately wanted to bond, and that depriving them of physical, emotional, and social attachment could create a near-paralyzing dysfunction. In the 1950s this work provided critical evidence for the emerging theory of infant attachment: a theory that, with its emphasis on rich, warm parent-child bonds and happy early experiences, still dominates child-development theory (and parenting books) today.

In the years since Suomi took over Harlow's Wisconsin lab as a 28-year-old wunderkind, he has both broadened and sharpened the inquiry Harlow started. New tools now let Suomi examine not just his monkeys' temperaments but also the physiological and genetic underpinnings of their behavior. His lab's naturalistic environment allows him to focus not just on mother-child interactions but also on the family and social environments that shape and respond to the monkeys' behavior. "Life in a rhesus-monkey colony is very, very complicated," Suomi says. The monkeys must learn to navigate a social system that is highly nuanced and hierarchical. "Those who can manage this, do well," Suomi told me. "Those who don't, don't."

Rhesus monkeys typically mature at about four or five years and live to about 20 in the wild. Their development parallels our own at a fairly neat 1-to-4 ratio: a 1-year-old monkey is much like a 4-year-old human being, a 4-year-old monkey is like a 16-year-old human being, and so on. A mother typically gives birth annually, starting at around age 4. Though the monkeys copulate all year, the females' fertility seasons are only a couple of months long. Since they tend to occur together, a troop usually produces crops of babies that have same-age peers.

For the first month, the mother keeps the baby attached to her or within arm's reach. At about two weeks, the baby starts to explore, at first within only a few feet of its mother. These forays grow in frequency, duration, and distance over the next six to seven months, but rarely do the babies pass out of the mother's sight line or earshot. If the young monkey gets frightened, it scampers back to the mother. Often she'll see trouble coming and pull the infant close.

When the monkey is about eight months old—a rhesus preschooler—its mother's mating time arrives. Anticipating another child, the mother allows the youngster to spend more and more time with its cousins, with older siblings in the maternal line, and with occasional visitors from other families or troops. The youngster's family group, friends, and allies still provide protection when necessary.

A maturing female will stay with this group all her life. A male, however, will leave—often under pressure from the females as he gets rowdier and rougher—when he's 4 or 5, or roughly the equivalent of a 16-to-20-year-old person. At first he'll join an all-male gang that lives more or less separately. After a few months to a year, he'll leave the gang and try to charm, push, or sidle his way into a new family or troop. If he succeeds, he becomes one of several adult males to serve as mate, companion, and muscle for the several females. But only about half the males make it that far. Their transition period exposes them to attacks from other young males, attacks from rival gangs, attacks from new troop members if they play their cards wrong, and predation during any time they lack a gang's or troop's protection. Many die in the transition.

Very early in his work, Suomi identified two types of monkeys that had trouble managing these relations. One type, which Suomi calls a "depressed" or "neurotic" monkey, accounted for about 20 percent of each generation. These monkeys are slow to leave their mothers' sides when young. As adults they remain tentative, withdrawn, and anxious. They form fewer bonds and alliances than other monkeys do.

The other type, generally male, is what Suomi calls a "bully": an unusually and indiscriminately aggressive monkey. These monkeys accounted for 5 to 10 percent of each generation. "Rhesus monkeys are fairly aggressive in general, even when young," Suomi says, "and their play involves a lot of rough-and-tumble. But usually no one gets hurt—except with these guys. They do stupid things most other monkeys know not to. They repeatedly confront dominant monkeys. They get between moms and their kids. They don't know how to calibrate their aggression, and they don't know how to read signs they should back off. Their conflicts tend to always escalate." These bullies also score poorly in tests of monkey self-control. For instance, in a "cocktail hour" test that Suomi sometimes uses, monkeys get unrestricted access to a neutral-tasting alcoholic drink for an hour. Most monkeys have three or four drinks and then stop. The bullies, Suomi says, "drink until they drop."

The neurotics and the bullies meet quite different fates. The neurotics mature late but do okay. The females become jumpy mothers, but how their children turn out depends on the environment in which the mothers raise them. If it's secure, they become more or less normal; if it's insecure, they become jumpy too. The males, meanwhile, stay within their

Four of approximately 400 rhesus monkeys that Stephen Suomi works with at the National Institutes of Health

mothers' family circles an unusually long time—up to eight years. They're allowed to do so because they don't make trouble. And their longer stay lets them acquire enough social savvy and diplomatic deference so that when they leave, they usually work their way into new troops more successfully than do males who break away younger. They don't get to mate as prolifically as more confident, more assertive males do; they seldom rise high in their new troops; and their low status can put them at risk in conflicts. But they're less likely to die trying to get in the door. They usually survive and pass on their genes.

