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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





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

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.

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 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 over the last few years. 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 serotonintransporter 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 badenvironment 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 protectiveallele peers. Non-abused teenagers with that same risk allele show lower rates of conduct disorder. Nonabused 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."

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 *becauseof* 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.



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I would dearly love to see research into the autism spectrum approached from this perspective. So much of what is currently diagnosed as autistic behaviour would, in a "normal" child, be interpreted as red flags for abuse, PTSD, and maternal deprivation -- maybe it's not autism at all that's being diagnosed, but secondary problems that autistic people are more susceptible to? People on the autism spectrum do have things in common, a cognitive style if you will (heightened senses and pattern-finding abilities primarily, which can and do manifest in any number of ways, good and bad), and we do seem to have a lower Dunbar number than neurotypicals, but what if that's all it is? What if autism spectrum people are just orchid outliers, just super-sensitive, and the criteria used to "diagnose" us are just artefacts of damage done by exposure to an especially hostile (for us) environment?

I'm not saying I think any of this is necessarily true, just that it could be, and that maybe the question should at least be asked.

9 people liked this. Like Reply
drdorotheaboehm 2 years ago
Deeply impressing article - Thank you, David Dobbs.
Seems like a good start is something vital. Makes me wonder, if it really is a the best of ideas to slam our smallest children in creches.
5 people liked this. Like Reply
angelachampagne 2 years ago
As a parent of two ADD/ADHD children I was very impressed by this article. I only wish that more people would read articles like this. It would help people understand how to teach and understand ADD/ADHD children so that they do not give up on them self at any age. Thank you for giving me some more insight to my children!!
7 people liked this. Like Reply
sarahbutland 2 years ago
I've read to my child every day since birth and he's only 4 months old now but it's amazing that he seems to be following along intently. He sits in his jumper chair usually and remains quiet as can be. I stop when he's no longer attentive and we read more the next day. I'm so amazed by him each and every hour.
5 people liked this. Like Reply
danaeris 2 years ago
I've known about the allele in question for years now, but I did not know about the new research in the area, and the orchid-gene hypothesis. I've been plagued with depression my whole life, but until a string of bad luck that lasted many years, I didn't begin to suffer from crippling fear and anxiety.
I've often wondered what the test would reveal about me, and I must admit I suspect that it would reveal, as yours did, that I have the SS alleles.
I've always thought that knowing something like that would be a two-edged sword. If you learn that you are an SS, it tells you that it isn't your imagination, and it isn't your fault. There's a real, solid reason for the way you are. But that can easily turn into an excuse, a prison you fashion for yourself that keeps you from trying, from striving.
Reading this article, and the way you now perceive your results, however, fills me with a surprising and unexpected feeling of hope. It doesn't matter whether or not my mood troubles are related to the orchid-gene. The important lesson I can take away from this story is that
suffering from a mood 'disorder' can be a matter of perspective. I may not have figured out how to draw out the positive edge of the blade, but there might still be a positive edge.
suffering from a mood 'disorder' can be a matter of perspective. I may not have figured out how to draw out the positive edge of the blade, but there might still be a positive edge. It makes me think of metals. Unlike iron, gold is relatively malleable and forgiving. Iron is brittle and will crack if you treat it like gold, but if you treat it properly, it has properties that make it valuable in its own way.

Thank you for showing people like me another way.

20 people liked this. Like Reply

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Mariska7 2 years ago

For people who work with gifted children and adults this is old news (but always worth repeating, since it still goes against the grain of conventional psych thinking and knowledge).

Gifted kids (and adults) are disproportionately endowed with a trait called overexcitability (from Kazimierz Dabrowski's Theory of Positive Disintegration -- I strongly recommend for all interested in Mr. Dobbs' article to look it up), which is inborn, significantly higher than average, sensitivity and responsiveness of the nervous system to internal and external stimuli. This increased sensitivity, which, by the way, correlates proportionately with intelligence and special abilities, predisposes one to greater risks for developmental and psychiatric problems, but it also contributes to higher than average creativity and achievement in all spheres of human functioning. There is quite a lot of research confirming the correlation between overexcitability and giftedness -- it's too bad that each discipline, even within the same field (i.e., psychology) has to rediscover the wheel without building on the already existing body of knowledge.

