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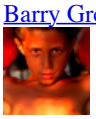

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The Science of Success

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.

By David Dobbs

Vault49

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.

VIDEO: Watch an interview with Stephen Suomi, one of the researchers featured in this story

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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 nonintervention 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.

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.

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.

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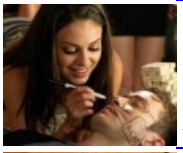
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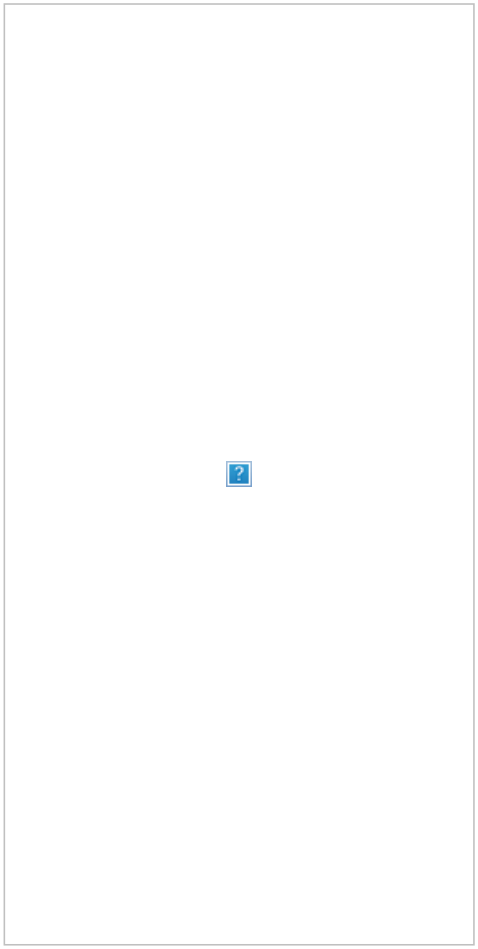

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