The bullies fare much worse. Even as babies and youths, they seldom make friends. And by the time they're 2 or 3, their extreme aggression leads the troop's females to simply run them out, by group force if necessary. Then the male gangs reject them, as do other troops. Isolated, most of them die before reaching adulthood. Few mate.

Suomi saw early on that each of these monkey types tended to come from a particular type of mother. Bullies came from harsh, censorious mothers who restrained their children from socializing. Anxious monkeys came from anxious, withdrawn, distracted mothers. The heritages were pretty clear-cut. But how much of these different personality types passed through genes, and how much derived from the manner in which the monkeys were raised?

To find out, Suomi split the variables. He took nervous infants of nervous mothers—babies who in standardized newborn testing were already jumpy themselves—and gave them to especially nurturing "supermoms." These babies turned out very close to normal. Meanwhile, Dario Maestripieri of the University of Chicago took secure, high-scoring infants from secure, nurturing mothers and had them raised by abusive mothers. This setting produced nervous monkeys.

The lesson seemed clear. Genes played a role—but environment played an equally important one.

WHEN TOOLS FOR the study of genes first became available, in the late 1990s, Suomi was quick to use them to more directly examine the balance between genes and environment in shaping his monkeys' development. He almost immediately struck gold, with a project he started in 1997 with Klaus-Peter Lesch, a psychiatrist from the University of Würzburg. The year before, Lesch had published data revealing, for the first time, that the human serotonin-transporter gene had three variants (the previously mentioned short/short, short/long, and long/long alleles) and that the two shorter versions magnified risk for depression, anxiety, and other problems. Asked to genotype Suomi's monkeys, Lesch did so. He found that they had the same three variants, though the short/short form was rare.

Suomi, Lesch, and NIH colleague J. Dee Higley set about doing a type of study now recognized as a classic "gene-by-environment" study. First they took cerebral spinal fluid from 132 juvenile rhesus monkeys and analyzed it for a serotonin metabolite, called 5-HIAA, that's considered a reliable indicator of how much serotonin the nervous system is processing. Lesch's studies had already shown that depressed people with the short-long serotonin-transporter allele had lower 5-HIAA levels, reflecting less-efficient serotonin processing. He and Suomi wanted to see if the finding would hold true in monkeys. If it did, it would provide more evidence for the genetic dynamic shown in Lesch's studies. And

finding such a dynamic in rhesus monkeys would confirm their value as genetic and behavioral models for studying human behavior.

After Suomi, Lesch, and Higley had grouped the monkeys' 5-HIAA levels according to their serotonin genotype (short/long or long/long, but not short/short, which was too rare to be of use), they also sorted the results by whether the monkeys had been raised by their mothers, or as orphans with only same-aged peers. When their colleague Allison Bennett charted the results on a bar graph showing 5-HIAA levels, all of the mother-reared monkeys, no matter which allele they had, showed serotonin processing in the normal range. The metabolite levels of the peer-raised monkeys, however, diverged sharply by genotype: the short/long monkeys in that group processed serotonin highly inefficiently (a risk factor for depression and anxiety), whereas the long/long monkeys processed it robustly. When Suomi saw the results, he realized that he finally had proof of a behaviorally relevant gene-by-environment interaction in his monkeys. "I took one look at that graph," he told me, "and said, 'Let's go pop some champagne.'"

Suomi and Lesch published their results in 2002 in *Molecular Psychiatry*, a relatively new journal about behavioral genetics. The paper formed part of a surge of gene-by-environment studies of mood and behavioral disorders. That same year, two psychologists at King's College, London, Avshalom Caspi and Terrie Moffitt, published the first of two large longitudinal studies (both drawing on life histories of hundreds of New Zealanders) that would prove particularly influential. The first, published in *Science*, showed that the short allele of another major neurotransmitter-processing gene (known as the MAOA gene) sharply increased the chance of antisocial behavior in human adults who'd been abused as children. The second, in 2003 and also in *Science*, showed that people with short/short or short/long serotonin-transporter alleles, if exposed to stress, faced a higher-than-normal risk of depression.

These and dozens of similar studies were critical to establishing the vulnerability hypothesis during the mid-2000s. Yet many of these studies also contained data that supported the orchid hypothesis—but went unnoticed or unremarked at the time. (Jay Belsky, the child-development psychologist, has recently documented more than two dozen such studies.) Both of Caspi and Moffitt's seminal papers in *Science*, for example, contain raw data and graphs showing that for people who did *not* face severe or repeated stress, the risk alleles in question heightened *resistance* to aggression or depression. And the data in Suomi and Lesch's 2002 *Molecular Psychiatry* paper, in which peer-reared monkeys with the risky serotonin-transporter allele appeared to process serotonin inefficiently, also showed that mother-reared infants with that same allele processed serotonin 10 percent *more* efficiently than even mother-raised infants who had the supposedly protective allele.