20 people liked this. Like Reply



Researcher 2 years ago

Investigators of children and aggressive or violent behavior are not including Subliminal Distraction exposure. In seven years searching I cannot find anyone in medicine or psychiatry aware that this problem exists.

Forty years ago SD caused mental breaks for office workers. The cubicle was designed to deal with the vision startle reflex to prevent believed-to-be temporary harmless episodes of confusion.

Investigators are unaware that children sitting on the floor fully mentally invested in play or sitting engrossed in a TV program subliminally detect movement in their peripheral vision.

That subliminal detection of threat-movement and the brain's reaction to it short of a vision startle reflex is a Subliminal Distraction.

It can be shown that SD exposure will produce fear, depression, paranoia, and panic attacks.

Too-close side-by-side seating in classrooms is the same design problem. No one has investigated to determine if ADD and ADHD is a child's reaction to Subliminal Distraction exposure.



CoachDebra 1 year ago It would be interesting to then test this theory to see if adults who because their genetic sensitivity and childhood issues could then be transitioned as adults given emotional nourishment and retraining, perhaps via NLP/Hypnosis or CBT, to enhance their creativity and those valuable traits that make them an orchid 5 people liked this. Like Reply

David Dobbs 1 year ago in reply to CoachDebra

Reply

when comments came in. There are some studies testing this in adults, and I'll be writing about them in my book, and perhaps in some articles meanwhile. You can keep up with me if you'd like at http://daviddobbs.net or my blog, http://neuronculture.com. Thanks for your interest. I too obviously find this fascinating, and it just gets juicier as I get deeper and deeper into the research and into the stories of people whose lives show these dynamics at work. David Dobbs http://daviddobbs.net http://neuronculture.com Like Reply CoachDebra 1 year ago in reply to David Dobbs Reading an article in an old New Yorker about the changes in the brains of children who have had traumatic experiences in their lives and the predictive nature for future disease (not just dysfunction). Seems to dovetail with this information. Reply Like general insurance 1 year ago Firstly you will need to check the Fork and Shock sag: this is the amount the forks and rear shock settle under load. To measure it do the following: push down on the forks a number of times to settle them, then mark the stanchion with a felt pen or put a cable tie where the dust seal is sitting. Next ask some for help to lift on the bars so the front wheel is just off the ground and measure the amount the forks have traveled down. This is the static sag (or unladen sag), This can be changed by adjusting the spring preload (more preload = less sag). Repeat the same process for the rear, this time measuring the distance from the wheel spindle to a fixed point on the tail. Now you are ready to begin setting up your suspension. The key is to do it a little at a time and make notes as you go. For road riding start with the wet track settings and work from there. Like Reply GideonW 1 year ago As a child, teen and now adult sufferer of both depression and ADD, I think the Dandelion & Orchid theory holds a lot of water. However, I believe there is an evolutionary reason for this particular genetic aberration; one which must be examined, for it holds the solution to the problem. I believe that one must examine nature's purpose for this aberration in order to understand how to address it and adapt it to modern life. Since the evolution of man from nomad to homesteader a fundamental change took place at the genetic level. Some people adapted to this new life as gatherers then farmers then craftsmen and so on through to the middle managers and cubicle

David Dobbs here, the author of the article. Sorry to be so very slow responding here. I thought I was getting auto email updates

these traits; they served and still serve a purpose, the continued survival of the species. The people carrying these particular traits are the

workers of modern society. These are the dandelions. Conversely, the other group of people became the hunters and warriors of society, charged with finding meat and protecting the homestead from predators, both animal and human. In this role, hyper-intelligence, hyperactive senses (distractibility), aggressive tendencies, emotional insensitivity, risk-taking, and a slew of other now undesirable traits were what kept those individuals alive as well as protecting and providing for society. And, this is why natural selection has not removed

orchids. In the right environment, for example as a warrior or athlete, the orchid would thrive and excel. His genetics would allow him to train harder, fight better, emotionally distance himself from his enemy thus making him a more effective killer, and so on. But, try sticking the warrior on a farm, or to make him a scribe, a factory lineman, or a cubicle worker, and he will wither and fail. He does not have the disposition for focus on detail or repetitive tasks...he is a warrior. I too, am a warrior. All of the men of my paternal line were warriors, genetically speaking that is. Though all also were soldiers as well. Those traits which society now attempts to medicate away, once kept we warriors alive, and more importantly allowed those that did not have those traits to survive and thrive under our protection. It's not just that we have orchids and dandelions, but also livestock and guard dogs, farmers and warriors, or however you wish to categorize this genetic difference. Now the question is how do we as parents of orchid children (or we the orchid adults) refocus the orchid traits into skills which will help our children (or us) adapt and excel in modern society?

3 people liked this. Like Reply

MAS 1 year ago in reply to GideonW

I'm sorry to hear you case Gideon. Besides depression I think you also suffer some insanity, but you've got some interesting points that have some truth.

The hunters-warriors never protected any society other than the small groups or tribes they would tag along. "Warrior" is a deceitful term of trouble-makers, that is what a warriors really is, or more specifically a thief, rapist and else. They destroyed the Roman Empire and started the Dark Ages, and so it was for other civilizations.

While these trouble makers do share many characteristics of the orchid type, they hardly come at half way to the full profile. Remember, the orchids break easily out of optimal circumstances, not what you'd expect from a hunter who has to adapt to the ever changing environment and always in the look for their next victim. Well, you well said that are more than orchids and dandelions, there is also live stock and guard dogs, but for the case that you describe you shouldn't have said "guard dogs", but "wolves", "snakes" and "wasps" are better analogies.

As for your question, this article has some good suggestions, like reading to children. It helps a lot.

Like	Reply
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GideonW 1 year ago in reply to MAS

Hello Mr. Pot, this is Mr. Kettle calling...really? Don't feel sorry for me, my need for thrill seeking and adrenaline has led me to great financial success as an entrepreneur. I thrive on the thrill of the hunt. It's my warrior nature to fight and to win. And in modern society, that means doing battle in the business world. A result of which has led me to a fulfilling work life and the amassing of wealth. Now, you dare insinuate through your barely legible "writing" skills that I suffer from insanity? Whether I do or not is my business, but someone espousing the idea that "Warriors" are all "deceitful," "trouble making," "thieves" and "rapists" should consider a little counseling and stronger meds for him/herself! By your logic, all US Marines are all theives and rapists? Navy SEALS are all deceitful trouble makers? The fact is that people like me, orchid warriors, now and for many millenia prior made is possible for people like you to not get eaten by wolves or murdered by brigands because you lack the proper survival instincts and skill set to protect yourself. So, rather that attacking other people's sanity, look long and hard in the mirror, reflect upon your weaknesses, and be thankful that the orchid warriors are out the protecting you. You're welcome!

2 people liked this. Like Reply

MAS 1 year ago in reply to GideonW

Yes, I committed some writing mistakes. I'm normally in a hurry! Thank you for replying to my blog and much more for presenting a great example on how warriors are nothing but trouble-makers. You're so nice, are you sure you're a trouble-maker? Warrior I mean!

Well, trouble makers come in several colors and sizes, some are deceitful, some are thieves, etc. And some are all. I don't think any of the US army branches has rapists or thieves (or too many of them at least) but they do not protect me or any other normal person. They protect multinational companies, mostly oil ones. However, they do parasitically live from my federal taxes, which are the largest and nobody escapes them, trouble-makers. And each soldier is instructed to kill, many actually do it, all are capable of, and the final goal was stealing from those nations.

All the protection that America –and almost any other nation- really needs is in the border, not on the other side of the world. Gone are the days when nations would swallow other nations. Spain conquered the Aztecs and Incas, all the way from Mexico to almost the South Pole; the Brits conquered India and from Africa to South Africa, etc. That was then, and by the way. For each protecting "warrior", there was another parasite warrior. No warriors at all would've been the solution to all wars. Or more generally, no trouble-makers could be the solution to all trouble, at least to a lot of it.

That was then. Nowadays, this whole nation of the USA cannot control two tiny sparsely population 3rd world nations. So, what protection have red-wingers been talking about for so long? Have you feared that Iraq (or some another one) could've invaded us first and conquered the whole nation? Do you realize how insane that is? Feared an atomic or bio attack? North Korea has already had such weapons since who knows and shown plenty of sings to be lethal. Meanwhile, job-security is the least of worries for the guy in charge over there. And for the other guy who supposedly got us in the war mess, what was his name? Osaka bin Roguen?