It's fascinating to examine these studies with the orchid hypothesis in mind. Focus on just the bad-environment results, and you see only vulnerability. Focus on the good-environment results, and you see that the risk alleles usually produce better results than the protective ones. Securely raised 7-year-old boys with the DRD4 risk allele for ADHD, for instance, show fewer symptoms than their securely raised protective-allele peers. Non-abused teenagers with that same risk allele show lower rates of conduct disorder. Non-abused teens with the risky serotonin-transporter allele suffer less depression than do non-abused teens with the protective allele. Other examples abound—even though, as Jay Belsky points out, the studies were designed and analyzed primarily to spot negative vulnerabilities. Belsky suspects that as researchers start to design studies that test for gene sensitivity rather than just risk amplification, and as they increasingly train their sights on positive environments and traits, the evidence for the orchid hypothesis will only grow.

Suomi gathered plenty of that evidence himself in the years after his 2002 study. He found, for example, that monkeys who carried the supposedly risky serotonin-transporter allele, and who had nurturing mothers and secure social positions, did better at many key tasks—creating playmates as youths, making and drawing on alliances later on, and sensing and responding to conflicts and other dangerous situations—than similarly blessed monkeys who held the supposedly protective allele. They also rose higher in their respective dominance hierarchies. They were more successful.

Suomi made another remarkable discovery. He and others assayed the serotonin-transporter genes of seven of the 22 species of macaque, the primate genus to which the rhesus monkey belongs. None of these species had the serotonin-transporter polymorphism that Suomi was beginning to see as a key to rhesus monkeys' flexibility. Studies of other key behavioral genes in primates produced similar results; according to Suomi, assays of the SERT gene in other primates studied to date, including chimps, baboons, and gorillas, turned up "nothing, nothing, nothing." The science is young, and not all the data is in. But so far, among all primates, only rhesus monkeys and human beings seem to have multiple polymorphisms in genes heavily associated with behavior. "It's just us and the rhesus," Suomi says.

This discovery got Suomi thinking about another distinction we share with rhesus monkeys. Most primates can thrive only in their specific environments. Move them and they perish. But two kinds, often called "weed" species, are able to live almost anywhere and to readily adapt to new, changing, or disturbed environments: human beings and rhesus monkeys. The key to our success may be our weediness. And the key to our weediness may be the many ways in which our behavioral genes can vary.

ONE MORNING THIS past May, Elizabeth Mallott, a researcher working at Suomi's lab, arrived to start her day at the main rhesus enclosure and found a half-dozen monkeys in her parking spot. They were huddling close together, bedraggled and nervous. As Mallott got out of her car and moved closer, she saw that some had bite wounds and scratches. Most monkeys who jump the enclosure's double electrified fences (it happens now and then) soon want to get back in. These monkeys did not. Neither did several others that Mallott found between the two fences.

After caging the escapees in an adjacent building, Mallott, now joined by Matthew Novak, another researcher who

knew the colony well, entered through the double gates. The colony, numbering about 100-odd monkeys, had been together for about 30 years. Changes in its hierarchy usually came slowly and subtly. But when Novak and Mallott started looking around, they realized that something big had happened. "Animals were in places they weren't supposed to be," Novak would later tell me. "Animals who don't hang out together were sitting together. Social rules were suspended."

It soon became apparent that the family group called Family 3, which for decades had ranked second to a group called Family 1, had staged a coup. Family 3 had grown larger than Family 1 several years before. But Family 1, headed by a savvy matriarch named Cocobean, had retained incumbency through authority, diplomacy, and momentum. A week or so before the coup, however, one of Cocobean's daughters, Pearl, had been moved from the enclosure to the veterinary facility because her kidneys seemed to be failing. Family 1's most formidable male, meanwhile, had grown old and arthritic. Pearl was especially close to Cocobean and, as the only daughter without children of her own, was particularly likely to defend her. Her absence, along with the male's infirmity, created a vulnerable moment for Family 1.