But the same corporate world has made sure to let the border remain as porous as to let millions and millions of illegals in and countless factories out to China through the years, devastating the middle class, especially men, now that the only good job market is in health care. This has been totally unproductive for a nation because hospitals cannot export its services. Here's where we've needed protection of warriors all these decades! But that would've meant solving trouble.

Anyways, next time I see a fight or something, I'll warn the bullies about my new orchid warrior friend who'll bust their behind. I'll send them right to you! You love action.



Keith Ferguson 1 year ago

While I feel that it's important to know that some people have different genetic architecture that can explain certain abnormalities, it's important to note in this study that ALL the kids did better when they were treated like orchids. Orchids may thrive under optimal conditions, but that's no reason to segregate them from dandelions, who are improved by those conditions, as well.

I would like to see more study done on the question of how culture affects this dynamic: is it possible that a child with gene A would be a dandelion in the US and an orchid in Japan?

4 people liked this. Like Reply



MAS 1 year ago in reply to Keith Ferguson

It was already noted that ALL kids did better in good conditions, it just figures. You missed the whole point, or the main idea as they say in the GRE tests. I thought the author was a bit repetitive but you showed that he actually needed to be even more repetitive.

The main point being stressed here for those studies was who does better in good conditions. Again, most people will do well in good conditions, some will do even better and those same ones turn out to be the same ones who fail otherwise.

This research can explain why so many children are given so much by their parents, teachers and the whole world with mediocre outcomes in life while other children who don't get so much get to move ahead in this world, because these fortunate children were not meant biologically to reach very high. They are dandelions, just like most people. And those other children who move ahead despite adversity are dandelions as well, not meant to reach very high either but not to fall either; meant to survive.

This research can help directing resources where they need to and avoid waste. Parents today will do the best and ultimately just spoil their children but schools can do different and focus on children in more proper ways. There are already schools for gifted children, but they strictly only pay attention to children from good parents. Children of poor parents normally face all that poverty brings with it and as the orchid theory shows, these children normally fail, become gang members, addicted to drugs, etc and nobody notices any potential in them. The schools for gifted children actually help increase the gap between rich and poor because it's usually rich parents the ones who provide the good condition that let people realize that a child may be gifted. If you are poor, nobody will pay much attention to your kid for real.

This same research can be applied to adults in all the different scenarios where they live or work.



MAS 1 year ago in reply to Keith Ferguson

Regarding your last question, bad and good conditions are at the end just the same in the whole world. the real difference among nations is that in some nations good conditions are better and in others bad conditions are worse (3rd world). An orchid child child should be the same everywhere. NOW, if you take the child raised in the USA and take him to Japan without proper support, will normally fail. But as being an orchid or dandelion, he'll stay put, just like his skin color, hair, eyes, etc.



Like

Reply

David Dobbs 1 year ago in reply to Keith Ferguson

Keith:

David Dobbs here, author of the article. Sorry so slow getting back.

You asked "I would like to see more study done on the question of how culture affects this dynamic: is it possible that a child with gene A would be a dandelion in the US and an orchid in Japan?"

Someone asked just that question about the serotonin transporter gene and its rates and effects globally. The answer is absolutely fascinating. I explored it in a post at Neuron Culture, my blog (<u>http:neuronculture.com</u>), in this post:

http://www.wired.com/wiredscie...

Rather mind-blowing.

Thanks for writing.

David Dobbs

Contributor, Atlantic Monthly, New York Times Magazine, Nature, Slate, National Geographic, Wired, and other publications

Author

The Orchid and The Dandelion (in progress)

Reply

Reef Madness: Charles Darwin, Alexander Agassiz, and the Meaning of Coral (Pantheon, 2005)

Bharat Nitish Ponnuswamy 1 month ago in reply to David Dobbs

thank you sir for your valuable research. i wish lawmakers would understand the sheer importance of what you are doing and think about necessary educational reforms in order to provide a environment better suited for the development of a "orchid child".

your article also brings about a whole new perspective of how correctional institutions should operate. I am eagerly awaiting your book.

ADD student from india

1 person liked this.

Like

Like Reply



rjpeers 1 year ago

This flower theory, although highly speculative, deserves attention, because it raises the question: do apparently "bad" genes for mental ill health have a good side to them? We await more evidence on the cited serotonin and dopamine genes, but hey guys, why no mention from David of Bipolar Disorder, which is a spectacular example of his thesis (indeed, it may be the ONLY example, if the aforementioned genes don't do much to improve the brain).

One only needs to read the literature on Bipolar and creativity--e.g. Kay Redfield Jamison's book Touched With Fire, or The Bipolar Advantage--to realize that these genes are really for Benign Unipolar Hypomania (as in Teddy Roosevelt--never manic or depressed; or George Handel, likewise). Like schizophrenia, where the genes normally lead only to benign schizotypy, Bipolar has only become common since the Western diet became notably fatty, about 1800, with the rising consumption of meat fat and dairy fats.

Fatty maternal diet causes anxiety in the offspring, and also converts the resulting anxiety to depression. Many writers and creative people are intelligent but anxiety-driven (e.g. they write overlong books!). Many creative and adventurous souls, too, are bipolar or--if more fortunate--merely Benign Unipolar Hypomanic, as were quite a few early American leaders and pioneers.

So both anxiety and mild hypomania can have useful social effects, but prior to the modern high-fat era, anxiety would have been rare-confined, like diabetes, to the wealthy--while the roughly 4 % of people with mild hypomania may have stood out in leadership, storytelling and the arts, and occasionally in science (e.g. Newton).

As for ADHD, this is a modern brain disease caused by refined seed oil consumption in pregnancy (the same oils, which are low in vitamin E, cause Alzheimer's in late life). ADHD kids have few advantages--but may be creative in architectural design (if they can concentrate long enough, and hold down a job!). The more common outcome is school truancy, drug abuse, unemployment and antisocial aggression. Any ADHD kid who ALSO has some predisposing gene, like DRD4 or whatever, will do a little better, because he is not all ADHD brain damage--part of his hyperactivity is genetic and harmless.

A sad example of anxiety having dual effects in an intelligent person is the author Colleen McCulloch, whose anxiety has driven her not only to be a prolific writer, but also to comfort-eat fatty treats, and to smoke: result being diabetes, breast cancer and encroaching blindness. Then there were the anxious but alcohol-afflicted poets and writers Henry Lawson, Dylan Thomas, James Joyce and Brendan Behan.

It remains to be shown whether the serotonin and dopamine genes, above, confer mental advantages similar to those undoubtedly linked to Benign Unipolar Hypomania. In the meantime, ordinary mortals might enjoy quicker wits, by avoiding fatty foods and refined oils(which oxidize and slow the brain); and by increasing their dietary intake of inositol (an anti-ageing, energizing seed sugar--in grains, nuts, legumes and citrus), and fish oil, choline (eggs, milk) and uridine (beets, broccoli)--these three nutrients are building blocks for new synapses and enhanced cognition (Prof R Wurtman, Boston). Happy eating!

3 people liked this. Like Reply



I'd like to see your peer reviewed data regarding fatty foods causing anxiety and depression, as well as that pertaining to "refined seed oil consumption in pregnancy" leading to ADHD. Sounds suspect at best.