"This may have been in the works for a couple weeks," Novak says. "But as far as we can reconstruct, the actual event, the night before we found the monkeys in the parking lot, started when a young female named Fiona"—a 3-year-old Family 1 member, a borderline bully known to have initiated many a scuffle—"started something with someone in Family 3. It escalated. Family 3 saw its chance. And they just started to take Family 1 out. You could see it from who was wounded and who wasn't, and who was sitting in preferred places, and who was run out of the colony, and who was suddenly extremely deferential. One other female in Family 1, Quark, was killed; another, Josie, was hurt so badly we had to put her down. They'd gone after all of Cocobean's other daughters, too. Somebody had bitten the big male in Family 1 so badly he couldn't use his arm. Fiona got roughed up pretty bad. It was a very systematic scuffle. They went right at the head of the group and worked their way down."

Soon after Novak described all this to me, he and I walked around the enclosure. Though it was the middle of a broiling July day, downtime for the monkeys, you could see hints of the new order. Family 3 calmly occupied what seemed to be the new center of power, a corncrib near the pond (one of several corncribs set out for shelter). They groomed one another, napped, and evenly stared at us as we stared at them. A more nervous bunch clustered in another crib down the hill. When we got within 30 feet, the largest monkey in the group shot up onto the cage bars. From 10 feet up it screamed at me, rattled the bars, and showed some nasty teeth.

From there I went to Suomi's office and asked him what he thought had happened. Suomi has thought a lot about this coup, and it's easy to see why. All of the important threads he'd been weaving together in his research were on display in this revolt: the importance of early experience; the interplay of environment, parenting, and genetic inheritance; the maddening primacy of family and social bonds; the repercussions of different traits in different circumstances. And now, in light of the orchid hypothesis, he was beginning to see that the threads might be woven together in a new way.

"About 15 years ago," he said, "Carol Berman, a monkey researcher at SUNY-Buffalo, spent a lot of time watching a large rhesus-monkey colony that lives on an island in Puerto Rico. She wanted to see what happened as the groups changed size over time. They'd start at about 30 or 40 individuals—a group that had split off from another—and then expand. At a certain point, often somewhere near a hundred, the group would reach its limit, and it, too, would split into smaller troops."

Such size limits, which vary among social species, are sometimes called "Dunbar numbers," after Robin Dunbar, a British evolutionary psychologist who argues that a species' group limit reflects how many social relationships its individuals can manage cognitively. Berman's observations suggested that the Dunbar number of a species reflects not just its cognitive powers but its temperamental and behavioral range as well.

Berman saw that when rhesus troops are small, the mothers can let their young play freely, because strangers rarely approach. But as a troop grows and the number of family groups rises, strangers or semi-strangers more often come near. The adult females become more vigilant, defensive, and aggressive. The kids and adult males follow suit. More and more monkeys receive upbringings that draw out the less sociable sides of their behavioral potentials; fights grow more common; rivalries grow more tense. Things finally get so bad that the troop must split. "And that's what happened here," Suomi said. "It's a very extensive feedback system. What happens at the dyadic level, between mother and infant, ultimately affects the very nature and survival of the larger social group."

Complain all you want that it's an increasingly ADHD world these days, but the orchid theory suggests that it's been an increasingly ADHD world for about 50,000 years.

Studies by Suomi and others show that such differences in early experience can wildly alter how genes express themselves—that is, whether, when, and how strongly the genes switch themselves on and off. Suomi suspects that early experiences may affect later patterns of gene expression and behavior as well, including how flexible and reactive an animal is, by helping to set the sensitivity level of key alleles. A tense upbringing, he says, will produce watchful caution or vigilant aggression in any monkey (the parents' way of preparing the offspring for tough times)—but this effect may be especially pronounced in monkeys with particularly plastic behavioral alleles.

That's what Suomi thinks may have happened in the run-up to what he calls the Palace Revolt. Fiona's injudicious aggression proved disastrous for her and Family 1. But Family 3, a group that had been diplomatically deferring to Family 1 for years, dramatically improved its fortunes by mounting an uncharacteristically aggressive and sustained counterattack.

Suomi speculates that in the tenser, more crowded conditions of the large colony, gene-environment interactions had made some of the monkeys in Family 3, particularly those with more-reactive "orchid" alleles, not more aggressive but more *potentially* aggressive. During the period when they could not afford to challenge the hierarchy—the period before Pearl's departure—aggressiveness would have led them into unwinnable, possibly fatal conflicts. But in Pearl's absence the odds changed—and the Family 3 monkeys exploited a rare and decisive opportunity by unleashing their aggressive potential.

The coup also showed something more straightforward: that a genetic trait tremendously maladaptive in one situation can prove highly adaptive in another. We needn't look far to see this in human behavior. To survive and evolve, every society needs some individuals who are more aggressive, restless, stubborn, submissive, social, hyperactive, flexible, solitary, anxious, introspective, vigilant—and even more morose, irritable, or outright violent—than the norm.