Like Reply

rjpeers 1 year ago in reply to GideonW

The simplifying and clarifying conclusions reached using the methods of inductive science (data collection followed by pattern recognition and a concluding hypothesis with inductive strength), being simple, are likely--to the uninitiated (esp. those unfamiliar with nutrition)--to look like "suspect" guesswork. They are not. My first clues to fatty maternal diet being the cause of anxiety came from studies linking such a diet with gestational diabetes (L Tapsell, 1997), and also linking that disorder with introversion in the offspring (B Metzger, 1997). It takes no great intelligence to draw a simple conclusion from that. In addition, studies on fetal development have shown that placental inflammation--seen in gestational diabetes--partially inactivates the fetal enzymatic barrier to maternal cortisol. Corticosteroid hormones are known, in turn, to cause offspring anxiety, by epigenetically altering glucocorticoid receptor expression in fetal brain (M Holmes, Edinburgh), resulting in permanently activated HPA stress axis. As for depression, numerous studies show that there is usually underlying anxiety: fatty diet converts anxiety to depression, by oxidizing and inflaming the brain (B Culver, Wyoming), and also exposing the brain to depressogenic inflammatory cytokines (B Baune). The scientific hypothesis underlying my research is based on dietary fatty acids and membrane structure: fatty diet (polyunsaturate/saturate ratio below 1:1) creates faulty, polyunsaturate-deficient plasma membranes (M Clandinin) and mitochondrial membranes (P Divakaran), resulting in insulin resistance (as seen in depression [S Koslow]), accompanied by mitochondrial superoxide release, which drives low-grade inflammation in all tissues, including brain. And so to ADHD: in 1990, being aware of identical-twin discordance in sporadic Alzheimer's, I deduced a dietary exposure, and soon found in the medical library that refined seed oils are seriously depleted of vitamin E in processing (D Herting, 1963). Knowing that the brain and retina have the highest concentrations of peroxidation-prone polyunsaturated fatty acids, I quickly identified, in my refined-oil consuming patients, a new syndrome of amnesia, photophobia and night blindness (Seed Oil Syndrome), in which the memory improved with vitamin E. Refined seed oils have now been directly linked with Alzheimer's (P Barberger-Gateau, 2008). I also noticed that young refined-oil using families often had children with ADHD: so I proceeded to a case/control study of pregnancy diet (albeit retrospective), that showed that 78 of 80 mothers of ADHD children had regular exposure to refined seed oils during pregnancy, compared with zero out of 80 mothers of controls. I later found that ADHD children taken off refined seed oils improved rapidly. Local paediatricians declined to repeat my study in formal and detailed fashion, so I have not published it, although perhaps I should have published a few cases as case reports, to draw attention to this important discovery. My ADHD theory is supported by studies showing that lipid peroxidation (known, since 1938, to be induced by vitamin E deficiency) slows the growth of fast-growing tissues, like cancer, or embryonic brain: the ADHD brain is about 3% smaller than average. In addition, B Ross (2003) has shown that some ADHD children expire on their breath the volatile gas ethane, which is a known marker of peroxidized Omega-3 fatty acids, which are richly concentrated in brain synapses. If you require more details, please contact me on info@drrobertpeers.com. I am an independent researcher at night, and a practising general practitioner (family physician) by day. I trained at Melbourne University, graduating in 1969. My most recent discovery is a simple nutrient found in seeds, that activates the same anti-ageing genes as caloric restriction; if you lack nutritional training, you might care to learn what this is.

Like Reply

rjpeers 1 year ago in reply to GideonW

Dear GideonW,

I have posted my reply to your comment on the Atlantic Monthly website, and hope you are familiar with dietary fatty acid research. You are welcome to contact me for further details, including--if you are interested--my recent discovery of an antiageing nutrient in seeds and beans, that also treats anxiety and depression very effectively, and even mops up toxic soluble beta amyloid peptides in Alzheimer brain.

Best, Rob

Like Reply



MAS 1 year ago

It was already time that someone would wonder why some genetical traits have remained in humankind for so long when they seem to be so negative. I've got you another trait that can well have as equally negative effects in life as alcoholism or depression but has barely gotten enough attention: Introversion. It comes mostly from our genes.

Since Sigmund Freud started talking about how sexual repression can lead to insanity like a century ago, the promotion for extroversion has grown and eventually got the full support of virtually all kind of media of all ideologies. It makes sense because those people are too prone to be extroverted and therefore will gladly promote their own values.

Meanwhile, introversion has been highly pushed away and plain despised. My favorite example comes from a recent movie. "What happens in Vegas" where the male main character is a typical media hero: an extroverted and loser guy who works actually better than he is recognized. The opposite character of him is an Asian guy, a typical introvert and shown as a total waste who is plain and physical pushed away by the movie's hero, a display of jerkness that the media turns into a winning and rightful attitude. I will use this movie as a reference point or media symbol from now on.

The media and the psychologists have been openly teaching us to defer to extrovert people and reject the introvert. Has it mattered? Quite indeed. Being an introvert is a serious barrier in getting jobs and being promoted or even lasting in jobs. The same applies when seeking a couple all along, I think especially for men, who are more prone to be introverted.

From the Vegas movie we should infer that the extroverted guy is the orchid person, gifted person who needs the proper support. Now, that is a movie, meant to be sell tickets and therefore it should people what they want to hear. Nowadays we have in USA and other nations a huge generation of people who grew spoiled and told how special and important they are, regardless of the truth. Sometimes parents and schools can even stop games to keep a team from beating the other one for fears of lowering their self-esteem or showing them that they are not really that gifted. These children grow up looking up at extroversion and becoming such or other wise deferring to them for good, regardless of how good an extrovert really is in anything. So, it seems that what the Vegas movie sells to the young audience is that despite their failing life, they are still great somehow, right what they want to hear.