All of this helps answer that fundamental evolutionary question about how risk alleles have endured. We have survived not despite these alleles but *because of* them. And those alleles haven't merely managed to slip through the selection process; they have been actively *selected for*. Recent analyses, in fact, suggest that many orchid-gene alleles, including those mentioned in this story, have emerged in humans only during the past 50,000 or so years. Each of these alleles, it seems, arose via chance mutation in one person or a few people, and began rapidly proliferating. Rhesus monkeys and human beings split from their common lineage about 25 million to 30 million years ago, so these polymorphisms must have mutated and spread on separate tracks in the two species. Yet in both species, these new alleles proved so valuable that they spread far and wide.

As the evolutionary anthropologists Gregory Cochran and Henry Harpending have pointed out, in *The 10,000 Year Explosion* (2009), the past 50,000 years—the period in which orchid genes seem to have emerged and expanded—is also the period during which *Homo sapiens* started to get seriously human, and during which sparse populations in Africa expanded to cover the globe in great numbers. Though Cochran and Harpending don't explicitly incorporate the orchid-gene hypothesis into their argument, they make the case that human beings have come to dominate the planet because certain key mutations allowed human evolution to accelerate—a process that the orchid-dandelion hypothesis certainly helps explain.

How this happened must have varied from context to context. If you have too many aggressive people, for example, conflict runs rampant, and aggression is selected out, because it becomes costly; when aggression decreases enough to be less risky, it becomes more valuable, and its prevalence again rises. Changes in environment or culture would likewise affect an allele's prevalence. The orchid variant of the DRD4 gene, for instance, increases risk of ADHD (a syndrome best characterized, Cochran and Harpending write, "by actions that annoy elementary-school teachers"). Yet attentional restlessness can serve people well in environments that reward sensitivity to new stimuli. The current growth of multitasking, for instance, may help select for just such attentional agility. Complain all you want that it's an increasingly ADHD world these days—but to judge by the spread of DRD4's risk allele, it's been an increasingly ADHD world for about 50,000 years.

EVEN IF YOU accept that orchid genes may grant us flexibility crucial to our success, it can be startling to ponder their dynamics up close and personal. After I FedExed away my vial of saliva for genotyping, I told myself more or less to forget it. To my surprise, I managed to. The e-mail that eventually arrived with the results, promised for a Monday, turned up three days early, during a Friday evening when I was simultaneously half-watching *Monsters, Inc.* with my kids and distractedly scanning the messages on my iPhone. At first I didn't really register what I was reading.

"David," the message began. "I ran the assay on the DNA from your saliva sample today. The assay ran well and your genotype is S/S. Good thing neither of us think of these things as deterministic or even having a fixed valence. Let me know if you want to talk about your result or genetic issues."

When I finished reading the message, the house seemed quieter, though it was not. As I looked out the window at our pear tree, its blossoms fallen but its fruit only nubbins, I felt a chill spread through my torso.

I hadn't thought it would matter.

Yet as I sat absorbing this information, the chill came to seem less the coldness of fear than a shiver of abrupt and inverted self-knowledge—of suddenly knowing with certainty something I had long suspected, and finding that it meant something other than I thought it would. The orchid hypothesis suggested that this particular allele, the rarest and riskiest of the serotonin-transporter gene's three variants, made me not just more vulnerable but more plastic. And that new way of thinking changed things. I felt no sense that I carried a handicap that would render my efforts futile should I again face deep trouble. In fact, I felt a heightened sense of agency. Anything and everything I did to improve my own environment and experience—every intervention I ran on myself, as it were—would have a magnified effect. In that light, my short/short allele now seems to me less like a trapdoor through which I might fall than like a springboard—slippery and somewhat fragile, perhaps, but a springboard all the same.

I DON'T PLAN to have any of my other key behavioral genes assayed. I don't plan on having my kids' genes done, either. What would it tell me? That I shape them in every encounter? I know this. Yet I do like thinking that when I take my son trolling for salmon, or listen to his younger brother's labyrinthine elaborations of his dreams, or sing "Sweet Betsy of Pike" with my 5-year-old daughter as we drive home from the lake, I'm flipping little switches that can help light them up. I don't know what all those switches are—and I don't need to. It's enough to know that together we can turn them on. A

David Dobbs is the author of Reef Madness: Charles Darwin, Alexander Agassiz, and the Meaning of Coral *(2005). He writes on science, medicine, nature, and culture, and blogs at neuronculture.com.*