Not much should be expected from an extrovert, their main goal is usually just having fun after all. I've ran into so many of them. And when they fail, they will be in denial. I think it's much more possible that it's the introvert who is the orchid.

As mentioned, the orchid people are very sensitive to new experiences and capable of learning from them. Therefore, they should be people who love experiences, therefore open to them, in the constant search for them. Most people would assume that it's the extroverted the one we can expect to be the new experience seekers and it actually makes sense, or sounds logical but some research has found out that it may not be the case, that it may be the introverts the seekers.

You can get your start in that search in the article "The United States of Mind" at The Wall Street Journal by Stephanie Simon. The web site has an interactive map that shows research by Peter Jason Rentfrow, lecturer at the University of Cambridge in England.

The map shows that the same areas with more people open to new experience live in areas well known for innovation, there you have California of course, all the West and North East coasts. And people in those areas turned out to be mostly introverted. This research shows something that always comes with the orchid types: failure. The areas ranking high in openness are also prone to have high crime rates. Remember those results from your article about those bully young apes? I cannot guarantee how much (if at all in fact) introversion is related to the orchids, but openness looks like a definite must for the orchids.

The extroverted regions do not fare too well. They are the rusty Mid-West and the backwards South. I lived for some years in the Mid West and can testify the veracity of the research. You find many extroverted people there, some pathologically so, always thinking in fun, and they are usually not open to new ideas because they simply have with the fun that they already have. Not for nothing they South has a saying "If it ain't broke, don't fix it!". Seeking truly new excitement or life styles is hard work. By the way, extroversion is also crime related. Just think about the macho type environments at the bars and football stadiums, places of fun whose audiences can be expected top be conventional for all the noise they make.

Despite the media has had to admit that being extrovert can actually lead to miserable lives as what we see from the celebrities they continue to preach their chaotic values. The Mid West and the South also have quite a bit of very sour people. Outsourcing jobs to China has badly affected their regions, but so is their messy lifestyle.

Fortunately, some opposition in the media has been slowly growing. Just a few months ago the magazine Psychology today finally released an article on the topic of introversion in a favorable way and the Skeptic magazine has just released an article on happiness with the purpose of debunking the current obsession with it. It didn't focus on extroversion but it did not defer to them at all. It plain mentioned that research has shown that one extrovert is usually good in a team, but more start to become a nuisance.

Finally, the possible link between introversion and the orchid types cold explain why introvert people can be lazy, incompetent or failing in any case, despite they come across as serious and hard-workers. That tendency exists and only that, tendency.

Like Reply



Guest 1 year ago

In-f*cking-credible. And I'm speaking from an American pov, but people who are able to, need to read and understand this. Even if you think the science is iffy, you should know that parenting in the early (plasticity) years of life is critical and how you treat them is what you'll get. Respectful parents = respectful children. With respect for a child as separate, independent; the dandelions *and* the orchids can benefit. An obedient child is your own sick desire, fear & hate is what it breeds. Independence and the journey to it, is evolutionarily & biologically necessary.

Of course, I'm aware of exceptions and the huge gray area in which they exist. But respect should be the default parental position when a child is born. And at least a modified version of attachment version, couldn't sleep with my babies, but they were in the bassinet an inch from my bedside and when they cried I responded. Attachment parenting is instinctual for animal mothers, how did we get so far gone and so willfully ignorant blind to our own humanity?

Answer: Religion is the curse for human animals. Was there a genetic defect in the first mother who ever allowed a man, a god, a promise of heaven, or a threat of hell to even try to tell her to deny her instincts and refuse to meet all the needs of her child until that child can successfully meet them himself? Why didn't she tell him to go fuck himself? Those are my existential questions. I know that ignorance is the root of this & it is what is. But the fact that "christian parenting" books even exist & sell millions of copies tells me that we are still willfully, perversely, inexusably, ignorant.

"It is a shameful thing to insult a little child--it has its feelings, it has its small dignity; and since it cannot defend them, it is surely an ignoble [a better descriptor exists I just can't think of what it is] act to injure them." \sim Mark Twain



David Dobbs 11 months ago in reply to Guest



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